MMWR™
Morbidity and Mortality Weekly Report
www.cdc.gov/mmwr

Recommendations and Reports | June 18, 2010 / Vol. 59 / No. RR-4

U.S. Medical Eligibility Criteria for Contraceptive Use, 2010

Adapted from the World Health Organization Medical Eligibility Criteria for Contraceptive Use, 4th edition

Continuing Education Examination available at http://www.cdc.gov/mmwr/cme/conted.html

DEPARTMENT OF HEALTH AND HUMAN SERVICES
CENTERS FOR DISEASE CONTROL AND PREVENTION

The *MMWR* series of publications is published by the Office of Surveillance, Epidemiology, and Laboratory Services, Centers for Disease Control and Prevention (CDC), U.S. Department of Health and Human Services, Atlanta, GA 30333.

Suggested Citation: Centers for Disease Control and Prevention. [Title]. MMWR 2010;59(No. RR-#):[inclusive page numbers].

CONTENTS

Disclosure of Relationship

CDC, our planners, and our presenters wish to disclose they have no financial interests or other relationships with the manufacturers of commercial products, suppliers of commercial services, or commercial supporters.

This document will not include any discussion of the unlabeled use of a product or a product under investigational use, with the exception that some of the recommendations included in this document may be inconsistent with package labeling.

There was no commercial support for this activity.

U S. Medical Eligibility Criteria for Contraceptive Use, 2010

Adapted from the World Health Organization Medical Eligibility Criteria for Contraceptive Use, 4th edition

Prepared by
Division of Reproductive Health, National Center for Chronic Disease Prevention and Health Promotion

Summary

CDC created U.S. Medical Eligibility Criteria for Contraceptive Use, 2010, from guidance developed by the World Health Organization (WHO) and finalized the recommendations after consultation with a group of health professionals who met in Atlanta, Georgia, during February 2009. This guidance comprises recommendations for the use of specific contraceptive methods by women and men who have certain characteristics or medical conditions. The majority of the U.S. guidance does not differ from the WHO guidance and covers >60 characteristics or medical conditions. However, some WHO recommendations were modified for use in the United States, including recommendations about contraceptive use for women with venous thromboembolism, valvular heart disease, ovarian cancer, and uterine fibroids and for postpartum and breastfeeding women. Recommendations were added to the U.S. guidance for women with rheumatoid arthritis, history of bariatric surgery, peripartum cardiomyopathy, endometrial hyperplasia, inflammatory bowel disease, and solid organ transplantation. The recommendations in this document are intended to assist health-care providers when they counsel women, men, and couples about contraceptive method choice. Although these recommendations are meant to serve as a source of clinical guidance, health-care providers should always consider the individual clinical circumstances of each person seeking family planning services.

Introduction

In 1996, the World Health Organization (WHO) published the first edition of the *Medical Eligibility Criteria for Contraceptive Use* (MEC), which gave evidence-based guidance on the safety of contraceptive method use for women and men worldwide who had specific characteristics and medical conditions. Since that time, WHO has regularly updated its guidance on the basis of new evidence, and the WHO MEC is now in its fourth edition (*1*).

CDC, through close collaboration with WHO, has contributed substantially during the last 15 years to creation of WHO's global family planning guidance, which includes four documents: the medical eligibility criteria for contraceptive use, the selected practice recommendations for contraceptive use, a decision-making tool for clients and providers, and a global family planning handbook. This WHO guidance has been based on the best available scientific evidence, and CDC has served as the lead for establishing that evidence base and presenting the evidence to WHO for use during its expert working group meetings to create and update the guidance.

WHO has always intended for its global guidance to be used by local or regional policy makers, managers of family planning programs, and the scientific community as a reference when they develop family planning guidance at the country or program level. The United Kingdom is one example of a country that has adapted the WHO MEC for its own use (*2*).

CDC undertook a formal process to adapt the WHO MEC at this time because the fourth edition of the WHO guidance is unlikely to undergo major revisions in the near future. Although the WHO guidance is already available in the United States through inclusion in textbooks, use by professional organizations, and incorporation into training programs, the adaptation of the guidance ensures its appropriateness for use in the United States and allows for further dissemination and implementation among U.S. health-care providers. Most of the U.S. guidance does not differ from the WHO guidance and covers approximately 60 characteristics or medical conditions. However, several changes have been made, including adaptations of selected WHO recommendations, addition of recommendations for new medical conditions, and removal of recommendations for contraceptive methods not currently available in the United States (Appendix A).

This document contains recommendations for health-care providers for the safe use of contraceptive methods by women and men with various characteristics and medical conditions. It is intended to assist health-care providers when they counsel women, men, and couples about contraceptive method choice. These recommendations are meant to be a source of clinical guidance; health-care providers should always consider the individual clinical circumstances of each person seeking family planning services.

Corresponding preparer: Kathryn M. Curtis, PhD, Division of Reproductive Health, CDC, MS K-34, 4770 Buford Highway NE, Atlanta, GA 30341; Telephone 770-488-6397; Fax: 770-488-6391; E-mail kmc6@cdc.gov

Methods

The process for adapting the WHO MEC for the United States comprised four major steps: 1) determination of the scope of and process for the adaptation, including a small meeting; 2) preparation and peer review of systematic reviews of the evidence to be used for the adaptation; 3) organization of a larger meeting to examine the evidence and provide input on the recommendations; and 4) finalization of the recommendations by CDC.

In June 2008, CDC held a 2-day meeting of eight key partners and U.S. family planning experts to determine the scope of and process for a U.S. adaptation of the WHO MEC. Participants were family planning providers, who also had expertise in conducting research on contraceptive safety and translating research evidence into guidance. WHO guidance is used widely around the world, including in the United States, and contains approximately 1,800 separate recommendations. In most cases, the evidence base would be the same for the U.S. and the WHO recommendation, and—because of the extensive collaboration between WHO and CDC in creating the international guidance—the process for determining the recommendations also would be the same. Therefore, CDC determined that the global guidance also should be the U.S. guidance, except when a compelling reason existed for adaptation, and that CDC would accept the majority of WHO guidance for use in the United States.

During the June 2008 meeting, CDC identified specific WHO recommendations for which a compelling reason existed to consider modification for the United States because of the availability of new scientific evidence or the context in which family planning services are provided in the United States. CDC also identified areas in which WHO guidance was inconsistent with current U.S. practice by contacting numerous professional and service organizations and individual providers. In addition, CDC assessed the need for adding recommendations for medical conditions not currently included in the WHO MEC. Through this process, a list was developed of existing WHO recommendations to consider adapting and new medical conditions to consider adding to the guidance.

A systematic review of the scientific evidence was conducted for each of the WHO recommendations considered for adaptation and for each of the medical conditions considered for addition to the guidance. The purpose of these systematic reviews was to identify direct evidence about the safety of contraceptive method use by women (or men) with selected conditions (e.g., risk for disease progression or other adverse health effects in women with rheumatoid arthritis who use combined oral contraceptives). Information about indirect evidence (e.g., evidence from healthy women or animal studies) or theoretical considerations was obtained when direct evidence was not available. CDC conducted systematic reviews following standard guidelines (*3,4*), included thorough searches of PubMed and other databases of the scientific literature, and used the U.S. Preventive Services Task Force system to grade the strength and quality of the evidence (*5*). Each systematic review was peer-reviewed by two or three experts before being used in the adaptation process. These systematic reviews have been submitted for publication in peer-reviewed journals.

For most recommendations in this document, a limited number of studies address the use of a specific contraceptive method by women with a specific condition. Therefore, within the WHO guidance, as well as with this U.S. adaptation of the guidance, most of the decisions about medical eligibility criteria were often necessarily based on 1) extrapolations from studies that primarily included healthy women, 2) theoretical considerations about risks and benefits, and 3) expert opinion. Evidence was particularly limited for newer contraceptive methods. The total body of evidence for each recommendation included evidence based on direct studies or observations of the contraceptive method used by women (or men) with the condition and may have included 1) evidence derived from effects of the contraceptive method used by women (or men) without the condition and 2) indirect evidence or theoretical concerns based on studies of suitable animal models, human laboratory studies, or analogous clinical situations.

In February 2009, CDC held a meeting of 31 experts who were invited to provide their individual perspective on the scientific evidence presented and the discussions on potential recommendations that followed. This group included obstetricians/gynecologists, pediatricians, family physicians, nurse-midwives, nurse practitioners, epidemiologists, and others with expertise in contraceptive safety and provision. For each topic discussed, the evidence from the systematic review was presented; for most of the topics, an expert in the specific medical condition (e.g., rheumatoid arthritis) also gave a brief presentation on the condition and specific issues about contraceptive safety. CDC gathered input from the experts during the meeting and finalized the recommendations in this document. CDC plans to develop a research agenda to address topics identified during the meeting that need further investigation.

How to Use This Document

These recommendations are intended to help health-care providers determine the safe use of contraceptive methods among women and men with various characteristics and medical conditions. Providers also can use the synthesis of information in these recommendations when consulting with women, men,

and couples about their selection of contraceptive methods. The tables in this document include recommendations for the use of contraceptive methods by women and men with particular characteristics or medical conditions. Each condition was defined as representing either an individual's characteristics (e.g., age, history of pregnancy) or a known preexisting medical/pathologic condition (e.g., diabetes and hypertension). The recommendations refer to contraceptive methods being used for contraceptive purposes; the recommendations do not consider the use of contraceptive methods for treatment of medical conditions because the eligibility criteria in these cases may differ. The conditions affecting eligibility for the use of each contraceptive method were classified under one of four categories (Box 1).

BOX 1. Categories of medical eligibility criteria for contraceptive use

1 = A condition for which there is no restriction for the use of the contraceptive method.
2 = A condition for which the advantages of using the method generally outweigh the theoretical or proven risks.
3 = A condition for which the theoretical or proven risks usually outweigh the advantages of using the method.
4 = A condition that represents an unacceptable health risk if the contraceptive method is used.

Using the Categories in Practice

Health-care providers can use these categories when assessing the safety of contraceptive method use for women and men with specific medical conditions or characteristics. Category 1 comprises conditions for which no restrictions exist for use of the contraceptive method. Classification of a method/condition as Category 2 indicates the method generally can be used, but careful follow-up may be required. For a method/condition classified as Category 3, use of that method usually is not recommended unless other more appropriate methods are not available or acceptable. The severity of the condition and the availability, practicality, and acceptability of alternative methods should be taken into account, and careful follow-up will be required. Hence, provision of a method to a woman with a condition classified as Category 3 requires careful clinical judgement and access to clinical services. Category 4 comprises conditions that represent an unacceptable health risk if the method is used. For example, a smoker aged <35 years generally can use combined oral contraceptives (COCs) (Category 2). However, for a woman aged ≥35 years who smokes <15 cigarettes per day, the use of COCs usually is not recommended unless other methods are not available or acceptable to her (Category 3). A woman aged ≥35 years who smokes ≥15 cigarettes per day should not use COCs because of unacceptable health risks, primarily the risk for myocardial infarction and stroke (Category 4). The programmatic implications of these categories may depend on the circumstances of particular professional or service organizations (e.g., in some settings, a Category 3 may mean that special consultation is warranted).

The recommendations address medical eligibility criteria for the initiation and continued use of all methods evaluated. The issue of continuation criteria is clinically relevant whenever a woman develops the condition while she is using the method. When the categories differ for initiation and continuation, these differences are noted in the columns *Initiation* and *Continuation*. Where *Initiation* and *Continuation* are not denoted, the category is the same for initiation and continuation of use.

On the basis of this classification system, the eligibility criteria for initiating and continuing use of a specific contraceptive method are presented in tables (Appendices A–M). In these tables, the first column indicates the condition. Several conditions were divided into subconditions to differentiate between varying types or severity of the condition. The second column classifies the condition for initiation and/or continuation into Category 1, 2, 3, or 4. For some conditions, the numeric classification does not adequately capture the recommendation; in this case, the third column clarifies the numeric category. These clarifications were determined during the discussions of the scientific evidence and the numeric classification and are considered a necessary element of the recommendation. The third column also summarizes the evidence for the recommendation, where evidence exists. The recommendations for which no evidence is cited are based on expert opinion from either the WHO or U.S. expert working group meetings and may be based on evidence from sources other than systematic reviews and presented at those meetings. For selected recommendations, additional comments appear in the third column and generally come from the WHO or the U.S. expert working group participants.

Recommendations for Use of Contraceptive Methods

The classifications for whether women with certain medical conditions or characteristics can use specific contraceptive methods are provided for combined hormonal contraceptive methods, including low-dose (containing ≤35 μg ethi-

nyl estradiol) combined oral contraceptive pills, combined hormonal patch, and combined vaginal ring (Appendix B); progestin-only contraceptive methods, including progestin-only pills, depot medroxyprogesterone acetate injections, and etonogestrel implants (Appendix C); emergency contraceptive pills (Appendix D); intrauterine contraception, including the copper intrauterine device (IUD) and the levonorgestrel IUD (Appendix E); use of copper IUDs for emergency contraception (Appendix F); barrier contraceptive methods, including male and female condoms, spermicides, diaphragm with spermicide, and cervical cap (Appendix G); fertility awareness-based methods (Appendix H); lactational amenorrhea method (Appendix I); coitus interruptus (Appendix J); and female and male sterilization (Appendix K). Tables at the end of the document summarize the classifications for the hormonal and intrauterine methods (Appendix L) and the evidence about potential drug interactions between hormonal contraceptives and antiretroviral therapies (Appendix M).

Contraceptive Method Choice

Many elements need to be considered by women, men, or couples at any given point in their lifetimes when choosing the most appropriate contraceptive method. These elements include safety, effectiveness, availability (including accessibility and affordability), and acceptability. The guidance in this document focuses primarily on the safety of a given contraceptive method for a person with a particular characteristic or medical condition. Therefore, the classification of Category 1 means that the method can be used in that circumstance with no restrictions with regard to safety but does not necessarily imply that the method is the best choice for that person; other factors, such as effectiveness, availability, and acceptability, may play a key role in determining the most appropriate choice. Voluntary informed choice of contraceptive methods is an essential guiding principle, and contraceptive counseling, where applicable, may be an important contributor to the successful use of contraceptive methods.

In choosing a method of contraception, the risk for sexually transmitted infections (STIs), including human immunodeficiency virus (HIV), also must be considered. Although hormonal contraceptives and IUDs are highly effective at preventing pregnancy, they do not protect against STIs. Consistent and correct use of the male latex condom reduces the risk for STIs (*6*). When a male condom cannot be used properly for infection prevention, a female condom should be considered (*7*). Women who use contraceptive methods other than condoms should be counseled about the use of condoms and the risk for STIs (*7*). Additional information about prevention and treatment of STIs is available from CDC's *Sexually Transmitted Diseases Treatment Guidelines* (http://www.cdc.gov/std/treatment) (*7*).

Contraceptive Method Effectiveness

Contraceptive method effectiveness is critically important in minimizing the risk for unintended pregnancy, particularly among women for whom an unintended pregnancy would pose additional health risks. The effectiveness of contraceptive methods depends both on the inherent effectiveness of the method itself and on how consistently and correctly it is used (Table 1). Methods that depend on consistent and correct use have a wide range of effectiveness.

Unintended Pregnancy and Increased Health Risk

For women with conditions that may make unintended pregnancy an unacceptable health risk, long-acting, highly effective contraceptive methods may be the best choice (Table 1). Women with these conditions should be advised that sole use of barrier methods for contraception and behavior-based methods of contraception may not be the most appropriate choice because of their relatively higher typical-use rates of failure (Table 1). Conditions included in the U.S. MEC for which unintended pregnancy presents an unacceptable health risk are identified throughout the document (Box 2).

Keeping Guidance Up to Date

As with any evidence-based guidance document, a key challenge is keeping the recommendations up to date as new scientific evidence becomes available. CDC will continue to work with WHO to identify and assess all new relevant evidence and to determine whether changes to the recommendations are warranted (*4*). In most cases, the U.S. MEC will follow any updates in the WHO guidance, which typically occur every 3–4 years (or sooner if warranted by new data). However, CDC will review any WHO updates for their application in the United States. CDC also will identify and assess any new literature for the recommendations and medical conditions that are not included in the WHO guidance. CDC will completely review the U.S. MEC every 3–4 years as well. Updates to the guidance will appear on the CDC U.S. MEC website: http://www.cdc.gov/reproductivehealth/UnintendedPregnancy/USMEC.htm.

TABLE 1. Percentage of women experiencing an unintended pregnancy during the first year of typical use and the first year of perfect use of contraception and the percentage continuing use at the end of the first year — United States

Method	Women experiencing an unintended pregnancy within the first year of use		Women continuing use at 1 year[§]
	Typical use*	Perfect use[†]	
No method[¶]	85%	85%	
Spermicides[**]	29%	18%	42%
Withdrawal	27%	4%	43%
Fertility awareness–based methods	25%		51%
Standard Days method[††]		5%	
TwoDay method™[††]		4%	
Ovulation method[††]		3%	
Sponge			
Parous women	32%	20%	46%
Nulliparous women	16%	9%	57%
Diaphragm[§§]	16%	6%	57%
Condom[¶¶]			
Female (Reality®)	21%	5%	49%
Male	15%	2%	53%
Combined pill and progestin-only pill	8%	0.3%	68%
Evra patch®	8%	0.3%	68%
NuvaRing®	8%	0.3%	68%
Depo-Provera®	3%	0.3%	56%
Intrauterine device			
ParaGard® (copper T)	0.8%	0.6%	78%
Mirena® (LNG-IUS)	0.2%	0.2%	80%
Implanon®	0.05%	0.05%	84%
Female sterilization	0.5%	0.5%	100%
Male sterilization	0.15%	0.10%	100%
Emergency contraceptive pills[***]	Not applicable	Not applicable	Not applicable
Lactational amenorrhea methods[†††]	Not applicable	Not applicable	Not applicable

Adapted from Trussell J. Contraceptive efficacy. In Hatcher RA, Trussell J, Nelson AL, Cates W, Stewart FH, Kowal D. Contraceptive technology. 19th revised ed. New York, NY: Ardent Media; 2007.

* Among typical couples who initiate use of a method (not necessarily for the first time), the percentage who experience an unintended pregnancy during the first year if they do not stop use for any other reason. Estimates of the probability of pregnancy during the first year of typical use for spermicides, withdrawal, fertility awareness-based methods, the diaphragm, the male condom, the pill, and Depo-Provera are taken from the 1995 National Survey of Family Growth corrected for underreporting of abortion; see the text for the derivation of estimates for the other methods.

† Among couples who initiate use of a method (not necessarily for the first time) and who use it *perfectly* (both consistently and correctly), the percentage who experience an unintended pregnancy during the first year if they do not stop use for any other reason. See the text for the derivation of the estimate for each method.

§ Among couples attempting to avoid pregnancy, the percentage who continue to use a method for 1 year.

¶ The percentages becoming pregnant in the typical use and perfect use columns are based on data from populations where contraception is not used and from women who cease using contraception to become pregnant. Of these, approximately 89% become pregnant within 1 year. This estimate was lowered slightly (to 85%) to represent the percentage who would become pregnant within 1 year among women now relying on reversible methods of contraception if they abandoned contraception altogether.

** Foams, creams, gels, vaginal suppositories, and vaginal film.

†† The TwoDay and Ovulation methods are based on evaluation of cervical mucus. The Standard Days method avoids intercourse on cycle days 8–19.

§§ With spermicidal cream or jelly.

¶¶ Without spermicides.

*** Treatment initiated within 72 hours after unprotected intercourse reduces the risk for pregnancy by at least 75%. The treatment schedule is 1 dose within 120 hours after unprotected intercourse and a second dose 12 hours after the first dose. Both doses of Plan B can be taken at the same time. Plan B (1 dose is 1 white pill) is the only dedicated product specifically marketed for emergency contraception. The Food and Drug Administration has in addition declared the following 22 brands of oral contraceptives to be safe and effective for emergency contraception: Ogestrel or Ovral (1 dose is 2 white pills); Levlen or Nordette (1 dose is 4 light-orange pills); Cryselle, Levora, Low-Ogestrel, Lo/Ovral, or Quasence (1 dose is 4 white pills); Tri-Levlen or Triphasil (1 dose is 4 yellow pills); Jolessa, Portia, Seasonale, or Trivora (1 dose is 4 pink pills); Seasonique (1 dose is 4 light blue-green pills); Empresse (1 dose is 4 orange pills); Alesse, Lessina, or Levlite (1 dose is 5 pink pills); Aviane (1 dose is 5 orange pills); and Lutera (1 dose is 5 white pills).

††† Lactational amenorrhea method is a highly effective *temporary* method of contraception. However, to maintain effective protection against pregnancy, another method of contraception must be used as soon as menstruation resumes, the frequency or duration of breastfeeding is reduced, bottle feeds are introduced, or the baby reaches 6 months of age.

BOX 2. Conditions associated with increased risk for adverse health events as a result of unintended pregnancy

- Breast cancer
- Complicated valvular heart disease
- Diabetes: insulin-dependent; with nephropathy/retinopathy/neuropathy or other vascular disease; or of >20 years' duration
- Endometrial or ovarian cancer
- Epilepsy
- Hypertension (systolic >160 mm Hg or diastolic >100 mm Hg)
- History of bariatric surgery within the past 2 years
- HIV/AIDS
- Ischemic heart disease
- Malignant gestational trophoblastic disease
- Malignant liver tumors (hepatoma) and hepatocellular carcinoma of the liver
- Peripartum cardiomyopathy
- Schistosomiasis with fibrosis of the liver
- Severe (decompensated) cirrhosis
- Sickle cell disease
- Solid organ transplantation within the past 2 years
- Stroke
- Systemic lupus erythematosus
- Thrombogenic mutations
- Tuberculosis

Acknowledgements

This report is based in part on the work of the Promoting Family Planning Team, Department of Reproductive Health and Research, World Health Organization, and its development of the *WHO Medical Eligibility Criteria for Contraceptive Use*, 4th edition.

References

1. WHO. Medical eligibility criteria for contraceptive use. 4th ed. Geneva: WHO; 2009. Available at http://www.who.int/reproductivehealth/publications/family_planning/9789241563888/en/index.html.
2. Faculty of Family Planning and Reproductive Health Care, Royal College of Obstetricians and Gynecologists. UK medical eligibility criteria for contraceptive use, 2005–2006. London: Faculty of Family Planning and Reproductive Health Care, 2006.
3. Stroup DF, Berlin JA, Morton SC, et al. Meta-analysis of observational studies in epidemiology: a proposal for reporting. Meta-analysis Of Observational Studies in Epidemiology (MOOSE) group. JAMA 2000;283:2008–12.
4. Mohllajee AP, Curtis KM, Flanagan RG, et al. Keeping up with evidence a new system for WHO's evidence-based family planning guidance. Am J Prev Med 2005;28:483–90.
5. Harris RP, Helfand M, Woolf SH, et al. Current methods of the US Preventive Services Task Force: a review of the process. Am J Prev Med 2001;20:21–35.
6. CDC. Condom fact sheet in brief. Available at http://www.cdc.gov/condomeffectiveness/docs/Condom_fact_Sheet_in_Brief.pdf.
7. CDC. Sexually transmitted diseases treatment guidelines, 2006. MMWR 2006;55(RR No. 11).

Appendix A

Summary of Changes to the World Health Organization Medical Eligibility Criteria for Contraceptive Use, 4th Edition, to Create the U.S. Medical Eligibility Criteria for Contraceptive Use, 2010

The classification additions, deletions, and modifications from the World Health Organization (WHO) Medical Eligibility Criteria for Contraceptive Use, 4th Edition, are summarized below (Tables 1–3). For conditions for which classification changed for ≥1 methods or the condition description underwent a major modification, WHO conditions and recommendations appear in curly brackets.

BOX. Categories for Classifying Hormonal Contraceptives and Intrauterine Devices

1 = A condition for which there is no restriction for the use of the contraceptive method.
2 = A condition for which the advantages of using the method generally outweigh the theoretical or proven risks.
3 = A condition for which the theoretical or proven risks usually outweigh the advantages of using the method.
4 = A condition that represents an unacceptable health risk if the contraceptive method is used.

TABLE 1. Summary of changes in classifications from WHO Medical Eligibility Criteria for Contraceptive Use, 4th edition*†

Condition	COC/P/R	POP	DMPA	Implants	LNG-IUD	Cu-IUD	Clarification
Breastfeeding							
a. <1 mo postpartum {WHO: <6 wks postpartum}	3§ {4}	2§ {3}	2§ {3}	2§ {3}			The US Department of Health and Human Services recommends that infants be exclusively breastfed during the first 4–6 months of life, preferably for a full 6 months. Ideally, breastfeeding should continue through the first year of life (*1*). {Not included in WHO MEC}
b. 1 mo to <6 mos {WHO: ≥6 wks to <6 mos postpartum}	2§ {3}						
Postpartum (in breastfeeding or nonbreastfeeding women), including post caesarean section							
a. <10 min after delivery of the placenta {WHO: <48 hrs, including insertion immediately after delivery of the placenta}					2 {1 if not breastfeeding and 3 if breastfeeding}		
b. 10 min after delivery of the placenta to <4 wks {WHO: ≥48 hrs to <4 wks}					2 {3}	2{3}	
Deep venous thrombosis (DVT)/pulmonary embolism (PE)							
a. History of DVT/PE, not on anticoagulant therapy							
ii. Lower risk for recurrent DVT/PE (no risk factors)	3 {4}						
b. Acute DVT/PE		2 {3}	2 {3}	2 {3}	2 {3}	2 {1}	
c. DVT/PE and established on anticoagulant therapy for at least 3 mos							

TABLE 1. (*Continued*) Summary of changes in classifications from WHO Medical Eligibility Criteria for Contraceptive Use, 4th edition*†

Condition	COC/P/R	POP	DMPA	Implants	LNG-IUD	Cu-IUD	Clarification
i. Higher risk for recurrent DVT/PE (≥1 risk factors) • Known thrombophilia, including antiphospholipid syndrome • Active cancer (metastatic, on therapy, or within 6 mos after clinical remission), excluding non-melanoma skin cancer • History of recurrent DVT/PE						2 {1}	
ii. Lower risk for recurrent DVT/PE (no risk factors)	3§ {4}					2 {1}	Women on anticoagulant therapy are at risk for gynecologic complications of therapy such as hemorrhagic ovarian cysts and severe menorrhagia. Hormonal contraceptive methods can be of benefit in preventing or treating these complications. When a contraceptive method is used as a therapy, rather than solely to prevent pregnancy, the risk/benefit ratio may be different and should be considered on a case-by-case basis. {Not included in WHO MEC}
Valvular heart disease b. Complicated¶ (pulmonary hypertension, risk for atrial fibrillation, history of subacute bacterial endocarditis)					1 {2}	1 {2}	
Ovarian cancer¶					1 {Initiation = 3, Continuation = 2}	1 {Initiation = 3, Continuation = 2}	
Uterine fibroids					2 {1 if no uterine distortion and 4 if uterine distortion is present}	2 {1 if no uterine distortion and 4 if uterine distortion is present}	

* For conditions for which classification changed for ≥1 methods or the condition description underwent a major modification, WHO conditions and recommendations appear in curly brackets.

† Abbreviations: WHO = World Health Organization; COC = combined oral contraceptive; P = combined hormonal contraceptive patch; R = combined hormonal vaginal ring; POP = progestin-only pill; DMPA = depot medroxyprogesterone acetate; LNG-IUD = levonorgestrel-releasing intrauterine device; Cu-IUD = copper intrauterine device; DVT = deep venous thrombosis; PE = pulmonary embolism; VTE = venous thromboembolism.

§ Consult the clarification column for this classification.

¶ Condition that exposes a women to increased risk as a result of unintended pregnancy.

TABLE 2. Summary of recommendations for medical conditions added to the U.S. Medical Eligibility Criteria for Contraceptive Use*

Condition	COC/P/R	POP	DMPA	Implants	LNG-IUD		Cu-IUD		Clarification
					Initiation	Continuation	Initiation	Continuation	
History of bariatric surgery†									
a. Restrictive procedures: decrease storage capacity of the stomach (vertical banded gastroplasty, laparoscopic adjustable gastric band, laparoscopic sleeve gastrectomy)	1	1	1	1	1		1		
b. Malabsorptive procedures: decrease absorption of nutrients and calories by shortening the functional length of the small intestine (Roux-en-Y gastric bypass, biliopancreatic diversion)	COCs: 3 P/R: 1	3	1	1	1		1		
Peripartum cardiomyopathy†									
a. Normal or mildly impaired cardiac function (New York Heart Association Functional Class I or II: patients with no limitation of activities or patients with slight, mild limitation of activity) (*2*)									
i <6 mos	4	1	1	1	2		2		
ii ≥6 mos	3	1	1	1	2		2		
b. Moderately or severely impaired cardiac function (New York Heart Association Functional Class III or IV: patients with marked limitation of activity or patients who should be at complete rest) (*2*)	4	2	2	2	2		2		
Rheumatoid arthritis									
a. On immunosuppressive therapy	2	1	2/3§	1	2	1	2	1	DMPA use among women on long-term corticosteroid therapy with a history of, or risk factors for, nontraumatic fractures is classified as Category 3. Otherwise, DMPA use for women with rheumatoid arthritis is classified as Category 2.
b. Not on immunosuppressive therapy	2	1	2	1	1		1		
Endometrial hyperplasia	1	1	1	1	1		1		
Inflammatory bowel disease (IBD) (ulcerative colitis, Crohn disease)	2/3§	2	2	1	1		1		For women with mild IBD, with no other risk factors for VTE, the benefits of COC/P/R use generally outweigh the risks (Category 2). However, for women with IBD with increased risk for VTE (e.g., those with active or extensive disease, surgery, immobilization, corticosteroid use, vitamin deficiencies, fluid depletion), the risks for COC/P/R use generally outweigh the benefits (Category 3).
Solid organ transplantation†									
a. Complicated: graft failure (acute or chronic), rejection, cardiac allograft vasculopathy	4	2	2	2	3	2	3	2	
b. Uncomplicated	2§	2	2	2	2		2		Women with Budd-Chiari syndrome should not use COC/P/R because of the increased risk for thrombosis.

* Abbreviations: COC = combined oral contraceptive; P = combined hormonal contraceptive patch; R = combined hormonal vaginal ring: POP = progestin-only pill; DMPA = depot medroxyprogesterone acetate; LNG-IUD = levonorgestrel-releasing intrauterine device; Cu-IUD = copper intrauterine device; IBD = inflammatory bowel disease; VTE = venous thromboembolism.

† Condition that exposes a women to increased risk as a result of unintended pregnancy.

§ Consult the clarification column for this classification.

TABLE 3. Summary of additional changes to the U.S. Medical Eligibility Criteria for Contraceptive Use

Condition/Contraceptive method	Change
Emergency contraceptive pills	History of bariatric surgery, rheumatoid arthritis, inflammatory bowel disease, and solid organ transplantation were added to Appendix D and given a Category 1.
Barrier methods	For 6 conditions—history of bariatric surgery, peripartum cardiomyopathy, rheumatoid arthritis, endometrial hyperplasia, inflammatory bowel disease, and solid organ transplantation—the barrier methods are classified as Category 1.
Sterilization	In general, no medical conditions would absolutely restrict a person's eligibility for sterilization. Recommendations from the World Health Organization (WHO) Medical Eligibility Criteria for Contraceptive Use about specific settings and surgical procedures for sterilization are not included here. The guidance has been replaced with general text on sterilization.
Other deleted items	Guidance for combined injectables, levonorgestrel implants, and norethisterone enanthate has been removed because these methods are not currently available in the United States. Guidance for "blood pressure measurement unavailable" and "history of hypertension, where blood pressure CANNOT be evaluated (including hypertension in pregnancy)" has been removed.
Unintended pregnancy and increased health risk	The following conditions have been added to the WHO list of conditions that expose a woman to increased risk as a result of unintended pregnancy: history of bariatric surgery within the past 2 years, peripartum cardiomyopathy, and receiving a solid organ transplant within 2 years.

References

1. Office on Women's Health, US Department of Health and Human Services. HHS blueprint for action on breastfeeding. Washington, DC: US Department of Health and Human Services, Office on Women's Health; 2000.
2. The Criteria Committee of the New York Heart Association. Nomenclature and criteria for diagnosis of diseases of the heart and great vessels. 9th ed. Boston, MA: Little, Brown & Co; 1994.

Appendix B
Classifications for Combined Hormonal Contraceptives

Combined hormonal contraceptives (CHCs) include low-dose (containing ≤35 μg ethinyl estradiol [EE]) combined oral contraceptives (COCs), the combined hormonal patch, and the combined vaginal ring. The combined hormonal patch and vaginal ring are relatively new contraceptive methods. Limited information is available about the safety of these methods among women with specific medical conditions. Moreover, epidemiologic data on the long-term effects of the combined hormonal patch and the vaginal ring were not available for review. Evidence indicates that the combined hormonal patch and the combined vaginal ring provide comparable safety and pharmacokinetic profiles to COCs with similar hormone formulations (*1–33*). Pending further studies, the evidence available for recommendations about COCs applies to the recommendations for the combined hormonal patch and vaginal ring. Therefore, the patch and ring should have the same categories (Box) as COCs, except where noted. The assigned categories should, therefore, be considered a preliminary, best judgement, which will be reevaluated as new data become available. CHCs do not protect against sexually transmitted infections (STIs) or human immunodeficiency virus (HIV).

BOX. Categories for Classifying Combined Hormonal Contraceptives

1 = A condition for which there is no restriction for the use of the contraceptive method.
2 = A condition for which the advantages of using the method generally outweigh the theoretical or proven risks.
3 = A condition for which the theoretical or proven risks usually outweigh the advantages of using the method.
4 = A condition that represents an unacceptable health risk if the contraceptive method is used.

TABLE. Classifications for combined hormonal contraceptives, including pill, patch, and ring*†

Condition	Category	Clarifications/Evidence/Comments
Personal Characteristics and Reproductive History		
Pregnancy	Not applicable	**Clarification:** Use of COCs, P, or R is not required. There is no known harm to the woman, the course of her pregnancy, or the fetus if COCs, P, or R are inadvertently used during pregnancy.
Age		
a. Menarche to <40 yrs	1	**Evidence:** Adolescents using 20 μg EE-containing COCs have lower BMD than do nonusers, and higher dose-containing COCs have little to no effect. (*34–41*). In premenopausal adult women, COC use has little to no effect on bone health while appearing to preserve bone mass in perimenopausal women (*26,42–90*). Postmenopausal women who have ever used COCs have similar BMD to postmenopausal women who have never used COCs (*54,58,68,81,91–110*). BMD in adolescent or premenopausal women may not accurately predict postmenopausal fracture risk (*109,111–122*).
b. ≥40 yrs	2	**Comment:** The risk for cardiovascular disease increases with age and might increase with CHC use. In the absence of other adverse clinical conditions, CHCs can be used until menopause.
Parity		
a. Nulliparous	1	
b. Parous	1	
Breastfeeding		**Clarification:** The U.S. Department of Health and Human Services recommends that infants be exclusively breastfed during the first 4–6 months of life, preferably for a full 6 months. Ideally, breastfeeding should continue through the first year of life (*123*).
a. <1 mo postpartum	3	
b. 1 mo to <6 mos postpartum	2	**Evidence:** Clinical studies demonstrate conflicting results about effects on milk volume in women exposed to COCs during lactation; no consistent effect on infant weight has been reported. Adverse health outcomes or manifestations of exogenous estrogen in infants exposed to CHCs through breast milk have not been demonstrated (*124–133*). In general, these studies are of poor quality, lack standard definitions of breastfeeding or outcome measures, and have not included premature or ill infants. Theoretical concerns about effects of CHCs on breast milk production are greater in the early postpartum period when milk flow is being established.
c. ≥6 mos postpartum	2	

TABLE. (*Continued*) Classifications for combined hormonal contraceptives, including pill, patch, and ring*†

Condition	Category	Clarifications/Evidence/Comments
Postpartum (in nonbreastfeeding women)		
a. <21 days	3	**Comment:** Theoretical concern exists about the association between CHC use up to 3 weeks postpartum and risk for thrombosis in the mother. Blood coagulation and fibrinolysis are essentially normalized by 3 weeks postpartum.
b. ≥21 days	1	
Postabortion		**Clarification:** COCs, P, or R may be started immediately postabortion.
a. First trimester	1	**Evidence:** Women who started taking COCs immediately after first trimester medical or surgical abortion did not experience more side effects or adverse vaginal bleeding outcomes or clinically significant changes in coagulation parameters than did women who used a placebo, an IUD, a nonhormonal contraceptive method, or delayed COC initiation (*134–140*). Limited evidence on women using the ring immediately after first trimester medical or surgical abortion found no serious adverse events and no infection related to use of the combined vaginal contraceptive ring during 3 cycles of follow-up postabortion (*141*).
b. Second trimester	1	
c. Immediate postseptic abortion	1	
Past ectopic pregnancy	1	**Comment:** The risk for future ectopic pregnancy is increased among women who have had an ectopic pregnancy in the past. CHCs protect against pregnancy in general, including ectopic gestation.
History of pelvic surgery	1	
Smoking		
a. Age <35 yrs	2	**Evidence:** COC users who smoked were at increased risk for cardiovascular diseases, especially myocardial infarction, than those who did not smoke. Studies also showed an increased risk for myocardial infarction with increasing number of cigarettes smoked per day (*142–153*).
b. Age ≥35 yrs		
i. <15 Cigarettes/day	3	
ii. ≥15 Cigarettes/day	4	
Obesity		
a. ≥30 kg/m^2 BMI	2	**Evidence:** Obese women who use COCs are more likely than obese women who do not use COCs to experience VTE. The absolute risk for VTE in healthy women of reproductive age is small. Limited evidence suggests that obese women who use COCs do not have a higher risk for acute myocardial infarction or stroke than do obese nonusers (*147,153–159*). Limited evidence is inconsistent about whether COC effectiveness varies by body weight or BMI (*160–165*). Limited evidence suggests obese women are no more likely to gain weight after 3 cycles of the vaginal ring or COC than overweight or normal weight women. A similar weight gain during the 3 months was noted between the COC group and the vaginal ring group across all BMI categories (*166*). The effectiveness of the patch decreased among women who weighed >90 kg; however, no association was found between pregnancy risk and BMI (*18*).
b. Menarche to <18 yrs and ≥30 kg/m^2 BMI	2	
History of bariatric surgery§		
a. Restrictive procedures: decrease storage capacity of the stomach (vertical banded gastroplasty, laparoscopic adjustable gastric band, laparoscopic sleeve gastrectomy)	1	**Evidence:** Limited evidence demonstrated no substantial decrease in effectiveness of oral contraceptives among women who underwent laparoscopic placement of an adjustable gastric band (*167*).
b. Malabsorptive procedures: decrease absorption of nutrients and calories by shortening the functional length of the small intestine (Roux-en-Y gastric bypass, biliopancreatic diversion)	COCs: 3 P/R: 1	**Evidence:** Limited evidence demonstrated no substantial decrease in effectiveness of oral contraceptives among women who underwent a biliopancreatic diversion (*168*); however, evidence from pharmacokinetic studies reported conflicting results of oral contraceptive effectiveness among women who underwent a jejunoileal bypass (*169,170*). **Comment:** Bariatric surgical procedures involving a malabsorptive component have the potential to decrease oral contraceptive effectiveness, perhaps further decreased by postoperative complications, such as long-term diarrhea and/or vomiting.
Cardiovascular Disease		
Multiple risk factors for arterial cardiovascular disease (such as older age, smoking, diabetes, and hypertension)	3/4	**Clarification:** When a woman has multiple major risk factors, any of which alone would substantially increase her risk for cardiovascular disease, use of COCs, P, or R might increase her risk to an unacceptable level. However, a simple addition of categories for multiple risk factors is not intended; for example, a combination of two risk factors assigned a category 2 might not necessarily warrant a higher category.
Hypertension		
For all categories of hypertension, classifications are based on the assumption that no other risk factors exist for cardiovascular disease. When multiple risk factors do exist, risk for cardiovascular disease might increase substantially. A single reading of blood pressure level is not sufficient to classify a woman as hypertensive.		
a. Adequately controlled hypertension	3	**Clarification:** Women adequately treated for hypertension are at reduced risk for acute myocardial infarction and stroke compared with untreated women. Although no data exist, COC, P, or R users with adequately controlled and monitored hypertension should be at reduced risk for acute myocardial infarction and stroke compared with untreated hypertensive COC, P, or R users.
b. Elevated blood pressure levels (properly taken measurements)		

TABLE. (*Continued*) Classifications for combined hormonal contraceptives, including pill, patch, and ring*†

Condition	Category	Clarifications/Evidence/Comments
i. Systolic 140–159 mm Hg or diastolic 90–99 mm Hg	3	**Evidence:** Among women with hypertension, COC users were at higher risk than nonusers for stroke, acute myocardial infarction, and peripheral arterial disease (*142,144,151–153,155,171–186*).
ii. Systolic ≥160 mm Hg or diastolic ≥100 mm Hg§	4	Discontinuation of COCs in women with hypertension might improve blood pressure control (*187*).
c. Vascular disease	4	
History of high blood pressure during pregnancy (where current blood pressure is measurable and normal)	2	**Evidence:** Women with a history of high blood pressure in pregnancy, who also used COCs, had a higher risk for myocardial infarction and VTE than did COC users who did not have a history of high blood pressure during pregnancy. The absolute risks for acute myocardial infarction and VTE in this population remained small (*153,172,184–186,188–193*).
Deep venous thrombosis (DVT)/ Pulmonary embolism (PE)		
a. History of DVT/PE, not on anticoagulant therapy		
i. Higher risk for recurrent DVT/PE (≥1 risk factors)	4	
• History of estrogen-associated DVT/PE		
• Pregnancy-associated DVT/PE		
• Idiopathic DVT/PE		
• Known thrombophilia, including antiphospholipid syndrome		
• Active cancer (metastatic, on therapy, or within 6 mos after clinical remission), excluding non-melanoma skin cancer		
• History of recurrent DVT/PE		
ii. Lower risk for recurrent DVT/PE (no risk factors)	3	
b. Acute DVT/PE	4	
c. DVT/PE and established on anticoagulant therapy for at least 3 mos		
i. Higher risk for recurrent DVT/PE (≥1 risk factors)	4	**Clarification:** Women on anticoagulant therapy are at risk for gynecologic complications of therapy, such as hemorrhagic ovarian cysts and severe menorrhagia. Hormonal contraceptive methods can be of benefit in preventing or treating these complications. When a contraceptive method is used as a therapy, rather than solely to prevent pregnancy, the risk/benefit ratio might differ and should be considered on a case-by-case basis.
• Known thrombophilia, including antiphospholipid syndrome		
• Active cancer (metastatic, on therapy, or within 6 mos after clinical remission), excluding non-melanoma skin cancer		
• History of recurrent DVT/PE		
ii. Lower risk for recurrent DVT/PE (no risk factors)	3	**Clarification:** Women on anticoagulant therapy are at risk for gynecologic complications of therapy, such as hemorrhagic ovarian cysts and severe menorrhagia. Hormonal contraceptive methods can be of benefit in preventing or treating these complications. When a contraceptive method is used as a therapy, rather than solely to prevent pregnancy, the risk/benefit ratio may differ and should be considered on a case-by-case basis.
d. Family history (first-degree relatives)	2	**Comment:** Some conditions that increase the risk for DVT/PE are heritable.
e. Major surgery		
i. With prolonged immobilization	4	
ii. Without prolonged immobilization	2	
f. Minor surgery without immobilization	1	
Known thrombogenic mutations§ (e.g., factor V Leiden; prothrombin mutation; protein S, protein C, and antithrombin deficiencies)	4	**Clarification**: Routine screening is not appropriate because of the rarity of the conditions and the high cost of screening. **Evidence:** Among women with thrombogenic mutations, COC users had a 2-fold to 20-fold higher risk for thrombosis than did nonusers (*159,194–216*).
Superficial venous thrombosis		
a. Varicose veins	1	**Comment:** Varicose veins are not risk factors for DVT/PE
b. Superficial thrombophlebitis	2	
Current and history of ischemic heart disease§	4	
Stroke§ (history of cerebrovascular accident)	4	

TABLE. (*Continued*) Classifications for combined hormonal contraceptives, including pill, patch, and ring*†

Condition	Category	Clarifications/Evidence/Comments
Known hyperlipidemias	2/3	**Clarification:** Routine screening is not appropriate because of the rarity of the conditions and the high cost of screening. Although some types of hyperlipidemias are risk factors for vascular disease, the category should be assessed according to the type, its severity, and the presence of other cardiovascular risk factors.
Valvular heart disease		
a. Uncomplicated	2	
b. Complicated§ (pulmonary hypertension, risk for atrial fibrillation, history of subacute bacterial endocarditis)	4	**Comment:** Among women with valvular heart disease, CHC use may further increase the risk for arterial thrombosis; women with complicated valvular heart disease are at greatest risk.
Peripartum cardiomyopathy§		
a. Normal or mildly impaired cardiac function (New York Heart Association Functional Class I or II: patients with no limitation of activities or patients with slight, mild limitation of activity) (*217*)		**Evidence:** No direct evidence exists about the safety of COCs/P/R among women with peripartum cardiomyopathy. Limited indirect evidence from noncomparative studies of women with cardiac disease demonstrated few cases of hypertension and transient ischemic attack in women with cardiac disease using COCs. No cases of heart failure were reported (*218*). **Comment:** COCs might increase fluid retention in healthy women; fluid retention may worsen heart failure in women with peripartum cardiomyopathy. COCs might induce cardiac arrhythmias in healthy women; women with peripartum cardiomyopathy have a high incidence of cardiac arrhythmias.
i. <6 mos	4	
ii. ≥6 mos	3	
b. Moderately or severely impaired cardiac function (New York Heart Association Functional Class III or IV: patients with marked limitation of activity or patients who should be at complete rest) (*217*)	4	**Evidence:** No direct evidence exists about the safety of COCs/P/R among women with peripartum cardiomyopathy. Limited indirect evidence from noncomparative studies of women with cardiac disease demonstrated few cases of hypertension and transient ischemic attack in women with cardiac disease using COCs. No cases of heart failure were reported (*218*). **Comment:** COCs might increase fluid retention in healthy women; fluid retention may worsen heart failure in women with peripartum cardiomyopathy. COCs might induce cardiac arrhythmias in healthy women; women with peripartum cardiomyopathy have a high incidence of cardiac arrhythmias.
Rheumatic Diseases		
Systemic lupus erythematosus (SLE)§		
Persons with SLE are at increased risk for ischemic heart disease, stroke, and VTE. Categories assigned to such conditions in the MEC should be the same for women with SLE who present with these conditions. For all categories of SLE, classifications are based on the assumption that no other risk factors for cardiovascular disease are present; these classifications must be modified in the presence of such risk factors.		
Many women with SLE can be considered good candidates for most contraceptive methods, including hormonal contraceptives (*219–237*).		
a. Positive (or unknown) antiphospholipid antibodies	4	**Evidence:** Antiphospholipid antibodies are associated with a higher risk for both arterial and venous thrombosis (*238,239*).
b. Severe thrombocytopenia	2	
c. Immunosuppressive treatment	2	
d. None of the above	2	
Rheumatoid arthritis		
a. On immunosuppressive therapy	2	**Evidence:** Limited evidence shows no consistent pattern of improvement or worsening of rheumatoid arthritis with use of oral contraceptives (*240–245*), progesterone (*246*), or estrogen (*247*).
b. Not on immunosuppressive therapy	2	
Neurologic Conditions		
Headaches	Initiation / Continuation	**Clarification:** Classification depends on accurate diagnosis of those severe headaches that are migrainous and those headaches that are not. Any new headaches or marked changes in headaches should be evaluated. Classification is for women without any other risk factors for stroke. Risk for stroke increases with age, hypertension and smoking.
a. Non-migrainous (mild or severe)	1 / 2	
b. Migraine		**Evidence:** Among women with migraine, women who also had aura had a higher risk for stroke than did those without aura (*248–250*). Women with a history of migraine who use COCs are about 2–4 times as likely to have an ischemic stroke as nonusers with a history of migraine (*142,157,179,180,249-254*). **Comment:** Aura is a specific focal neurologic symptom. For more information about this and other diagnostic criteria, see: Headache Classification Subcommittee of the International Headache Society. The International Classification of Headache Disorders, 2nd ed. Cephalalgia. 2004;24(Suppl 1). Available http://www.i-h-s.org/upload/ct_clas/ihc_II_main_no_print.pdf.
i. Without aura		
• Age <35 yrs	2 / 3	
• Age ≥35 yrs	3 / 4	
ii. With aura, at any age	4 / 4	
Epilepsy§	1	**Clarification:** If a woman is taking anticonvulsants, refer to the section on drug interactions. Certain anticonvulsants lower COC effectiveness. The extent to which P or R use is similar to COC use in this regard remains unclear.

TABLE. (*Continued*) Classifications for combined hormonal contraceptives, including pill, patch, and ring*†

Condition	Category	Clarifications/Evidence/Comments
Depressive Disorders		
Depressive disorders	1	**Clarification**: The classification is based on data for women with selected depressive disorders. No data on bipolar disorder or postpartum depression were available. Drug interactions potentially can occur between certain antidepressant medications and hormonal contraceptives. **Evidence**: COC use did not increase depressive symptoms in women with depression compared with baseline or with nonusers with depression (*255–264*).
Reproductive Tract Infections and Disorders		
Vaginal bleeding patterns		
a. Irregular pattern without heavy bleeding	1	**Comment:** Irregular menstrual bleeding patterns are common among healthy women.
b. Heavy or prolonged bleeding (includes regular and irregular patterns)	1	**Clarification:** Unusually heavy bleeding should raise suspicion of a serious underlying condition. **Evidence:** A Cochrane Collaboration Review identified 1 randomized controlled trial evaluating the effectiveness of COC use compared with naproxen and danazol in treating menorrhagic women. Women with menorrhagia did not report worsening of the condition or any adverse events related to COC use (*265*).
Unexplained vaginal bleeding (suspicious for serious condition)		
Before evaluation	2	**Clarification:** If pregnancy or an underlying pathological condition (such as pelvic malignancy) is suspected, it must be evaluated and the category adjusted after evaluation. **Comment:** No conditions that cause vaginal bleeding will be worsened in the short term by use of CHCs.
Endometriosis	1	**Evidence:** A Cochrane Collaboration Review identified 1 randomized controlled trial evaluating the effectiveness of COC use compared with a gonadotropin-releasing hormone analogue in treating the symptoms of endometriosis. Women with endometriosis did not report worsening of the condition or any adverse events related to COC use (*266*).
Benign ovarian tumors (including cysts)	1	
Severe dysmenorrhea	1	**Evidence:** Risk for side effects with COC use was not higher among women with dysmenorrhea than among women not using COCs. Some COC users had a reduction in pain and bleeding (*267,268*).
Gestational trophoblastic disease		
a. Decreasing or undetectable β–hCG levels	1	**Evidence:** After molar pregnancy evacuation, the balance of evidence found COC use did not increase the risk for postmolar trophoblastic disease, and β-hCG levels regressed more rapidly in some COC users than in nonusers (*269–275*). Limited evidence suggests that use of COCs during chemotherapy does not significantly affect the regression or treatment of postmolar trophoblastic disease compared with women who used a nonhormonal contraceptive method or DMPA during chemotherapy (*276*).
b. Persistently elevated β-hCG levels or malignant disease§	1	
Cervical ectropion	1	**Comment:** Cervical ectropion is not a risk factor for cervical cancer, and restriction of CHC use is unnecessary.
Cervical intraepithelial neoplasia	2	**Evidence:** Among women with persistent HPV infection, long-term COC use (≥5 years) might increase the risk for carcinoma in situ and invasive carcinoma (*21,277*). Limited evidence on women with low-grade squamous intraepithelial lesions found use of the vaginal ring did not worsen the condition (*21*).
Cervical cancer (awaiting treatment)	2	**Comment:** Theoretical concern exists that CHC use might affect prognosis of the existing disease. While awaiting treatment, women may use CHCs. In general, treatment of this condition can render a woman sterile.
Breast Disease		
a. Undiagnosed mass	2	**Clarification:** The woman should be evaluated as early as possible.
b. Benign breast disease	1	
c. Family history of cancer	1	**Evidence:** Women with breast cancer susceptibility genes (such as *BRCA1* and *BRCA2*) have a higher baseline risk for breast cancer than do women without these genes. The baseline risk for breast cancer is also higher among women with a family history of breast cancer than among those who do not have such a history. However, current evidence does not suggest that the increased risk for breast cancer among women with either a family history of breast cancer or breast cancer susceptibility genes is modified by the use of COCs (*278–295*).
d. Breast cancer§		
i. Current	4	**Comment:** Breast cancer is a hormonally sensitive tumor, and the prognosis for women with current or recent breast cancer might worsen with CHC use.
ii. Past and no evidence of current disease for 5 yrs	3	
Endometrial hyperplasia	1	
Endometrial cancer§	1	**Comment:** COC use reduces the risk for endometrial cancer; whether P or R use reduces the risk for endometrial cancer is not known. While awaiting treatment, women may use COCs, P, or R. In general, treatment of this condition renders a woman sterile.

TABLE. (*Continued*) Classifications for combined hormonal contraceptives, including pill, patch, and ring*†

Condition	Category	Clarifications/Evidence/Comments
Ovarian cancer§	1	**Comment:** COC use reduces the risk for ovarian cancer; whether P or R use reduces the risk for ovarian cancer is not known. While awaiting treatment, women may use COCs, P, or R. In general, treatment of this condition can render a woman sterile.
Uterine fibroids	1	**Comment:** COCs do not appear to cause growth of uterine fibroids, and P and R also are not expected to cause growth.
Pelvic inflammatory disease (PID)		
a. Past PID (assuming no current risk factors for STIs)		**Comment:** COCs might reduce the risk for PID among women with STIs but do not protect against HIV or lower genital tract STIs. Whether use of P or R reduces the risk for PID among women with STIs is unknown, but they do not protect against HIV or lower genital tract STIs.
i. With subsequent pregnancy	1	
ii. Without subsequent pregnancy	1	
b. Current PID	1	
STIs		
a. Current purulent cervicitis or chlamydial infection or gonorrhea	1	
b. Other STIs (excluding HIV and hepatitis)	1	
c. Vaginitis (including *Trichomonas vaginalis* and bacterial vaginosis)	1	
d. Increased risk for STIs	1	**Evidence:** Evidence suggests that chlamydial cervicitis may be increased among COC users at high risk for STIs. For other STIs, there is either evidence of no association between COC use and STI acquisition or too limited evidence to draw any conclusions (*296–376*).
HIV/AIDS		
High risk for HIV	1	**Evidence:** The balance of the evidence suggests no association between oral contraceptive use and HIV acquisition, although findings from studies conducted among higher risk populations have been inconsistent (*377–415*).
HIV infection§	1	**Evidence:** Most studies suggest no increased risk for HIV disease progression with hormonal contraceptive use, as measured by changes in CD4 cell count, viral load, or survival. Studies observing that women with HIV who use hormonal contraception have increased risks of acquiring STIs are generally consistent with reports among uninfected women. One direct study found no association between hormonal contraceptive use and an increased risk for HIV transmission to uninfected partners; several indirect studies reported mixed results about whether hormonal contraception is associated with increased risk for HIV-1 DNA or RNA shedding from the genital tract (*377,416–432*).
AIDS§	1	**Clarification**: Drug interactions may occur between hormonal contraceptives and ARV therapy; refer to the section on drug interactions.
Other Infections		
Schistosomiasis		
a. Uncomplicated	1	**Evidence:** Among women with uncomplicated schistosomiasis, COC use had no adverse effects on liver function (*433–439*).
b. Fibrosis of liver§ (if severe, see cirrhosis)	1	
Tuberculosis§		**Clarification:** If a woman is taking rifampicin, refer to the section on drug interactions. Rifampicin is likely to decrease COC effectiveness. The extent to which P or R use is similar to COC use in this regard remains unclear.
a. Nonpelvic	1	
b. Pelvic	1	
Malaria	1	
Endocrine Conditions		
Diabetes		
a. History of gestational disease	1	**Evidence:** The development of noninsulin-dependant diabetes in women with a history of gestational diabetes is not increased by use of COCs (*440–447*). Likewise, lipid levels appear to be unaffected by COC use (*448–450*).
b. Nonvascular disease		**Evidence:** Among women with insulin- or noninsulin-dependent diabetes, COC use had limited effect on daily insulin requirements and no effect on long-term diabetes control (e.g., glycosylated hemoglobin levels) or progression to retinopathy. Changes in lipid profile and hemostatic markers were limited, and most changes remained within normal values (*451–460*).
i. Noninsulin-dependent	2	
ii. Insulin-dependent§	2	
c. Nephropathy/retinopathy/neuropathy§	3/4	**Clarification:** The category should be assessed according to the severity of the condition.
d. Other vascular disease or diabetes of >20 yrs' duration§	3/4	**Clarification:** The category should be assessed according to the severity of the condition.

TABLE. (*Continued*) Classifications for combined hormonal contraceptives, including pill, patch, and ring*†

Condition	Category		Clarifications/Evidence/Comments
Thyroid disorders			
a. Simple goiter	1		
b. Hyperthyroid	1		
c. Hypothyroid	1		
Gastrointestinal Conditions			
Inflammatory bowel disease (IBD) (ulcerative colitis, Crohn disease)	2/3		**Clarification:** For women with mild IBD and no other risk factor for VTE, the benefits of COC/P/R use generally outweigh the risks (Category 2). However, for women with IBD who are at increased risk for VTE (e.g., those with active or extensive disease, surgery, immobilization, corticosteroid use, vitamin deficiencies, or fluid depletion), the risks of COC/P/R use generally outweigh the benefits (Category 3). **Evidence:** Risk for disease relapse was not significantly higher among women with IBD using oral contraceptives (most studies did not specify formulation) than among nonusers (*461–465*). Absorption of COCs among women with mild ulcerative colitis and no or small ileal resections was similar to the absorption among healthy women (*466,467*). Findings might not apply to women with Crohn disease or more extensive bowel resections. No data exist that evaluate the increased risk for VTE among women with IBD using COCs/P/R. However, women with IBD are at higher risk than unaffected women for VTE (*468*).
Gallbladder disease			
a. Symptomatic			**Comment:** COCs, P, or R might cause a small increased risk for gallbladder disease. COCs, P, or R might worsen existing gallbladder disease.
i. Treated by cholecystectomy	2		
ii. Medically treated	3		
iii. Current	3		
b. Asymptomatic	2		
History of cholestasis			
a. Pregnancy-related	2		**Comment:** History of pregnancy-related cholestasis might predict an increased risk for COC-related cholestasis.
b. Past COC-related	3		**Comment:** History of COC-related cholestasis predicts an increased risk with subsequent COC use.
Viral hepatitis	Initiation	Continuation	
a. Acute or flare	3/4	2	**Clarification for initiation:** The category should be assessed according to the severity of the condition.
b. Carrier	1	1	**Evidence:** Data suggest that in women with chronic hepatitis, COC use does not increase the rate or severity of cirrhotic fibrosis, nor does it increase the risk for hepatocellular carcinoma (*469,470*). For women who are carriers, COC use does not appear to trigger liver failure or severe dysfunction (*471-473*). Evidence is limited for COC use during active hepatitis (*474*).
c. Chronic	1	1	
Cirrhosis			
a. Mild (compensated)	1		
b. Severe§ (decompensated)	4		
Liver tumors			
a. Benign			**Evidence:** Limited direct evidence suggests that hormonal contraceptive use does not influence either progression or regression of liver lesions among women with focal nodular hyperplasia (*475,476*).
i. Focal nodular hyperplasia	2		
ii. Hepatocellular adenoma§	4		
b. Malignant§ (hepatoma)	4		
Anemias			
Thalassemia	1		**Comment:** Anecdotal evidence from countries where thalassemia is prevalent indicates that COC use does not worsen the condition.
Sickle cell disease§	2		
Iron deficiency anemia	1		**Comment:** CHC use may decrease menstrual blood loss.
Solid Organ Transplantation			
Solid organ transplantation§			
a. Complicated: graft failure (acute or chronic), rejection, cardiac allograft vasculopathy	4		**Evidence:** Limited evidence of COC and P users indicated no overall changes in biochemical measures. However, one study reported discontinuations of COC use in 2 (8%) of 26 women as a result of serious medical complications, and in one case report, a woman developed cholestasis associated with high-dose COC use (*477–480*).
b. Uncomplicated	2		**Clarification:** Women with Budd-Chiari syndrome should not use COC/P/R because of the increased risk for thrombosis. **Evidence:** Limited evidence of COC and P users indicated no overall changes in biochemical measures. However, one study reported discontinuations of COC use in 2 (8%) of 26 women as a result of serious medical complications, and in one case report, a woman developed cholestasis associated with high-dose COC use (*477–480*).

TABLE. (*Continued*) Classifications for combined hormonal contraceptives, including pill, patch, and ring*†

Condition	Category	Clarifications/Evidence/Comments
Drug Interactions		
Antiretroviral (ARV) therapy		**Clarification:** ARV drugs have the potential to either decrease or increase the bioavailability of steroid hormones in hormonal contraceptives. Limited data (summarized in Appendix M) suggest potential drug interactions between many ARV drugs (particularly some non-NNRTIs and ritonavir-boosted protease inhibitors) and hormonal contraceptives. These interactions might alter the safety and effectiveness of both the hormonal contraceptive and the ARV drug. Thus, if a woman on ARV treatment decides to initiate or continue hormonal contraceptive use, the consistent use of condoms is recommended to both prevent HIV transmission and compensate for any possible reduction in the effectiveness of the hormonal contraceptive. When a COC is chosen, a preparation containing a minimum of 30 μg EE should be used.
a. Nucleoside reverse transcriptase inhibitors (NRTIs)	1	
b. Non-nucleoside reverse transcriptase inhibitors (NNRTIs)	2	
c. Ritonavir-boosted protease inhibitors	3	
Anticonvulsant therapy		**Clarification:** Although the interaction of certain anticonvulsants with COCs, P, or R is not harmful to women, it is likely to reduce the effectiveness of COCs, P, or R. Use of other contraceptives should be encouraged for women who are long-term users of any of these drugs. When a COC is chosen, a preparation containing a minimum of 30 μg EE should be used.
a. Certain anticonvulsants (phenytoin, carbamazepine, barbiturates, primidone, topiramate, oxcarbazepine)	3	**Evidence:** Use of certain anticonvulsants might decrease the effectiveness of COCs (*481–484*).
b. Lamotrigine	3	**Clarification:** The recommendation for lamotrigine applies only for situations where lamotrigine monotherapy is taken concurrently with COCs. Anticonvulsant treatment regimens that combine lamotrigine and nonenzyme-inducing antiepileptic drugs (such as sodium valproate) do not interact with COCs. **Evidence:** Pharmacokinetic studies show levels of lamotrigine decrease significantly during COC use (*485–489*). Some women who used both COCs and lamotrigine experienced increased seizure activity in one trial (*485*).
Antimicrobial therapy		
a. Broad-spectrum antibiotics	1	**Evidence:** Most broad-spectrum antibiotics do not affect the contraceptive effectiveness of COCs(*490–526*), P (*527*) or R (*528*).
b. Antifungals	1	**Evidence:** Studies of antifungal agents have shown no clinically significant pharmacokinetic interactions with COCs (*529–538*) or R (*539*).
c. Antiparasitics	1	**Evidence:** Studies of antiparasitic agents have shown no clinically significant pharmacokinetic interactions with COCs (*433,540–544*).
d. Rifampicin or rifabutin therapy	3	**Clarification:** Although the interaction of rifampicin or rifabutin therapy with COCs, P, or R is not harmful to women, it is likely to reduce the effectiveness of COCs, P, or R. Use of other contraceptives should be encouraged for women who are long-term users of either of these drugs. When a COC is chosen, a preparation containing a minimum of 30 μg EE should be used. **Evidence:** The balance of the evidence suggests that rifampicin reduces the effectiveness of COCs (*545–560*). Data on rifabutin are limited, but effects on metabolism of COCs are less than with rifampicin, and small studies have not shown evidence of ovulation (*547,554*).

* Abbreviations: STI = sexually transmitted infection; HIV = human immunodeficiency virus; COC = combined oral contraceptive; P = patch; R = ring; EE = ethinyl estradiol; BMD = bone mineral density; CHC = combined hormonal contraceptive; IUD = intrauterine device; VTE = venous thromboembolism; BMI = body mass index; DVT = deep venous thrombosis; PE = pulmonary embolism; SLE = systemic lupus erythematosus; MEC = Medical Eligibility Criteria; hCG = human chorionic gonadotropin; DMPA = depot medroxyprogesterone acetate; HPV = human papillomavirus; PID = pelvic inflammatory disease; AIDS = acquired immunodeficiency syndrome; ARV = antiretroviral; IBD = inflammatory bowel disease; NRTI = nucleoside reverse transcriptase inhibitor; NNRTI = non-nucleoside reverse transcriptase inhibitor.

† COCs/P/R do not protect against STI/HIV. If risk for STI/HIV (including during pregnancy or postpartum) exists, the correct and consistent use of condoms is recommended, either alone or with another contraceptive method. Consistent and correct use of the male latex condom reduces the risk for STI/HIV transmission.

§ Condition that exposes a woman to increased risk as a result of unintended pregnancy.

References

1. Abrams LS, Skee D, Natarajan J, Wong FA, Lasseter KC. Multiple-dose pharmacokinetics of a contraceptive patch in healthy women participants. Contraception 2001;64:287–94.
2. Audet M-C, Moreau M, Koltun WD, et al. Evaluation of contraceptive efficacy and cycle control of a transdermal contraceptive patch vs. an oral contraceptive: a randomized trial. JAMA 2001;285:2347–54.
3. Boonyarangkul A, Taneepanichskul S. Comparison of cycle control and side effects between transdermal contraceptive patch and an oral contraceptive in women older than 35 years. J Med Assoc Thai 2007;90:1715–9.
4. Burkman RT. The transdermal contraceptive patch: a new approach to hormonal contraception. Int J Fertil 2002;47:69–76.
5. Cole JA, Norman H, Doherty M, Walker AM. Venous thromboembolism, myocardial infarction, and stroke among transdermal contraceptive system users. Obstet Gynecol 2007;109:339–46.
6. Devineni D, Skee D, Vaccaro N, et al. Pharmacokinetics and pharmacodynamics of a transdermal contraceptive patch and an oral contraceptive. J Clin Pharmacol 2007;47:497–509.
7. Dittrich R, Parker L, Rosen JB, et al. Transdermal contraception: evaluation of three transdermal norelgestromin/ethinyl estradiol doses in a randomized, multicenter, dose-response study. Am J Obstet Gynecol 2002;186:15–20.
8. Helmerhorst FM, Cronje HS, Hedon B, et al. Comparison of efficacy, cycle control, compliance and safety in users of a contraceptive patch vs. an oral contraceptive. Int J Gynaecol Obstet 2000;70:78.
9. Jick S, Kaye J, Li L, Jick H. Further results on the risk of nonfatal venous thromboembolism in users of the contraceptive transdermal patch compared to users of oral contraceptives containing norgestimate and 35 μg of ethinyl estradiol. Contraception 2007;76:4–7.

10. Jick SS, Jick H. Cerebral venous sinus thrombosis in users of four hormonal contraceptives: levonorgestrel-containing oral contraceptives, norgestimate-containing oral contraceptives, desogestrel-containing oral contraceptives and the contraceptive patch. Contraception 2006;74:290–2.
11. Jick SS, Kaye J, Russmaann S, Jick H. Risk of nonfatal venous thromboembolism in women using a contraceptive transdermal patch and oral contraceptives containing norgestimate and 35 microg of ethinyl estradiol. Contraception 2006;73:223–8.
12. Jick SS, Jick H. The contraceptive patch in relation to ischemic stroke and acute myocardial infarction. Pharmacotherapy 2007;27:218–20.
13. Pierson RA, Archer DF, Moreau M, et al. Ortho EvraEvra versus oral contraceptives: follicular development and ovulation in normal cycles and after an intentional dosing error. Fertil Steril 2003;80:34–42.
14. Radowicki S, Skorzeqska K, Szlendak K. Safety evaluation of a transdermal contraceptive system with an oral contraceptive. Ginekologia Polska 2005;76:884–9.
15. Smallwood GH, Meador ML, Lenihan JP, et al. Efficacy and safety of a transdermal contraceptive system. Obstet Gynecol 2001;98:799–805.
16. Urdl W, Apter D, Alperstein A, et al. Contraceptive efficacy, compliance and beyond: factors related to satisfaction with once-weekly transdermal compared with oral contraception. Eur J Obstet Gynecol Reprod Biol 2005;121:202–10.
17. White T, Ozel B, Jain JK, Stanczyk FZ. Effects of transdermal and oral contraceptives on estrogen-sensitive hepatic proteins. Contraception 2006;74:293–6.
18. Zieman M, Guillebaud JG, Weisberg E, et al. Contraceptive efficacy and cycle control with the Ortho Evra/Evra transdermal system: the analysis of pooled data. Fertil Steril 2002;77:s13–s18.
19. Ahrendt HJ, Nisand I, Bastianelli C, et al. Efficacy, acceptability and tolerability of the combined contraceptive ring, NuvaRing, compared with an oral contraceptive containing 30 microg of ethinyl estradiol and 3 mg of drospirene. Contraception 2006;74:451–7.
20. Bjarnadottir RI, Tuppurainen M, Killick SR. Comparison of cycle control with a combined contraceptive vaginal ring and oral levonorgestrel/ethinyl estradiol. Am J Obstet Gynecol 2002;186:389–95.
21. Dieben T, Roumen FJ, Apter D. Efficacy, cycle control, and user acceptibility of a novel combined contraceptive vaginal ring. Obstet Gynecol 2002;100:585–93.
22. Duijkers I, Killick SR, Bigrigg A, et al. A comparative study on the effects of a contraceptive vaginal ring NuvaRing and an oral contraceptive on carbohydrate metabolism and adrenal and thyroid function. Eur J Contracept Reprod Health Care 2004;9:131–40.
23. Duijkers I, Klipping C, Verhoeven CH, et al. Ovarian function with the contraceptive vaginal ring or an oral contraceptive: a randomized study. Hum Reprod 2004;19:2668–73.
24. Elkind-Hirsch KE, Darensbourg C, Ogden B, et al. Contraceptive vaginal ring use for women has less adverse metabolic effets than an oral contraceptive. Contraception 2007;76:348–56.
25. Magnusdottir EM, Bjarnadottir RI, Onundarson PT, et al. The contraceptive vaginal ring (NuvaRing) and hemostasis: a comparative study. Contraception 2004;69:461–7.
26. Massai R, Makarainen L, Kuukankorpi A, Klipping C, Duijkers I, Dieben T. The combined contraceptive vaginal ring and bone mineral density in healthy pre-menopausal women. Hum Reprod 2005;20:2764–8.
27. Milsom I, Lete I, Bjertnaes A, et al. Effects on cycle control and bodyweight of the combined contraceptive ring, NuvaRing, versus an oral contraceptive containing 30 microg ethinyl estradiol and 3 mg drospirenone. Hum Reprod 2006;21:2304–11.
28. Oddsson K, Leifels-Fischer B, de Melo NR, et al. Efficacy and safety of a contraceptive vaginal ring (NuvaRing) compared with a combined oral contraceptive: a 1-year randomized trial. Contraception 2005;71:176–82.
29. Sabatini R, Cagiano R. Comparison profiles of cycle control, side effects and sexual satisfaction of three hormonal contraceptives. Contraception 2006;74:220–3.
30. Timmer CJ, Mulders TM. Pharmacokinetics of etonogestrel and ethinylestradiol released from a combined contraceptive vaginal ring. Clin Pharmacokinet 2000;39:233–42.
31. Tuppurainen M, Klimscheffskij R, Venhola M, et al. The combined contraceptive vaginal ring (NuvaRing) and lipid metabolism: a comparative study. Contraception 2004;69:389–94.
32. van den Heuvel MW, van Bragt AJM, Alnabawy AKM, Kaptein MCJ. Comparison of ethylestradiol pharmacokinetics in three hormonal contraceptive formulations: the vaginal ring, the transdermal patch and an oral contraceptive. Contraception 2005;72:168–74.
33. Veres S, Miller L, Burington B. A comparison between the vaginal ring and oral contraceptives. Obstet Gynecol 2004;104:555–63.
34. Beksinska ME, Kleinschmidt I, Smit JA, Farley TM. Bone mineral density in adolescents using norethisterone enanthate, depot-medroxyprogesterone acetate or combined oral contraceptives for contraception. Contraception 2007;75:438–43.
35. Cromer BA, Blair JM, Mahan JD, Zibners L, Naumovski Z. A prospective comparison of bone density in adolescent girls receiving depot medroxyprogesterone acetate (Depo-Provera), levonorgestrel (Norplant), or oral contraceptives. J Pediatr 1996;129:671–6.
36. Cromer BA, Stager M, Bonny A, et al. Depot medroxyprogesterone acetate, oral contraceptives and bone mineral density in a cohort of adolescent girls. J Adolesc Health 2004;35:434–41.
37. Lara-Torre E, Edwards CP, Perlman S, Hertweck SP. Bone mineral density in adolescent females using depot medroxyprogesterone acetate. J Pediatr Adolesc Gynecol 2004;17:17–21.
38. Lloyd T, Taylor DS, Lin HM, et al. Oral contraceptive use by teenage women does not affect peak bone mass: a longitudinal study. Fertil Steril 2000;74:734–8.
39. Lloyd T, Petit MA, Lin HM, Beck TJ. Lifestyle factors and the development of bone mass and bone strength in young women. J Pediatr 2004;144:776–82.
40. Polatti F, Perotti F, Filippa N, Gallina D, Nappi RE. Bone mass and long-term monophasic oral contraceptive treatment in young women. Contraception 1995;51:221–4.
41. Wallace LS, Ballard JE. Lifetime physical activity and calcium intake related to bone density in young women. J Womens Health Gend Based Med 2002;11:389–98.
42. Afghani A, Abbott AV, Wiswell RA, et al. Bone mineral density in Hispanic women: role of aerobic capacity, fat-free mass, and adiposity. Int J Sports Med 2004;25:384–90.
43. Bahamondes L, Juliato CT, Villarreal M, et al. Bone mineral density in users of two kinds of once-a-month combined injectable contraceptives. Contraception 2006;74:259–63.
44. Berenson AB, Radecki CM, Grady JJ, Rickert VI, Thomas A. A prospective, controlled study of the effects of hormonal contraception on bone mineral density. Obstet Gynecol 2001;98:576–82.
45. Berenson AB, Breitkopf CR, Grady JJ, Rickert VI, Thomas A. Effects of hormonal contraception on bone mineral density after 24 months of use. Obstet Gynecol 2004;103:899–906.
46. Burr DB, Yoshikawa T, Teegarden D, et al. Exercise and oral contraceptive use suppress the normal age-related increase in bone mass and strength of the femoral neck in women 18–31 years of age. Bone 2000;27:855–63.
47. Castelo-Branco C, Martinez de Osaba MJ, Pons F, Vanrell JA. Effects on bone mass of two oral contraceptives containing ethinylestradiol and cyproterone acetate or desogestrel: results of a 2-year follow-up. Eur J Contracept Reprod Health Care 1998;3:79–84.
48. Cobb KL, Kelsey JL, Sidney S, Ettinger B, Lewis CE. Oral contraceptives and bone mineral density in white and black women in CARDIA. Coronary Risk Development in Young Adults. Osteoporos Int 2002;13:893–900.
49. Collins C, Thomas K, Harding A, et al. The effect of oral contraceptives on lumbar bone density in premenopausal women. J La State Med Soc 1988;140:35–9.

50. de Papp AE, Bone HG, Caulfield MP, et al. A cross-sectional study of bone turnover markers in healthy premenopausal women. Bone 2007;40:1222–30.
51. Elgan C, Samsioe G, Dykes AK. Influence of smoking and oral contraceptives on bone mineral density and bone remodeling in young women: a 2-year study. Contraception 2003;67:439–47.
52. Elgan C, Dykes AK, Samsioe G. Bone mineral density changes in young women: a two year study. Gynecol Endocrinol 2004;19:169–77.
53. Endrikat J, Mih E, Dusterberg B, et al. A 3-year double-blind, randomized, controlled study on the influence of two oral contraceptives containing either 20 microg or 30 microg ethinylestradiol in combination with levonorgestrel on bone mineral density. Contraception 2004;69:179–87.
54. Fortney JA, Feldblum PJ, Talmage RV, Zhang J, Godwin SE. Bone mineral density and history of oral contraceptive use. J Reprod Med 1994;39:105–9.
55. Garnero P, Sornay-Rendu E, Delmas PD. Decreased bone turnover in oral contraceptive users. Bone 1995;16:499–503.
56. Goldsmith N, Johnston J. Bone mineral: effects of oral contraceptives, pregnancy, and lactation. J Bone Joint Surg (American) 1975;57–A:657–68.
57. Hall ML, Heavens J, Cullum ID, Ell PJ. The range of bone density in normal British women. Br J Radiol 1990;63:266–9.
58. Hansen M, Overgaard K, Riis B, Christiansen C. Potential risk factors for development of postmenopausal osteoporosis—examined over a 12-year period. Osteoporos Int 1991;1:95–102.
59. Hartard M, Bottermann P, Bartenstein P, Jeschke D, Schwaiger M. Effects on bone mineral density of low-dosed oral contraceptives compared to and combined with physical activity. Contraception 1997;55:87–90.
60. Hartard M, Kleinmond C, Wiseman M, Weissenbacher ER, Felsenberg D, Erben RG. Detrimental effect of oral contraceptives on parameters of bone mass and geometry in a cohort of 248 young women. Bone 2007;40:444–50.
61. Hawker GA, Forsmo S, Cadarette SM, et al. Correlates of forearm bone mineral density in young Norwegian women: The Nord-Trondelag health study. Am J Epidemiol 2002;156:01.
62. Hreshchyshyn MM, Hopkins A, Zylstra S, Anbar M. Associations of parity, breast-feeding, and birth control pills with lumbar spine and femoral neck bone densities. Am J Obstet Gynecol 1988;159:318–22.
63. Kanders B, Lindsay R, Dempster D, Markhard L, Valiquette G. Determinants of bone mass in young healthy women. In: Christiansen, C Arnaud CD, Nordin BEC, Parfitt AM, Peck WA, Riggs BL, eds. Osteoporosis: proceedings of the Copenhagen Symposium on Osteoporosis. Copenhagen: Department of Clinical Chemistry, Glostrup Hospital: 1984:337–40.
64. Kleerekoper M, Brienza RS, Schultz LR, Johnson CC. Oral contraceptive use may protect against low bone mass. Henry Ford Hospital Osteoporosis Cooperative Research Group. Arch Intern Med 1991;151:1971–6.
65. Kritz-Silverstein D, Barrett-Connor E. Bone mineral density in postmenopausal women as determined by prior oral contraceptive use. Am J Public Health 1993;83:100–2.
66. Laitinen K, Valimaki M, Keto P. Bone mineral density measured by dual-energy X-ray absorptiometry in healthy Finnish women. Calcif Tissue Int 1991;48:224–31.
67. Lau EMC, Lynn H, Woo J, Melton III LJ. Areal and volumetric bone density in Hong Kong Chinese: A comparison with Caucasians living in the United States. Osteoporos Int 2003;14:01.
68. Lindsay R, Tohme J, Kanders B. The effect of oral contraceptive use on vertebral bone mass in pre- and post-menopausal women. Contraception 1986;34:333–40.
69. Lloyd T, Buchanan JR, Ursino GR, et al. Long-term oral contraceptive use does not affect trabecular bone density. Am J Obstet Gynecol 1989;160:402–4.
70. MacDougall J, Davies MC, Overton CE, et al. Bone density in a population of long term oral contraceptive pill users does not differ from that in menstruating women. Br J Fam Plann 1999;25:96–100.
71. Mais V, Fruzzetti F, Ajossa S, et al. Bone metabolism in young women taking a monophasic pill containing 20 mcg ethinylestradiol: a prospective study. Contraception 1993;48:445–52.
72. Masaryk P, Lunt M, Benevolenskaya L, et al. Effects of menstrual history and use of medications on bone mineral density: the EVOS Study. Calcif Tissue Int 1998;63:271–6.
73. Mazess RB, Barden HS. Bone density in premenopausal women: effects of age, dietary intake, physical activity, smoking, and birth-control pills. Am J Clin Nutr 1991;53:132–42.
74. Melton III LJ, Bryant SC, Wahner HW, et al. Influence of breastfeeding and other reproductive factors on bone mass later in life. Osteoporos Int 1993;3:76–83.
75. Murphy S, Khaw KT, Compston JE. Lack of relationship between hip and spine bone mineral density and oral contraceptive use. Eur J Clin Invest 1993;23:108–11.
76. Nappi C, Di Spiezio SA, Acunzo G, et al. Effects of a low-dose and ultra–low-dose combined oral contraceptive use on bone turnover and bone mineral density in young fertile women: a prospective controlled randomized study. Contraception 2003;67:355–9.
77. Nappi C, Di Spiezio SA, Greco E, et al. Effects of an oral contraceptive containing drospirenone on bone turnover and bone mineral density. Obstet Gynecol 2005;105:53–60.
78. Nelson M, Mayer AB, Rutherford O, Jones D. Calcium intake, physical activity and bone mass in pre-menopausal women. J Hum Nutr Diet 1991;4:171–8.
79. Ott SM, Scholes D, LaCroix AZ, et al. Effects of contraceptive use on bone biochemical markers in young women. J Clin Endocrinol Metab 2001;86:179–85.
80. Paoletti AM, Orru M, Lello S, et al. Short-term variations in bone remodeling markers of an oral contraception formulation containing 3 mg of drospirenone plus 30 microg of ethinyl estradiol: observational study in young postadolescent women. Contraception 2004;70:293–8.
81. Pasco JA, Kotowicz MA, Henry MJ, et al. Oral contraceptives and bone mineral density: A population-based study. Am J Obstet Gynecol 2000;182:265–9.
82. Perrotti M, Bahamondes L, Petta C, Castro S. Forearm bone density in long-term users of oral combined contraceptives and depot medroxyprogesterone acetate. Fertil Steril 2001;76:469–73.
83. Petitti DB, Piaggio G, Mehta S, Cravioto MC, Meirik O. Steroid hormone contraception and bone mineral density: a cross-sectional study in an international population. The WHO Study of Hormonal Contraception and Bone Health. Obstet Gynecol 2000;95:736–44.
84. Picard D, Ste-Marie LG, Coutu D, et al. Premenopausal bone mineral content relates to height, weight and calcium intake during early adulthood. Bone Miner 1988;4:299–309.
85. Prior JC, Kirkland SA, Joseph L, et al. Oral contraceptive use and bone mineral density in premenopausal women: cross-sectional, population-based data from the Canadian Multicentre Osteoporosis Study. Can Med Assoc J 2001;165:1023–9.
86. Recker RR, Davies KM, Hinders SM, et al. Bone gain in young adult women. JAMA 1992;268:2403–8.
87. Reed SD, Scholes D, LaCroix AZ, et al. Longitudinal changes in bone density in relation to oral contraceptive use. Contraception 2003;68:177–82.
88. Rodin A, Chapman M, Fogelman I. Bone density in users of combined oral contraception. Preliminary reports of a pilot study. Br J Fam Plann 1991;16:125–9.
89. Shoepe HA, Snow CM. Oral contraceptive use in young women is associated with lower bone mineral density than that of controls. Osteoporos Int 2005;16:1538–44.
90. Stevenson JC, Lees B, Devenport M, Cust MP, Ganger KF. Determinants of bone density in normal women: risk factors for future osteoporosis? BMJ 1989;298;924–8.

91. Beksinska M, Smit J, Kleinschmidt I, Farley T, Mbatha F. Bone mineral density in women aged 40–49 years using depot-medroxyprogesterone acetate, norethisterone enanthate or combined oral contraceptives for contraception. Contraception 2005;71:170–5.
92. Berning B, van KC, Schutte HE, et al. Determinants of lumbar bone mineral density in normal weight, non-smoking women soon after menopause. A study using clinical data and quantitative computed tomography. Bone Miner 1993;21:129–39.
93. Forsmo S, Schei B, Langhammer A, Forsen L. How do reproductive and lifestyle factors influence bone density in distal and ultradistal radius of early postmenopausal women? The Nord-Trondelag Health Survey, Norway. Osteoporos Int 2001;12:222–9.
94. Gambacciani M, Spinetti A, Taponeco F, et al. Longitudinal evaluation of perimenopausal vertebral bone loss: effects of a low-dose oral contraceptive preparation on bone mineral density and metabolism. Obstet Gynecol 1994;83:392–6.
95. Gambacciani M, Spinetti A, Cappagli B, et al. Hormone replacement therapy in perimenopausal women with a low dose oral contraceptive preparation: effects on bone mineral density and metabolism. Maturitas 1994;19(2):25–31).
96. Gambacciani M, Cappagli B, Ciaponi M, Benussi C, Genazzani AR. Hormone replacement therapy in perimenopause: effect of a low dose oral contraceptive preparation on bone quantitative ultrasound characteristics. Menopause 1999;6:43–8.
97. Gambacciani M, Ciaponi M, Cappagli B, Benussi C, Genazzani AR. Longitudinal evaluation of perimenopausal femoral bone loss: effects of a low-dose oral contraceptive preparation on bone mineral density and metabolism. Osteoporos Int 2000;11:544–8.
98. Gambacciani M, Cappagli B, Lazzarini V, et al. Longitudinal evaluation of perimenopausal bone loss: effects of different low dose oral contraceptive preparations on bone mineral density. Maturitas 2006;54:176–80.
99. Grainge MJ, Coupland CAC, Cliffe SJ, Chilvers CED, Hosking DJ. Reproductive, menstrual and menopausal factors: which are associated with bone mineral density in early postmenopausal women? Osteoporos Int 2001;12:777–87.
100. Johnell O, Nilsson BE. Life-style and bone mineral mass in perimenopausal women. Calcif Tissue Int 1984;36:354–6.
101. Liu SL, Lebrun CM. Effect of oral contraceptives and hormone replacement therapy on bone mineral density in premenopausal and perimenopausal women: a systematic review. Br J Sports Med 2006;40:11–24.
102. Progetto Menopausa Italia Study Group. Risk of low bone density in women attending menopause clinics in Italy. Maturitas 2002;42:105–11.
103. Shargil AA. Hormone replacement therapy in perimenopausal women with a triphasic contraceptive compound: a three-year prospective study. Int J Fertil 1985;30.
104. Sowers MF, Wallace RB, Lemke JH. Correlates of forearm bone mass among women during maximal bone mineralization. Prev Med 1985;14:585–96..
105. Sultana S, Choudhury S, Choudhury SA. Effect of combined oral contraceptives on bone mineral density in pre and postmenopausal women. Mymensingh Med J 2002;11:12–4.
106. Taechakraichana N, Limpaphayom K, Ninlagarn T, et al. A randomized trial of oral contraceptive and hormone replacement therapy on bone mineral density and coronary heart disease risk factors in postmenopausal women. Obstet Gynecol 2000;95:87–94.
107. Taechakraichana N, Jaisamrarn U, Panyakhamlerd K, Chaikittisilpa S, Limpaphayom K. Difference in bone acquisition among hormonally treated postmenopausal women with normal and low bone mass. J Med Assoc Thai 2001;84 Suppl 2:S586–S592.
108. Tavani A, La Vecchia C, Franceschi S. Oral contraceptives and bone mineral density. Am J Obstet Gynecol 2001;184:249–50.
109. Tuppurainen M, Kroger H, Saarikoski S, Honkanen R, Alhava E. The effect of previous oral contraceptive use on bone mineral density in perimenopausal women. Osteoporos Int 1994;4:93-8.
110. Volpe A, Amram A, Cagnacci A, Battaglia C. Biochemical aspects of hormonal contraception: effects on bone metabolism. Eur J Contracept Reprod Health Care 1997;2:123-6.
111. Barad D, Kooperberg C, Wactawski-Wende J, et al. Prior oral contraception and postmenopausal fracture: a Women's Health Initiative observational cohort study. Fertil Steril 2005;84:374–83.
112. Cobb KL, Bachrach LK, Sowers M, et al. The effect of oral contraceptives on bone mass and stress fractures in female runners. Med Sci Sports Exerc 2007;39:1464–73.
113. Cooper C, Hannaford P, Croft P, Kay CR. Oral contraceptive pill use and fractures in women: a prospective study. Bone 1993;14:41–5.
114. Johansson C, Mellstrom D. An earlier fracture as a risk factor for new fracture and its association with smoking and menopausal age in women. Maturitas 1996;24:97–106.
115. La Vecchia C, Tavani A, Gallus S. Oral contraceptives and risk of hip fractures. Lancet 1999;354:335–6.
116. Mallmin H, Ljunghall S, Persson I, Bergstrom R. Risk factors for fractures of the distal forearm: a population-based case-control study. Osteoporos Int 1994;4:298–304.
117. Michaelsson K, Baron JA, Farahmand BY, Persson I, Ljunghall S. Oral-contraceptive use and risk of hip fracture: a case-control study. Lancet 1999;353:1481–4.
118. Michaelsson K, Baron JA, Farahmand BY, Ljunghall S. Influence of parity and lactation on hip fracture risk. Am J Epidemiol 2001;153:1166–72.
119. O'Neill TW, Marsden D, Adams JE, Silman AJ. Risk factors, falls, and fracture of the distal forearm in Manchester, UK. J Epidemiol Community Health 1996;50:288–92.
120. O'Neill TW, Silman AJ, Naves DM, et al. Influence of hormonal and reproductive factors on the risk of vertebral deformity in European women. European Vertebral Osteoporosis Study Group. Osteoporos Int 1997;7:72–8.
121. Vessey M, Mant J, Painter R. Oral contraception and other factors in relation to hospital referral for fracture. Findings in a large cohort study. Contraception 1998;57:231–5.
122. Vestergaard P, Rejnmark L, Mosekilde L. Oral contraceptive use and risk of fractures. Contraception 2006;73:571–6.
123. Office on Women's Health, US Department of Health and Human Services. HHS blueprint for action on breastfeeding. Washington, DC: US Department of Health and Human Services, Office on Women's Health; 2000.
124. Kaern T. Effect of an oral contraceptive immediately post partum on initiation of lactation. Br Med J 1967;3:644–5.
125. Miller GH, Hughes LR. Lactation and genital involution effects of a new low-dose oral contraceptive on breast-feeding mothers and their infants. Obstet Gynecol 1970;35:44–50.
126. Gambrell RD. Immediate postpartum oral contraception. Obstet Gynecol 1970;36:101–6.
127. Guiloff E, Ibarrapo A, Zanartu J, et al. Effect of contraception on lactation. Am J Obstet Gynecol 1974;118:42–5.

128. Diaz S, Peralta O, Juez G, et al. Fertility regulation in nursing women. 3. Short-term influence of a low-dose combined oral-contraceptive upon lactation and infant growth. Contraception 1983;27:1–11.
129. Croxatto HB, Diaz S, Peralta O, et al. Fertility regulation in nursing women. 4. Long-term influence of a low-dose combined oral-contraceptive initiated at day 30 postpartum upon lactation and infant growth. Contraception 1983;27:13–25.
130. Peralta O, Diaz S, Juez G, et al. Fertility regulation in nursing women. 5. Long-term nfluence of a low-dose combined oral-contraceptive initiated at day 90 postpartum upon lactation and infant growth. Contraception 1983;27:27–38.
131. World Health Organization Special Programme of Research Development and Research Training in Human Reproduction. Effects of hormonal contraceptives on milk volume and infant growth. Contraception 1984;30:505–22.
132. Nilsson S, Melbin T, Hofvander Y, et al. Long-term follow-up of children breast-fed by women using oral contraceptives. Contraception 1986;34:443–53.
133. World Health Organization Task Force on Oral Contraceptives Special Programme of Research Development and Research Training in Human Reproduction. Effects of hormonal contraceptives on breast milk composition and infant growth. Stud Fam Plann 1988;19:361–9.
134. Lahteenmaki P. Influence of oral contraceptives on immediate postabortal pituitary-ovarian function. Acta Obstet Gynecol Scand 1978;Suppl 76:1–43.
135. Lahteenmaki P, Rasi V, Luukkainen T, Myllyä G. Coagulation factors in women using oral contraceptives or intrauterine contraceptive devices immediately after abortion. Am J Obstet Gynecol 1981;141:175–9.
136. Martin CW, Brown AH, Baird DT. A pilot study of the effect of methotrexate or combined oral contraceptive on bleeding patterns after induction of abortion with mifepristone and a prostaglandin pessary. Contraception 1998;58:99–103.
137. Niswonger JWH, London GD, Anderson GV, Wolfe L. Oral contraceptives during immediate postabortal period. Obstet Gynecol 1968;32:325–7.
138. Peterson WF. Contraceptive therapy following therapeutic abortion. Obstet Gynecol 1974;44:853–7.
139. Tang OS, et al. A randomized double-blind placebo-controlled study to assess the effect of oral contraceptive pills on the outcome of medical abortion with mifepristone and misoprostol. Hum Reprod 1999;14:722–5.
140. Tang OS, Gao PP, Cheng L, Lee SW, Ho PC. The effect of contraceptive pills on the measured blood loss in medical termination of pregnancy by mifepristone and misoprostol: a randomized placebo controlled trial. Hum Reprod 2002;17:99–102.
141. Fine PM, Tryggestad J, Meyers NJ, Sangi-Haghpeykar H. Safety and acceptability with the use of a contraceptive vaginal ring after surgical or medical abortion. Contraception 2007;75:367–71.
142. Gillum LA, Mamidipudi SK, Johnston SC. Ischaemic stroke risk with oral contraceptives: a meta-analysis. JAMA 2000;284:72–8.
143. Jick SS, Walker AM, Stergachis A, Jick H. Oral contraceptives and breast cancer. Br J Cancer 1989;59:618–21.
144. Khader YS, Rice J, John L, Abueita O. Oral contraceptive use and the risk of myocardial infarction: a meta-analysis. Contraception 2003;68:11–7.
145. Lawson DH, Davidson JF, Jick H. Oral contraceptive use and venous thromboembolism: absence of an effect of smoking. BMJ 1977;2:729–30.
146. Lidegaard O, Edstrom B, Kreiner S. Oral contraceptives and venous thromboembolism. A case-control study. Contraception 1998;57:291–301.
147. Nightingale AL, Lawrenson RA, Simpson EL, Williams TJ, MacRae KD, Farmer RD. The effects of age, body mass index, smoking and general health on the risk of venous thromboembolism in users of combined oral contraceptives. Eur J Contracept Reprod Health Care 2000;5:265–74.
148. Petitti D, Wingerd J, Pellegrin F, Ramcharan S. Risk of vascular disease in women. Smoking, oral contraceptives, noncontraceptive estrogens, and other factors. JAMA 1979;242:1150–4.
149. Rosenberg L, Palmer JR, Rao RS, Shapiro S. Low-dose oral contraceptive use and the risk of myocardial infarction. Arch Intern Med 2001;161:1065–70.
150. Straneva P, Hinderliter A, Wells E, Lenahan H, Girdler S. Smoking, oral contraceptives, and cardiovascular reactivity to stress. Obstet Gynecol 2000;95:78–83.
151. Tanis BC, van den Bosch MA, Kemmeren JM, et al. Oral contraceptives and the risk of myocardial infarction. N Engl J Med 2001;345:1787–93.
152. van den Bosch MA, Kemmeren JM, Tanis BC, et al. The RATIO study: oral contraceptives and the risk of peripheral arterial disease in young women. J Thromb Haemost 2003;1:439–44.
153. World Health Organization. Venous thromboembolic disease and combined oral contraceptives: results of international multicentre case-control study. Lancet 1995;346:1575–82.
154. Abdollahi M, Cushman M, Rosendaal FR. Obesity: risk of venous thrombosis and the interaction with coagulation factor levels and oral contraceptive use. Thromb Haemost 2003;89:493–8.
155. Lidegaard O, Edstrom B, Kreiner S. Oral contraceptives and venous thromboembolism: a five-year national case-control study. Contraception 2002;65:187–96.
156. Pomp ER, le CS, Rosendaal FR, Doggen CJ. Risk of venous thrombosis: obesity and its joint effect with oral contraceptive use and prothrombotic mutations. Br J Haematol 2007;139:289–96.
157. Schwartz SM, Petitti DB, Siscovick DS, et al. Stroke and use of low-dose oral contraceptives in young women: a pooled analysis of two US studies. Stroke 1998;29:2277–84.
158. Sidney S, Siscovick DS, Petitti DB, et al. Myocardial infarction and use of low-dose oral contraceptives: a pooled analysis of 2 US studies. Circulation 1998;98:1058–63.
159. Sidney S, Petitti DB, Soff GA, et al. Venous thromboembolic disease in users of low-estrogen combined estrogen-progestin oral contraceptives. Contraception 2004;70:3–10.
160. Brunner Huber LR, Hogue CJ, Stein AD, Drews C, Zieman M. Body mass index and risk for oral contraceptive failure: a case-cohort study in South Carolina. Ann Epidemiol 2006;16:637–43.
161. Brunner Huber LR, Toth JL. Obesity and oral contraceptive failure: findings from the 2002 National Survey of Family Growth. Am J Epidemiol 2007;166:1306–11.
162. Brunner LR, Hogue CJ. The role of body weight in oral contraceptive failure: results from the 1995 National Survey of Family Growth. Ann Epidemiol 2005;15:492–9.
163. Holt VL, Cushing-Haugen KL, Daling JR. Body weight and risk of oral contraceptive failure. Obstet Gynecol 2002;99:820–7.
164. Holt VL, Scholes D, Wicklund KG, Cushing-Haugen KL, Daling JR. Body mass index, weight, and oral contraceptive failure risk. Obstet Gynecol 2005;105:46–52.

165. Vessey M. Oral contraceptive failures and body weight: findings in a large cohort study. J Fam Plann Reprod Health Care 2001;27:90–1.
166. O'Connell KJ, Osborne LM, Westoff C. Measured and reported weight change for women using a vaginal contraceptive ring vs. a low-dose oral contraceptive. Contraception 2005;72:323–7.
167. Weiss HG, Nehoda H, Labeck B, et al. Pregnancies after adjustable gastric banding. Obes Surg 2001;11:303–6.
168. Gerrits EG, Ceulemans R, van HR, Hendrickx L, Totte E. Contraceptive treatment after biliopancreatic diversion needs consensus. Obes Surg 2003;13:378–82.
169. Victor A, Odlind V, Kral JG. Oral contraceptive absorption and sex hormone binding globulins in obese women: effects of jejunoileal bypass. Gastroenterol Clin North Am 1987;16:483–91.
170. Andersen AN, Lebech PE, Sorensen TI, Borggaard B. Sex hormone levels and intestinal absorption of estradiol and D-norgestrel in women following bypass surgery for morbid obesity. Int J Obes 1982;6:91–6.
171. Collaborative Group for the Study of Stroke in Young Women. Oral contraceptives and stroke in young women: associated risk factors. JAMA 1975;231:718–22.
172. Croft P, Hannaford P. Risk factors for acute myocardial infarction in women: evidence from the Royal College of General Practitioners' Oral Contraception Study. BMJ 1989;298:165–8.
173. D'Avanzo B, La Vecchia C, Negri E, Parazzini F, Franceschi S. Oral contraceptive use and risk of myocardial infarction: an Italian case-control study. J Epidemiol Community Health 1994;48:324–8.
174. Dunn NR, Faragher B, Thorogood M, et al. Risk of myocardial infarction in young female smokers. Heart 1999;82:581–3.
175. Hannaford P, Croft P, Kay CR. Oral contraception and stroke: evidence from the Royal College of General Practitioners' Oral Contraception Study. Stroke 1994;25:935–42.
176. Heinemann LA, Lewis MA, Spitzer WO, Thorogood M, Guggenmoos-Holzmann I, Bruppacher R. Thromboembolic stroke in young women. A European case-control study on oral contraceptives. Contraception 1998;57:29–37.
177. Kemmeren JM, Tanis BC, van den Bosch MA, et al. Risk of Arterial Thrombosis in Relation to Oral Contraceptives (RATIO) study: oral contraceptives and the risk of ischemic stroke. Stroke 2002;33:1202–8.
178. Lewis MA, Heinemann LA, Spitzer WO, MacRae KD, Bruppacher R. The use of oral contraceptives and the occurrence of acute myocardial infarction in young women. Results from the Transnational Study on Oral Contraceptives and the Health of Young Women. Contraception 1997;56:129–40.
179. Lidegaard O. Oral contraception and risk of a cerebral thromboembolic attack: results of a case-control study. BMJ 1993;306:956–63.
180. Lidegaard O. Oral contraceptives, pregnancy and the risk of cerebral thromboembolism: the influence of diabetes, hypertension, migraine and previous thrombotic disease. Br J Obstet Gynaecol 1995;102:153–9.
181. Lubianca JN, Faccin CS, Fuchs FD. Oral contraceptives: a risk factor for uncontrolled blood pressure among hypertensive women. Contraception 2003;67:19–24.
182. Narkiewicz K, Graniero GR, D'Este D, Mattarei M, Zonzin P, Palatini P. Ambulatory blood pressure in mild hypertensive women taking oral contraceptives. A case-control study. Am J Hypertens 1995;8:249–53.
183. Siritho S, Thrift AG, McNeil JJ, et al. Risk of ischemic stroke among users of the oral contraceptive pill: The Melbourne Risk Factor Study (MERFS) Group. Stroke 2003;34:1575–80.
184. World Health Organization. Haemorrhagic stroke, overall stroke risk, and combined oral contraceptives: results of an international, multicentre, case-control study. WHO Collaborative Study of Cardiovascular Disease and Steroid Hormone Contraception. Lancet 1996;348:505–10.
185. World Health Organization. Ischaemic stroke and combined oral contraceptives: results of an international, multicentre, case-control study. WHO Collaborative Study on Cardiovascular Disease and Steroid Hromone Contraception. Lancet 1996;348:498–505.
186. World Health Organization. Acute myocardial infarction and combined oral contraceptives: results of an international multicentre case-control study. WHO Collaborative Study on Cardiovascular Disease and Steroid Hormone Contraception. Lancet 1997;349:1202–9.
187. Lubianca JN, Moreira LB, Gus M, Fuchs FD. Stopping oral contraceptives: an effective blood pressure-lowering intervention in women with hypertension. J Hum Hypertens 2005;19:451–5.
188. Aberg H, Karlsson L, Melander S. Studies on toxaemia of pregnancy with special reference to blood pressure. ll. Results after 6–11 years' follow-up. Ups J Me Sci 1978;83:97–102.
189. Carmichael SM, Taylor MM, Ayers CR. Oral contraceptives, hypertension, and toxemia. Obstet Gynecol 1970;35:371–6.
190. Meinel H, Ihle R, Laschinski M. Effect of hormonal contraceptives on blood pressure following pregnancy-induced hypertension [in German]. Zentralbl Gynäkol 1987;109:527–31.
191. Pritchard JA, Pritchard SA. Blood pressure response to estrogen-porgestin oral contraceptive after pregnancy-induced hypertension. Am J Obstet Gynecol 1977;129:733–9.
192. Sibai BM, Taslimi MM, el-Nazer A, Amon E, Mabie BC, Ryan GM. Maternal-perinatal outcome associated with the syndrome of hemolysis, elevated liver enzymes, and low platelets in severe preeclampsia-eclampsia. Am J Obstet Gynecol 1986;155:501–9.
193. Sibai BM, Ramadan MK, Chari RS, Friedman SA. Pregnancies complicated by HELLP syndrome (hemolysis, elevated liver enzymes, and low platelets): subsequent pregnancy outcome and long-term prognosis. Am J Obstet Gynecol 1995;172:125–9.
194. Anderson BS, Olsen J, Nielsen GL, et al. Third generation oral contraceptives and heritable thrombophilia as risk factors of non-fatal venous thromboembolism. Thromb Haemost 1998;79:28–31.
195. Aznar J, Mira Y, Vaya A, et al. Factor V Leiden and prothrombin G20210A mutations in young adults with cryptogenic ischemic stroke. Thromb Haemost 2004;91:1031–4.
196. Bennet L, Odeberg H. Resistance to activated protein C, highly prevalent amongst users of oral contraceptives with venous thromboembolism. J Intern Med 1998;244:27–32.
197. Bloemenkamp KW, Rosendaal FR, Helmerhorst FM, et al. Enhancement by factor V Leiden mutation of risk of deep-vein thrombosis associated with oral contraceptives containing a third-generation progestagen [comment]. Lancet 1995;346:1593–6.
198. Bloemenkamp KW, Rosendaal FR, Helmerhorst FM, et al. Higher risk of venous thrombosis during early use of oral contraceptives in women with inherited clotting defects [comment]. Arch Intern Med 2000;160:49–52.
199. de Bruijn SF, Stam J, Koopman MM, et al. Case-control study of risk of cerebral sinu thrombosis in oral contraceptive users and in [correction of who are] carriers of heriditary prothrombotic conditions. The Cerebral Venous Sinus Thrombosis Study Group. BMJ 1998;316:589–92.

200. Emmerich J, Rosendaal FR, Cattaneo M, et al. Combined effect of factor V Leiden and prothrombin 20210A on the risk of venous thromboembolism—pooled analysis of 8 case-control studies including 2310 cases and 3204 controls. Study Group for Pooled-Analysis in Venous Thromboembolism. Thromb Haemost 2001;86:809–16.
201. Gadelha T, Andre C, Juca AA, et al. Prothrombin 20210A and oral contraceptive use as risk factors for cerebral venous thrombosis. Cerebrovasc Dis 2005;19:49–52.
202. Legnani C, Palareti G, Guazzaloca G, et al. Venous thromboembolism in young women: role of thrombophilic mutations and oral contraceptive use. Eur Heart J 2002;23:984–90.
203. Martinelli I, Sacchi E, Landi G, et al. High risk of cerebral-vein thrombosis in carriers of a prothrombin-gene mutation and in users of oral contraceptives [comment]. N Eng J Med 1998;338:1793–7.
204. Martinelli I, Taioli E, Bucciarelli P, et al. Interaction between the G20210A mutation of the prothrombin gene and oral contraceptive use in deep vein thrombosis. Arterioscler Thromb Vasc Biol 1999;19:700–3.
205. Martinelli I, Battaglioli T, Bucciarelli P, et al. Risk factors and recurrence rate of primary deep vein thrombosis of the upper extremities. Circulation 2004;110:566–70.
206. Martinelli I, Battaglia C, Burgo I, et al. Oral contraceptive use, thrombophilia and their interaction in young women with ischemic stroke. Haematologica 2006;91:844–7.
207. Middeldorp S, Meinardi JR, Koopman MM, et al. A prospective study of asymptomatic carriers of the factor V Leiden mutation to determine the incidence of venous thromboembolism [comment]. Ann Intern Med 2001;135:322–7.
208. Pabinger I, Schneider B. Thrombotic risk of women with hereditary antithrombin lll-, protein C- and protein S-deficiency taking oral contraceptive medication. The GTH Study Group on Natural Inhibitors. Thromb Haemost 1994;71:548–52.
209. Pezzini A, Grassi M, Iacoviello L, et al. Inherited thrombophilia and stratification of ischaemic stroke risk among users of oral contraceptives. J Neurol Neurosurg Psychiatry 2007;78:271–6.
210. Santamaria A, Mateo J, Oliver A, et al. Risk of thrombosis associated with oral contraceptives of women from 97 families with inherited thrombophilia: high risk of thrombosis in carriers of the G20210A mutation of the prothrombin gene. Haematologica 2001;86:965–71.
211. Slooter AJ, Rosendaal FR, Tanis BC, et al. Prothrombotic conditions, oral contraceptives, and the risk of ischemic stroke. J Thromb Haemost 2005;3:1213–7.
212. Spannagl M, Heinemann LA, Schramm W. Are factor V Leiden carriers who use oral contraceptives at extreme risk of venous thromboembolism? Eur J Contracept Reprod Health Care 2000;5:105–12.
213. van Boven HH, Vandenbroucke JP, Briet E, et al. Gene-gene and gene-environment interactions determine risk of thrombosis in families with inherited antithrombin deficiency. Blood 1999;94:2590–4.
214. van Vlijmen EF, Brouwer JL, Veeger NJ, et al. Oral contraceptives and the absolute risk of venous thromboembolism in women with single or multiple thrombophilic defects: results from a retrospective family cohort study. Arch Intern Med 2007;167:282–9.
215. Vandenbroucke JP, Koster T, Briet E, et al. Increased risk of venous thrombosis in oral-contraceptive users who are carriers of factor V Leiden mutation [comment]. Lancet 1994;344:1453–7.
216. Vaya AM. Prothrombin G20210A mutation and oral contraceptive use increase upper-extremity deep vein thrombotic risk. Thromb Haemost 2003;89:452–7.
217. The Criteria Committee of the New York Heart Association. Nomenclature and criteria for diagnosis of diseases of the heart and great vessels. 9th ed. Boston, MA: Little, Brown & Co; 1994.
218. Avila WS, Grinberg M, Melo NR, Aristodemo PJ, Pileggi F. Contraceptive use in women with heart disease [in Portuguese]. Arq Bras Cardiol 1996;66:205–11.
219. Bernatsky S, Ramsey-Goldman R, Gordon C, et al. Factors associated with abnormal Pap results in systemic lupus erythematosus. Rheumatology (Oxford) 2004;43:1386–9.
220. Bernatsky S, Clarke A, Ramsey-Goldman R, et al. Hormonal exposures and breast cancer in a sample of women with systemic lupus erythematosus. Rheumatology (Oxford) 2004;43:1178–81.
221. Chopra N, Koren S, Greer WL, et al. Factor V Leiden, prothrombin gene mutation, and thrombosis risk in patients with antiphospholipid antibodies. J Rheumatol 2002;29:1683–8.
222. Esdaile JM, Abrahamowicz M, Grodzicky T, et al. Traditional Framingham risk factors fail to fully account for accelerated atherosclerosis in systemic lupus erythematosus. Arthritis Rheum 2001;44:2331–7.
223. Julkunen HA. Oral contraceptives in systemic lupus erythematosus: side-effects and influence on the activity of SLE. Scand J Rheumatol 1991;20:427–33.
224. Julkunen HA, Kaaja R, Friman C. Contraceptive practice in women with systemic lupus erythematosus. Br J Rheumatol 1993;32:227–30.
225. Jungers P, Dougados M, Pelissier C, et al. Influence of oral contraceptive therapy on the activity of systemic lupus erythematosus. Arthritis Rheum 1982;25:618–23.
226. Manzi S, Meilahn EN, Rairie JE, et al. Age-specific incidence rates of myocardial infarction and angina in women with systemic lupus erythematosus: comparison with the Framingham Study. Am J Epidemiol 1997;145:408–15.
227. McAlindon T, Giannotta L, Taub N, et al. Environmental factors predicting nephristis in systemic lupus erythematosus. Ann Rheum Dis 1993;52:720–4.
228. McDonald J, Stewart J, Urowitz MB, et al. Peripheral vascular disease in patients with systemic lupus erythematosus. Ann Rheum Dis 1992;51:56–60.
229. Mintz G, Gutierrez G, Deleze M, et al. Contraception with progestogens in systemic lupus erythematosus. Contraception 1984;30:29–38.
230. Petri M. Musculoskeletal complications of systemic lupus erythematosus in the Hopkins Lupus Cohort: an update. Arthritis Care Res 1995;8:137–45.
231. Petri M, Kim MY, Kalunian KC, et al. Combined oral contraceptives in women with systemic lupus erythematosus. N Engl J Med 2005;353:2550–8.
232. Petri M. Lupus in Baltimore: evidence-based 'clinical perarls' from the Hopkins Lupus Cohort. Lupus 2005;14:970–3.
233. Sanchez-Guerrero J, Uribe AG, Jimenez-Santana L, et al. A trial of contraceptive methods in women with systemic lupus erythematosus. N Engl J Med 2005;353:2539–49.
234. Sarabi ZS, Chang E, Bobba R, et al. Incidence rates of arterial and venous thrombosis after diagnosis of systemic lupus erythematosus. Arthritis Rheum 2005;53:609–12.
235. Schaedel ZE, Dolan G, Powell MC. The use of the levonorgestrel-releasing intrauterine system in the management of menorrhagia in women with hemostatic disorders. Am J Obstet Gynecol 2005;193:1361–3.
236. Somers E, Magder LS, Petri M. Antiphospholipid antibodies and incidence of venous thrombosis in a cohort of patients with systemic lupus erythematosus. J Rheumatol 2002;29:2531–6.

237. Urowitz MB, Bookman AA, Koehler BE, et al. The bimodal mortality pattern of systemic lupus erythematosus. Am J Med 1976;60:221–5.
238. Choojitarom K, Verasertniyom O, Totemchokchyakarn K, et al. Lupus nephritis and Raynaud's phenomenon are significant risk factors for vascular thrombosis in SLE patients with positive antiphospholipid antibodies. Clin Rheumatol 2008;27:345–51.
239. Wahl DG, Guillemin F, de Maistre E, et al. Risk for venous thrombosis related to antiphospholipid antibodies in systemic lupus erythematosus—a meta-analysis. Lupus 1997;6:467–73.
240. Demers R, Blais JA, Pretty H. Rheumatoid arthritis treated by norethynodrel associated with mestranol: clinical aspects and laboratory tests [in French]. Can Med Assoc J 1966;95:350–4.
241. Drossaers-Bakker KW, Zwinderman AH, Van ZD, Breedveld FC, Hazes JM. Pregnancy and oral contraceptive use do not significantly influence outcome in long term rheumatoid arthritis. Ann Rheum Dis 2002;61:405-8.
242. Gilbert M, Rotstein J, Cunningham C, et al. Norethynodrel with mestranol in treatment of rheumatoid arthritis. JAMA 1964;190:235.
243. Gill D. Rheumatic complaints of women using anti-ovulatory drugs. An evaluation. J Chronic Dis 1968;21:435–44.
244. Hazes JM, Dijkmans BA, Vandenbroucke JP, Cats A. Oral contraceptive treatment for rheumatoid arthritis: an open study in 10 female patients. Br J Rheumatol 1989;28 Suppl 1:28–30.
245. Ostensen M, Aune B, Husby G. Effect of pregnancy and hormonal changes on the activity of rheumatoid arthritis. Scand J Rheumatol 1983;12:69–72.
246. Vignos PJ, Dorfman RI. Effect of large doses of progesterone in rheumatoid arthritis. Am J Med Sci 1951;222:29–34.
247. Bijlsma JW, Huber-Bruning O, Thijssen JH. Effect of oestrogen treatment on clinical and laboratory manifestations of rheumatoid arthritis. Ann Rheum Dis 1987;46:777–9.
248. Carolei A, Marini C, De Matteis G. History of migraine and risk of cerebral ischaemia in young adults. The Italian National Research Council Study Group on Stroke in the Young. Lancet 1996;347:1503–6.
249. Chang CL, Donaghy M, Poulter N. Migraine and stroke in young women: case-control study. The World Health Organisation Collaborative Study of Cardiovascular Disease and Steroid Hormone Contraception. BMJ 1999;318:13–8.
250. Tzourio C, Tehindrazanarivelo A, Iglesias S, et al. Case-control study of migraine and risk of ischaemic stroke in young women. BMJ 1995;310:830–3.
251. Oral contraceptives and stroke in young women. Associated risk factors. JAMA 1975;231:718–22.
252. Etminan M, Takkouche B, Isorna FC, Samii A. Risk of ischaemic stroke in people with migraine: systematic review and meta-analysis of observational studies. BMJ 2005;330:63.
253. Lidegaard O. Oral contraceptives, pregnancy, and the risk of cerebral thromboembolism: the influence of diabetes, hypertension, migraine and previous thrombotic disease [letter]. Br J Obstet Gynaecol 1996;103:94.
254. Nightingale AL, Farmer RD. Ischemic stroke in young women: a nested case-control study using the UK General Practice Research Database. Stroke 2004;35:1574–8.
255. Cromer BA, Smith RD, Blair JM, Dwyer J, Brown RT. A prospective study of adolescents who choose among levonorgestrel implant (Norplant), medroxyprogesterone acetate (Depo-Provera), or the combined oral contraceptive pill as contraception. Pediatrics 1994;94:687–94.
256. Deijen JB, Duyn KJ, Jansen WA, Klitsie JW. Use of a monophasic, low-dose oral contraceptive in relation to mental functioning. Contraception 1992;46:359–67.
257. Duke JM, Sibbritt DW, Young AF. Is there an association between the use of oral contraception and depressive symptoms in young Australian women? Contraception 2007;75:27–31.
258. Gupta N, O'Brien R, Jacobsen LJ, et al. Mood changes in adolescents using depo-medroxyprogesterone acetate for contraception: a prospective study. Am J Obstet Gynecol 2001;14:71–6.
259. Herzberg BN, Draper KC, Johnson AL, Nicol GC. Oral contraceptives, depression, and libido. BMJ 1971;3:495–500.
260. Koke SC, Brown EB, Miner CM. Safety and efficacy of fluoxetine in patients who receive oral contraceptive therapy. Am J Obstet Gynecol 2002;187:551–5.
261. O'Connell K, Davis AR, Kerns J. Oral contraceptives: side effects and depression in adolescent girls. Contraception 2007;75:299–304.
262. Westoff C, Truman C. Depressive symptoms and Depo-Provera. Contraception 1998;57:237–40.
263. Westoff C, Truman C, Kalmuss D, et al. Depressive symptoms and Norplant contraceptive implants. Contraception 1998;57:241–5.
264. Young EA, Kornstein SG, Harvey AT, et al. Influences of hormone-based contraception on depressive symptoms in premenopausal women with major depression. Psychoneuroendocrinology 2007;32:843–53.
265. Iyer V, Farquhar C, Jepson R. Oral contraceptive pills for heavy menstrual bleeding [review]. Cochrane Database Syst Rev 2000;CD000154.
266. Davis L, Kennedy SS, Moore J, Prentice A. Modern combined oral contraceptives for pain associated with endometriosis. Cochrane Database Syst Rev 2007;CD001019.
267. Hendrix SL, Alexander NJ. Primary dysmenorrhea treatment with a desogetrel-containing low-dose oral contraceptive. Contraception 2002;66:393–9.
268. Proctor ML, Roberts H, Farquhar C. Combined oral contraceptive pill (OCP) as treatment for primary dysmenorrhoea. Cochrane Database Syste Rev 2001;CD002120.
269. Adewole IF, Oladokun A, Fawole AO, Olawuyi JF, Adeleye JA. Fertility regulatory methods and development of complications after evacuation of complete hydatidiform mole. J Obstet Gynecol 2000;20:68–9.
270. Berkowitz RS, Goldstein DP, Marean AR, Bernstein M. Oral contraceptives and post-molar trophoblastic disease. Obstet Gynecol 1981;58:474–7.
271. Curry SL, Schlaerth JB, Kohorn EI, et al. Hormonal contraception and trophoblastic sequelae after hydatidiform mole (a Gynecologic Oncology Group Study). Am J Obstet Gynecol 1989;160:805–9.
272. Deicas RE, Miller DS, Rademaker AW, Lurain JR. The role of contraception in the development of postmolar trophoblastic tumour. Obstet Gynecol 1991;78:221–6.
273. Goldberg GL, Cloete K, Bloch B, Wiswedel K, Altaras MM. Medroxyprogesterone acetate in non-metastatic gestational trophoblastic disease. Br J Obstet Gynaecol 1987;94:22–5.
274. Ho Yuen B, Burch P. Relationship of oral contraceptives and the intrauterine contraceptive devices to the regression of concentration of the beta subunit of human chorionic gonadotropin and invasive complications after molar pregnancy. Am J Obstet Gynecol 1983;145:214–7.
275. Morrow P, Nakamura R, Schlaerth J, Gaddis O, Eddy G. The influence of oral contraceptives on the postmolar human chorionic gonadotropin regression curve. Am J Obstet Gynecol 1985;151:906–14.
276. Eddy GL, Schlaerth JB, Natlick RH, et al. Postmolar trophoblastic disease in women using hormonal contraception with and without estrogen. Obstet Gynecol 1983;62:736–40.

277. Smith JS. Cervical cancer and use of hormonal conraceptives: a systematic review. Lancet 2003;361:1159–67.
278. Black MM, Barclay THC, Polednak A, et al. Family history, oral contraceptive useage, and breast cancer. Cancer 1983;51:2147–51.
279. Brinton LA, Hoover R, Szklo M, Fraumeni JF. Oral contraceptives and breast cancer. Int J Epidemiol 1982;11:316–22.
280. Brohet RM, Goldgar DE, Easton DF, et al. Oral contraceptives and breast cancer risk in the International BRCA1/2 Carrier Cohort Study: a report from EMBRACE, GENEPSO, GEO-HEBON, and the IBCCS Collaborating Group. J Clin Oncol 2007;25 :3831–6.
281. Claus EB, Stowe M, Carter D. Oral contraceptives and the risk of ductal breast carcinoma in situ. Breast Cancer Res Treat 2003;81:129–36.
282. Collaborative Group on Hormonal Factors in Breast Cancer. Familial breast cancer: collaborative reanalysis of individual data from 52 epidemiological studies including 58 209 women with breast cancer and 101 986 women without the disease. Lancet 2001;358:1389–99.
283. Grabrick DM, Hartmann LC, Cerhan JR, et al. Risk of breast cancer with oral contraceptive use in women with a family history of breast cancer [comment]. JAMA 2000;284:1791–8.
284. Gronwald J, Byrski T, Huzarski T, et al. Influence of selected lifestyle factors on breast and ovarian cancer risk in *BRCA1* mutation carriers from Poland. Breast Cancer Res Treat 2006;95:105–9.
285. Haile RW, Thomas DC, McGuire V, et al. *BRCA1* and *BRCA2* mutation carriers, oral contraceptive use, and breast cancer before age 50. Cancer Epidemiol Biomarkers Prev 2006;15:1863–70.
286. Harris NV, Weiss NS, Francis AM, Polissar L. Breast cancer in relation to patterns of oral contraceptive use. Am J Epidemiol 1982;116:643–51.
287. Hennekens CH, Speizer FE, Lipnick RJ, et al. A case-control study of oral contraceptive use and breast cancer. J Natl Cancer Inst 1984;72:39–42.
288. Jernstrom H, Loman N, Johannsson OT, Borg A, Olsson H. Impact of teenage oral contraceptive use in a population-based series of early-onset breast cancer cases who have undergone *BRCA* mutation testing. Eur J Cancer 2005;41:2312–20.
289. Marchbanks PA, McDonald JA, Wilson HG, et al. Oral contraceptives and the risk of breast cancer. N Engl J Med 2002;346:2025–32.
290. Milne RL, Knight JA, John EM, et al. Oral contraceptive use and risk of early-onset breast cancer in carriers and noncarriers of *BRCA1* and *BRCA2* mutations. Cancer Epidemiol Biomarkers Prev 2005;14 :350–6.
291. Narod S, Dube MP, Klijn J, et al. Oral contraceptives and the risk of breast cancer in *BRCA1* and *BRCA2* mutation carriers. J Natl Cancer Inst 2002;94:1773–9.
292. Rosenberg L, Palmer JR, Rao RS, et al. Case-control study of oral contraceptive use and risk of breast cancer. Am J Epidemiol 1996;143:25–37.
293. Silvera SAN, Miller AB, Rohan TE. Oral contraceptive use and risk of breast cancer among women with a family history of breast cancer: a prospective cohort study. Cancer Causes Control 2005;16:1059–63.
294. Ursin G, Henderson BE, Haile RW, et al. Does oral contraceptive use increase the risk of breast cancer in women with *BRCA1/BRCA2* mutations more than in other women? Cancer Res 1997;57:3678–81.
295. Ursin G, Ross RK, Sullivan-Halley J, et al. Use of oral contraceptives and risk of breast cancer in young women. Breast Cancer Res Treat 1998;50:175–84.
296. Determinants of cervical *Chlamydia trachomatis* infection in Italy. The Italian MEGIC Group. Genitourin Med 1993;69:123–5.
297. Ackers JP, Lumsden WH, Catterall RD, Coyle R. Antitrichomonal antibody in the vaginal secretions of women infected with *T. vaginalis*. Br J Vener Dis 1975;51:319–23.
298. Acosta-Cazares B, Ruiz-Maya L, Escobedo dlP. Prevalence and risk factors for *Chlamydia trachomatis* infection in low-income rural and suburban populations of Mexico. Sex Transm Dis 1996;23:283–8.
299. Addiss DG, Vaughn ML, Holzhueter MA, Bakken LL, Davis JP. Selective screening for *Chlamydia trachomatis* infection in non-urban family planning clinics in Wisconsin. Fam Plann Perspect 1987;19:252–6.
300. Arya OP, Mallinson H, Goddard AD. Epidemiological and clinical correlates of chlamydial infection of the cervix. Br J Vener Dis 1981;57:118–24.
301. Austin H, Louv WC, Alexander WJ. A case-control study of spermicides and gonorrhea. JAMA 1984;251:2822–4.
302. Avonts D, Sercu M, Heyerick P, et al. Incidence of uncomplicated genital infections in women using oral contraception or an intrauterine device: a prospective study. Sex Transm Dis 1990;17:23–9.
303. Baeten JM, Nyange PM, Richardson BA, et al. Hormonal contraception and risk of sexually transmitted disease acquisition: results from a prospective study. Am J Obstet Gynecol 2001;185:380–5.
304. Barbone F, Austin H, Louv WC, Alexander WJ. A follow-up study of methods of contraception, sexual activity, and rates of trichomoniasis, candidiasis, and bacterial vaginosis. Am J Obstet Gynecol 1990;163:510–4.
305. Barnes RC, Katz BP, Rolfs RT, et al. Quantitative culture of endocervical *Chlamydia trachomatis*. J Clin Microbiol 1990;28:774–80.
306. Berger GS, Keith L, Moss W. Prevalence of gonorrhoea among women using various methods of contraception. Br J Vener Dis 1975;51:307–9.
307. Bhattacharyya MN, Jephcott AE. Diagnosis of gonorrhea in women—influence of the contraceptive pill. J Am Vener Dis Assoc 1976;2:21–4.
308. Blum M, Pery J, Kitai E. The link between contraceptive methods and *Chlamydia trachomatis* infection. Adv Contracept 1988;4:233–9.
309. Bontis J, Vavilis D, Panidis D, et al. Detection of *Chlamydia trachomatis* in asymptomatic women: relationship to history, contraception, and cervicitis. Adv Contracept 1994;10:309–15.
310. Bramley M, Kinghorn G. Do oral contraceptives inhibit *Trichomonas vaginalis*? Sex Transm Dis 1979;6:261–3.
311. Bro F, Juul S. Predictors of *Chlamydia trachomatis* infection in women in general practice. Fam Pract 1990;7:138–43.
312. Burns DC, Darougar S, Thin RN, Lothian L, Nicol CS. Isolation of *Chlamydia* from women attending a clinic for sexually transmitted disease. Br J Vener Dis 1975;51:314–8.
313. Ceruti M, Canestrelli M, Condemi V, et al. Methods of contraception and rates of genital infections. Clin Exp Obstet Gynecol 1994;21:119–23.
314. Chacko M, Lovchik J. *Chlamydia trachomatis* infection in sexually active adolescents: prevalence and risk factors. Pediatrics 1984;73:836–40.
315. Cottingham J, Hunter D. *Chlamydia trachomatis* and oral contraceptive use: a quantitative review. Genitourin Med 1992;68:209–16.
316. Crowley T, Horner P, Hughes A, et al. Hormonal factors and the laboratory detection of *Chlamydia trachomatis* in women: implications for screening? Int J STD AIDS 1997;8:25–31.
317. Edwards D, Phillips D, Stancombe S. *Chlamydia trachomatis* infection at a family planning clinic. N Z Med J 1985;98:333–5.
318. Evans BA, Kell PD, Bond RA, et al. Predictors of seropositivity to herpes simplex virus type 2 in women. Int J STD AIDS 2003;14:30–6.

319. Evans DL, Demetriou E, Shalaby H, Waner JL. Detection of *Chlamydia trachomatis* in adolescent females using direct immunofluorescence. Clin Pediatr (Phila) 1988;27:223–8.
320. Fish AN, Fairweather DV, Oriel JD, Ridgway GL. *Chlamydia trachomatis* infection in a gynaecology clinic population: identification of high-risk groups and the value of contact tracing. Eur J Obstet Gynecol Reprod Biol 1989;31:67–74.
321. Fouts AC, Kraus SJ. *Trichomonas vaginalis*: reevaluation of its clinical presentation and laboratory diagnosis. J Infect Dis 1980;141:137–43.
322. Fraser JJ, Jr., Rettig PJ, Kaplan DW. Prevalence of cervical *Chlamydia trachomatis* and *Neisseria gonorrhoeae* in female adolescents. Pediatrics 1983;71:333–6.
323. Gertig DM, Kapiga SH, Shao JF, Hunter DJ. Risk factors for sexually transmitted diseases among women attending family planning clinics in Dar-es-Salaam, Tanzania. Genitourin Med 1997;73:39–43.
324. Green J, de Gonzalez AB, Smith JS, et al. Human papillomavirus infection and use of oral contraceptives. Br J Cancer 2003;88:1713–20.
325. Griffiths M, Hindley D. Gonococcal pelvic inflammatory disease, oral contraceptives, and cervical mucus. Genitourin Med 1985;61:67.
326. Han Y, Morse DL, Lawrence CE, Murphy D, Hipp S. Risk profile for *Chlamydia* infection in women from public health clinics in New York State. J Community Health 1993;18:1–9.
327. Handsfield HH, Jasman LL, Roberts PL, et al. Criteria for selective screening for *Chlamydia trachomatis* infection in women attending family planning clinics. JAMA 1986;255:1730–4.
328. Hanna NF, Taylor-Robinson D, Kalodiki-Karamanoli M, Harris JR, McFadyen IR. The relation between vaginal pH and the microbiological status in vaginitis. Br J Obstet Gynaecol 1985;92:1267–71.
329. Harrison HR, Costin M, Meder JB, et al. Cervical *Chlamydia trachomatis* infection in university women: relationship to history, contraception, ectopy, and cervicitis. Am J Obstet Gynecol 1985;153:244–51.
330. Hart G. Factors associated with genital chlamydial and gonococcal infection in females. Genitourin Med 1992;68:217–20.
331. Herrmann B, Espinoza F, Villegas RR, et al. Genital chlamydial infection among women in Nicaragua: validity of direct fluorescent antibody testing, prevalence, risk factors and clinical manifestations. Genitourin Med 1996;72:20–6.
332. Hewitt AB. Oral contraception among special clinic patients. With particular reference to the diagnosis of gonorrhoea. Br J Vener Dis 1970;46:106–7.
333. Hilton AL, Richmond SJ, Milne JD, Hindley F, Clarke SK. *Chlamydia* A in the female genital tract. Br J Vener Dis 1974;50:1–10.
334. Hiltunen-Back E, Haikala O, Kautiainen H, Paavonen J, Reunala T. A nationwide sentinel clinic survey of *Chlamydia trachomatis* infection in Finland. Sex Transm Dis 2001;28:252–8.
335. Jacobson DL, Peralta L, Farmer M, et al. Relationship of hormonal contraception and cervical ectopy as measured by computerized planimetry to chlamydial infection in adolescents. Sex Transm Dis 2000;27:313–9.
336. Jaffe LR, Siqueira LM, Diamond SB, Diaz A, Spielsinger NA. *Chlamydia trachomatis* detection in adolescents. A comparison of direct specimen and tissue culture methods. J Adolesc Health Care 1986;7:401–4.
337. Jick H, Hannan MT, Stergachis A, et al. Vaginal spermicides and gonorrhea. JAMA 1982;248:1619–21.
338. Johannisson G, Karamustafa A, Brorson J. Influence of copper salts on gonococci. Br J Vener Dis 1976;52:176–7.
339. Keith L, Berer GS, Moss W. Cervical gonorrhea in women using different methods of contraception. J Am Vener Dis Assoc 1976;3:17–9.
340. Kinghorn GR, Waugh MA. Oral contraceptive use and prevalence of infection with *Chlamydia trachomatis* in women. Br J Vener Dis 1981;57:187–90.
341. Lavreys L, Chohan B, Ashley R, et al. Human herpesvirus 8: seroprevalence and correlates in prostitutes in Mombasa, Kenya. J Infect Dis 2003;187:359–63.
342. Lefevre JC, Averous S, Bauriaud R, et al. Lower genital tract infections in women: comparison of clinical and epidemiologic findings with microbiology. Sex Transm Dis 1988;15:110–3.
343. Louv WC, Austin H, Perlman J, Alexander WJ. Oral contraceptive use and the risk of chlamydial and gonococcal infections. Am J Obstet Gynecol 1989;160:396–402.
344. Lowe TL, Kraus SJ. Quantitation of *Neisseria gonorrhoeae* from women with gonorrhea. J Infect Dis 1976;133:621–6.
345. Lycke E, Lowhagen GB, Hallhagen G, Johannisson G, Ramstedt K. The risk of transmission of genital *Chlamydia trachomatis* infection is less than that of genital *Neisseria gonorrhoeae* infection. Sex Transm Dis 1980;7:6–10.
346. Macaulay ME, Riordan T, James JM, et al. A prospective study of genital infections in a family-planning clinic. 2. *Chlamydia* infection—the identification of a high-risk group. Epidemiol Infect 1990;104:55–61.
347. Magder LS, Harrison HR, Ehret JM, Anderson TS, Judson FN. Factors related to genital *Chlamydia trachomatis* and its diagnosis by culture in a sexually transmitted disease clinic. Am J Epidemiol 1988;128:298–308.
348. Magder LS, Klontz KC, Bush LH, Barnes RC. Effect of patient characteristics on performance of an enzyme immunoassay for detecting cervical *Chlamydia trachomatis* infection. J Clin Microbiol 1990;28:781–4.
349. Masse R, Laperriere H, Rousseau H, Lefebvre J, Remis RS. *Chlamydia trachomatis* cervical infection: prevalence and determinants among women presenting for routine gynecologic examination. Can Med Assoc J 1991;145:953–61.
350. McCormack WM, Reynolds GH. Effect of menstrual cycle and method of contraception on recovery of *Neisseria gonorrhoeae*. JAMA 1982;247:1292–4.
351. Morrison CS, Bright P, Wong EL, et al. Hormonal contraceptive use, cervical ectopy, and the acquisition of cervical infections. Sex Transm Dis 2004;31:561–7.
352. Nayyar KC, O'Neill JJ, Hambling MH, Waugh MA. Isolation of *Chlamydia trachomatis* from women attending a clinic for sexually transmitted diseases. Br J Vener Dis 1976;52:396–8.
353. Oh MK, Feinstein RA, Soileau EJ, Cloud GA, Pass RF. *Chlamydia trachomatis* cervical infection and oral contraceptive use among adolescent girls. J Adolesc Health Care 1989;10:376–81.
354. Oriel JD, Powis PA, Reeve P, Miller A, Nicol CS. Chlamydial infections of the cervix. Br J Vener Dis 1974;50:11–6.
355. Oriel JD, Johnson AL, Barlow D, et al. Infection of the uterine cervix with *Chlamydia trachomatis*. J Infect Dis 1978;137:443–51.
356. Paavonen J, Vesterinen E. *Chlamydia trachomatis* in cervicitis and urethritis in women. Scand J Infect Dis Suppl 1982;32:45–54.
357. Park BJ, Stergachis A, Scholes D, et al. Contraceptive methods and the risk of *Chlamydia trachomatis* infection in young women. Am J Epidemiol 1995;142:771–8.
358. Pereira LH, Embil JA, Haase DA, Manley KM. Cytomegalovirus infection among women attending a sexually transmitted disease clinic: association with clinical symptoms and other sexually transmitted diseases. Am J Epidemiol 1990;131:683–92.

359. Rahm VA, Odlind V, Pettersson R. *Chlamydia trachomatis* in sexually active teenage girls. Factors related to genital chlamydial infection: a prospective study. Genitourin Med 1991;67:317–21.
360. Reed BD, Huck W, Zazove P. Differentiation of *Gardnerella vaginalis*, *Candida albicans*, and *Trichomonas vaginalis* infections of the vagina. J Fam Pract 1989;28:673–80.
361. Ripa KT, Svensson L, Mardh PA, Westrom L. *Chlamydia trachomatis* cervicitis in gynecologic outpatients. Obstet Gynecol 1978;52:698–702.
362. Ruijs GJ, Kauer FM, van Gijssel PM, Schirm J, Schroder FP. Direct immunofluorescence for *Chlamydia trachomatis* on urogenital smears for epidemiological purposes. Eur J Obstet Gynecol Reprod Biol 1988;27:289–97.
363. Schachter J, Stoner E, Moncada J. Screening for chlamydial infections in women attending family planning clinics. West J Med 1983;138:375–9.
364. Sellors JW, Karwalajtys TL, Kaczorowski J, et al. Incidence, clearance and predictors of human papillomavirus infection in women. Can Med Assoc J 2003;168:421–5.
365. Sessa R, Latino MA, Magliano EM, et al. Epidemiology of urogenital infections caused by *Chlamydia trachomatis* and outline of characteristic features of patients at risk. J Med Microbiol 1994;41:168–72.
366. Shafer MA, Beck A, Blain B, et al. *Chlamydia trachomatis*: important relationships to race, contraception, lower genital tract infection, and Papanicolaou smear. J Pediatr 1984;104:141–6.
367. Smith JS, Herrero R, Munoz N, et al. Prevalence and risk factors for herpes simplex virus type 2 infection among middle-age women in Brazil and the Philippines. Sex Transm Dis 2001;28:187–94.
368. Staerfelt F, Gundersen TJ, Halsos AM, et al. A survey of genital infections in patients attending a clinic for sexually transmitted diseases. Scand J Infect Dis Suppl 1983;40:53–7.
369. Svensson L, Westrom L, Mardh PA. *Chlamydia trachomatis* in women attending a gynaecological outpatient clinic with lower genital tract infection. Br J Vener Dis 1981;57:259–62.
370. Tait IA, Rees E, Hobson D, Byng RE, Tweedie MC. Chlamydial infection of the cervix in contacts of men with nongonococcal urethritis. Br J Vener Dis 1980;56:37–45.
371. Vaccarella S, Herrero R, Dai M, et al. Reproductive factors, oral contraceptive use, and human papillomavirus infection: pooled analysis of the IARC HPV prevalence surveys. Cancer Epidemiol Biomarkers Prev 2006;15:2148–53.
372. Willmott FE, Mair HJ. Genital herpesvirus infection in women attending a venereal diseases clinic. Br J Vener Dis 1978;54:341–3.
373. Winer RL, Lee SK, Hughes JP, et al. Genital human papillomavirus infection: incidence and risk factors in a cohort of female university students. Am J Epidemiol 2003;157:218–26. Erratum in Am J Epidemiol. 2003;157:858.
374. Winter L, Goldy AS, Baer C. Prevalence and epidemiologic correlates of *Chlamydia trachomatis* in rural and urban populations. Sex Transm Dis 1990;17:30–6.
375. Wolinska WH, Melamed MR. Herpes genitalis in women attending Planned Parenthood of New York City. Acta Cytol 1970;14:239–42.
376. Woolfitt JM, Watt L. Chlamydial infection of the urogenital tract in promiscuous and non-promiscuous women. Br J Vener Dis 1977;53:93–5.
377. European Study Group on Heterosexual Transmission of HIV. Comparison of female to male and male to female transmission of HIV in 563 stable couples. BMJ 1992;304:809–13.
378. Aklilu M, Messele T, Tsegaye A, et al. Factors associated with HIV-1 infection among sex workers of Addis Ababa, Ethiopia. AIDS 2001;15:87–96.
379. Allen S, Serufilira A, Gruber V, et al. Pregnancy and contraception use among urban Rwandan women after HIV testing and counseling. Am J Public Health 1993;83:705–10.
380. Baeten JM, Benki S, Chohan V, et al. Hormonal contraceptive use, herpes simplex virus infection, and risk of HIV-1 acquisition among Kenyan women. AIDS 2007;21:1771–7.
381. Chao A, Bulterys M, Musanganire F, et al. Risk factors associated with prevalent HIV-1 infection among pregnant women in Rwanda. National University of Rwanda–Johns Hopkins University AIDS Research Team. Int J Epidemiol 1994;23:371–80.
382. Cohen CR, Duerr A, Pruithithada N, et al. Bacterial vaginosis and HIV seroprevalence among female commercial sex workers in Chiang Mai, Thailand. AIDS 1995;9:1093–7.
383. Criniti A, Mwachari CW, Meier AS, et al. Association of hormonal contraception and HIV-seroprevalence in Nairobi, Kenya. AIDS 2003;17:2667–9.
384. de Vincenzi I. A longitudinal study of human immunodeficiency virus transmission by heterosexual partners. European Study Group on Heterosexual Transmission of HIV [comment]. N Engl J Med 1994;331:341–6.
385. Ellerbrock TV, Lieb S, Harrington PE, et al. Heterosexually transmitted human immunodeficiency virus infection among pregnant women in a rural Florida community [comment]. N Engl J Med 1992;327:1704–9.
386. Gray JA, Dore GJ, Li Y, et al. HIV-1 infection among female commercial sex workers in rural Thailand. AIDS 1997;11:89–94.
387. Guimaraes MD, Munoz A, Boschi-Pinto C, Castilho EA. HIV infection among female partners of seropositive men in Brazil. Rio de Janeiro Heterosexual Study Group. Am J Epidemiol 1995;142:538–47.
388. Hira SK, Kamanga J, Macuacua R, Feldblum PJ. Oral contraceptive use and HIV infection. Int J STD AIDS 1990;1:447–8.
389. Kapiga SH, Shao JF, Lwihula GK, Hunter DJ. Risk factors for HIV infection among women in Dar-es-Salaam, Tanzania. J Acquir Immune Defic Syndr 1994;7:301–9.
390. Kapiga SH, Lyamuya EF, Lwihula GK, Hunter DJ. The incidence of HIV infection among women using family planning methods in Dar es Salaam, Tanzania. AIDS 1998;12:75–84.
391. Kilmarx PH, Limpakarnjanarat K, Mastro TD, et al. HIV-1 seroconversion in a prospective study of female sex workers in northern Thailand: continued high incidence among brothel-based women. AIDS 1998;12:1889–98.
392. Kunanusont C, Foy HM, Kreiss JK, et al. HIV-1 subtypes and male-to-female transmission in Thailand. Lancet 1995;345:1078–83.
393. Laga M, Manoka A, Kivuvu M, et al. Non-ulcerative sexually transmitted diseases as risk factors for HIV-1 transmission in women: results from a cohort study [comment]. AIDS 1993;7:95–102.
394. Lavreys L, Baeten JM, Martin HL, Jr., et al. Hormonal contraception and risk of HIV-1 acquisition: results of a 10-year prospective study. AIDS 2004;18:695–7.
395. Limpakarnjanarat K, Mastro TD, Saisorn S, et al. HIV-1 and other sexually transmitted infections in a cohort of female sex workers in Chiang Rai, Thailand. Sex Transm Infect 1999;75:30–5.
396. Martin HL, Jr, Nyange PM, Richardson BA, et al. Hormonal contraception, sexually transmitted diseases, and risk of heterosexual transmission of human immunodeficiency virus type 1. J Infect Dis 1998;178:1053–9.

397. Mati JK, Hunter DJ, Maggwa BN, Tukei PM. Contraceptive use and the risk of HIV infection in Nairobi, Kenya. Int J Gynaecol Obstet 1995;48:61–7.
398. Morrison CS, Richardson BA, Mmiro F, et al. Hormonal contraception and the risk of HIV acquisition. AIDS 2007;21:85–95.
399. Moss GB, Clemetson D, D'Costa L, et al. Association of cervical ectopy with heterosexual transmission of human immunodeficiency virus: results of a study of couples in Nairobi, Kenya. J Infect Dis 1991;164:588–91.
400. Myer L, Denny L, Wright TC, Kuhn L. Prospective study of hormonal contraception and women's risk of HIV infection in South Africa. Int J Epidemiol 2007;36:166–74.
401. Nagachinta T, Duerr A, Suriyanon V, et al. Risk factors for HIV-1 transmission from HIV-seropositive male blood donors to their regular female partners in northern Thailand. AIDS 1997;11:1765–72.
402. Nicolosi A, Correa Leite ML, Musicco M, et al. The efficiency of male-to-female and female-to-male sexual transmission of the human immunodeficiency virus: a study of 730 stable couples. Italian Study Group on HIV Heterosexual Transmission [comment]. Epidemiology 1994;5:570–5.
403. Nzila N, Laga M, Thiam MA, et al. HIV and other sexually transmitted diseases among female prostitutes in Kinshasa. AIDS 1991;5:715–21.
404. Pineda JA, Aguado I, Rivero A, et al. HIV-1 infection among non-intravenous drug user female prostitutes in Spain. No evidence of evolution to pattern II. AIDS 1992;6:1365–9.
405. Plourde PJ, Plummer FA, Pepin J, et al. Human immunodeficiency virus type 1 infection in women attending a sexually transmitted diseases clinic in Kenya [comment]. J Infect Dis 1992;166:86–92.
406. Plummer FA, Simonsen JN, Cameron DW, et al. Cofactors in male-female sexual transmission of human immunodeficiency virus type 1 [comment]. J Infect Dis 1991;163:233–9.
407. Rehle T, Brinkmann UK, Siraprapasiri T, et al. Risk factors of HIV-1 infection among female prostitutes in Khon Kaen, northeast Thailand. Infection 1992;20:328–31.
408. Saracco A, Musicco M, Nicolosi A, et al. Man-to-woman sexual transmission of HIV: longitudinal study of 343 steady partners of infected men. J Acquir Immune Defic Syndr 1993;6:497–502.
409. Simonsen JN, Plummer FA, Ngugi EN, et al. HIV infection among lower socioeconomic strata prostitutes in Nairobi. AIDS 1990;4:139–44.
410. Sinei SK, Fortney JA, Kigondu CS, et al. Contraceptive use and HIV infection in Kenyan family planning clinic attenders. Int J STD AIDS 1996;7:65–70.
411. Siraprapasiri T, Thanprasertsuk S, Rodklay A, et al. Risk factors for HIV among prostitutes in Chiangmai, Thailand. AIDS 1991;5:579–82.
412. Spence MR, Robbins SM, Polansky M, Schable CA. Seroprevalence of human immunodeficiency virus type I (HIV-1) antibodies in a family-planning population. Sex Transm Dis 1991;18:143–5.
413. Taneepanichskul S, Phuapradit W, Chaturachinda K. Association of contraceptives and HIV-1 infection in Thai female commercial sex workers. Aust N Z J Obstet Gynaecol 1997;37:86–8.
414. Temmerman M, Chomba EN, Ndinya-Achola J, et al. Maternal human immunodeficiency virus-1 infection and pregnancy outcome. Obstet Gynecol 1994;83:495–501.
415. Ungchusak K, Rehle T, Thammapornpilap P, et al. Determinants of HIV infection among female commercial sex workers in northeastern Thailand: results from a longitudinal study. J Acquir Immune Defic Syndr Hum Retrovirol 1996;12:500–7. Eerratum in J Acquir Immune Defic Syndr Hum Retrovirol 1998;18:192.
416. Allen S, Stephenson R, Weiss H, et al. Pregnancy, hormonal contraceptive use, and HIV-related death in Rwanda. J Womens Health (Larchmt) 2007;16:1017–27.
417. Cejtin HE, Jacobson L, Springer G, et al. Effect of hormonal contraceptive use on plasma HIV-1-RNA levels among HIV-infected women. AIDS 2003;17:1702–4.
418. Clark RA, Kissinger P, Williams T. Contraceptive and sexually transmitted diseases protection among adult and adolescent women infected with human immunodeficiency virus. Int J STD AIDS 1996;7:439–42.
419. Clark RA, Theall KP, Amedee AM, et al. Lack of association between genital tract HIV-1 RNA shedding and hormonal contraceptive use in a cohort of Louisiana women. Sex Transm Dis 2007;34:870–2.
420. Clemetson DB, Moss GB, Willerford DM, et al. Detection of HIV DNA in cervical and vaginal secretions. Prevalence and correlates among women in Nairobi, Kenya. JAMA 1993;269:2860–4.
421. Kaul R, Kimani J, Nagelkerke NJ, et al. Risk factors for genital ulcerations in Kenyan sex workers. The role of human immunodeficiency virus type 1 infection. Sex Transm Dis 1997;24:387–92.
422. Kilmarx PH, Limpakarnjanarat K, Kaewkungwal J, et al. Disease progression and survival with human immunodeficiency virus type 1 subtype E infection among female sex workers in Thailand. J Infect Dis 2000;181:1598–606.
423. Kovacs A, Wasserman SS, Burns D, et al. Determinants of HIV-1 shedding in the genital tract of women. Lancet 2001;358:1593–601.
424. Kreiss J, Willerford DM, Hensel M, et al. Association between cervical inflammation and cervical shedding of human immunodeficiency virus DNA. J Infect Dis 1994;170:1597–601.
425. Lavreys L, Chohan V, Overbaugh J, et al. Hormonal contraception and risk of cervical infections among HIV-1-seropositive Kenyan women. AIDS 2004;18:2179–84.
426. Mostad SB, Overbaugh J, DeVange DM, et al. Hormonal contraception, vitamin A deficiency, and other risk factors for shedding of HIV-1 infected cells from the cervix and vagina. Lancet 1997;350:922–7.
427. Richardson BA, Otieno PA, Mbori-Ngacha D, et al. Hormonal contraception and HIV-1 disease progression among postpartum Kenyan women. AIDS 2007;21:749–53.
428. Seck K, Samb N, Tempesta S, et al. Prevalence and risk factors of cervicovaginal HIV shedding among HIV-1 and HIV-2 infected women in Dakar, Senegal. Sex Transm Infect 2001;77:190–3.
429. Stringer EM, Kaseba C, Levy J, et al. A randomized trial of the intrauterine contraceptive device vs hormonal contraception in women who are infected with the human immunodeficiency virus. Am J Obstet Gynecol 2007;197:144–8.
430. Taneepanichskul S, Intaraprasert S, Phuapradit W, Chaturachinda K. Use of Norplant implants in asymptomatic HIV-1 infected women. Contraception 1997;55:205–7.
431. Taneepanichskul S, Tanprasertkul C. Use of Norplant implants in the immediate postpartum period among asymptomatic HIV-1-positive mothers. Contraception 2001;64:39–41.
432. Wang CC, McClelland RS, Overbaugh J, et al. The effect of hormonal contraception on genital tract shedding of HIV-1. AIDS 2004;18:205–9.

433. el-Raghy L, Black DJ, Osman F, Orme ML, Fathalla M. Contraceptive steroid concentrations in women with early active schistosomiasis: lack of effect of antischistosomal drugs. Contraception 1986;33:373–7.
434. Gad-el-Mawla N, Abdallah A. Liver function in bilharzial females receiving contraceptive pills. Acta Hepato 1969;16:308–10.
435. Gad-el-Mawla N, el-Roubi O, Sabet S, Abdallah A. Plasma lipids and lipoproteins in bilharzial females during oral contraceptive therapy. J Egypt Med Assoc 1972;55:137–47.
436. Shaaban MM, Hammad WA, Falthalla MF, et al. Effects of oral contraception on liver function tests and serum proteins in women with active schistosomiasis. Contraception 1982;26:75–82.
437. Shaaban MM, Ghaneimah SA, Mohamed MA, Abdel-Chani S, Mostafa SA. Effective of oral contraception on serum bile acid. Int J Gynaecol Obstet 1984;22:111–5.
438. Sy FS, Osteria TS, Opiniano V, Gler S. Effect of oral contraceptive on liver function tests of women with schistosomiasis in the Philippines. Contraception 1986;34:283–94.
439. Tagy AH, Saker ME, Moussa AA, Kolgah A. The effect of low-dose combined oral contraceptive pills versus injectable contracetpive (Depot Provera) on liver function tests of women with compensated bilharzial liver fibrosis. Contraception 2001;64:173–6.
440. Beck P, Wells SA. Comparison of the mechanisms underlying carbohydrate intolerance in subclinical diabetic women during pregnancy and during post-partum oral contraceptive steroid treatment. J Clin Endocrinol Metab 1969;29:807–18.
441. Kjos SL, Peters RK, Xiang A, et al. Contraception and the risk of type 2 diabetes mellitus in Latina women with prior gestational diabetes mellitus. JAMA 1998;280:533–8.
442. Kung AW, Ma JT, Wong VC, et al. Glucose and lipid metabolism with triphasic oral contraceptives in women with history of gestational diabetes. Contraception 1987;35:257–69.
443. Radberg T, Gustafson A, Skryten A, Karlsson K. Metabolic studies in gestational diabetic women during contraceptive treatment: effects on glucose tolerance and fatty acid composition of serum lipids. Gynecol Obstet Invest 1982;13:17–29.
444. Skouby SO, Molsted-Pedersen L, Kuhl C. Low dosage oral contraception in women with previous gestational diabetes. Obstet Gynecol 1982;59:325–8.
445. Skouby SO, Andersen O, Kuhl C. Oral contraceptives and insulin receptor binding in normal women and those with previous gestational diabetes. Am J Obstet Gynecol 1986;155:802–7.
446. Skouby SO, Andersen O, Saurbrey N, Kuhl C. Oral contraception and insulin sensitivity: in vivo assessment in normal women and women with previous gestational diabetes. J Clin Endocrinol Metab 1987;64:519–23.
447. Xiang AH, Kawakubo M, Kjos SL, Buchanan TA. Long-acting injectable progestin contraception and risk of type 2 diabetes in Latino women with prior gestational diabetes mellitus. Diabetes Care 2006;29:613–7.
448. Kjos SL, Shoupe D, Douyan S, et al. Effect of low-dose oral contraceptives on carbohydrate and lipid metabolism in women with recent gestational diabetes: results of a controlled, randomized, prospective study. Am J Obstet Gynecol 1990;163:1822–7.
449. Radberg T, Gustafson A, Skryten A, Karlsson K. Metabolic studies in women with previous gestational diabetes during contraceptive treatment: effects on serum lipids and high density lipoproteins. Acta Endocrinol (Copenh) 1982;101:134–9.
450. Skouby SO, Kuhl C, Molsted-Pedersen L, Petersen K, Christensen MS. Triphasic oral contraception: metabolic effects in normal women and those with previous gestational diabetes. Am J Obstet Gynecol 1985;153:495–500.
451. Beck P, Arnett DM, Alsever RN, Eaton RP. Effect of contraceptive steroids on arginine-stimulated glucagon and insulin secretion in women. ll. Carbohydrate and lipid phsiology in insulin-dependent diabetics. Metabolism 1976;25:23–31.
452. Diab KM, Zaki MM. Contraception in diabetic women: comparative metabolic study of norplant, depot medroxyprogesterone acetate, low dose oral contraceptive pill and CuT380A. J Obstet Gynecol Res 2000;26:17–26.
453. Garg SK, Chase P, Marshall G, et al. Oral contraceptives and renal and retinal complications in young women with insulin-dependent diabetes mellitus. JAMA 1994;271:1099–102.
454. Grigoryan OR, Grodnitskaya EE, Andreeva EN, et al. Contraception in perimenopausal women with diabetes mellitus. Gynecol Endocrinol 2006;22:198–206.
455. Margolis KL, Adami H-O, Luo J, Ye W, Weiderpass E. A prospective study of oral contraceptive use and risk of myocardial infarction among Swedish women. Fertil Steril 2007;88:310–6.
456. Petersen KR, Skouby SO, Sidelmann J, Jespersen J. Assessment of endothelial function during oral contraception on women with insulin-dependent diabetes mellitus. Metabolism 1994;43:1379–83.
457. Petersen KR, Skouby SO, Jespersen J. Balance of coagulation activity with fibrinolysis during use of oral contraceptives in women with insulin-dependent diabetes millitus. Int J Fertil 1995;40:105–11.
458. Radberg T, Gustafson A, Skryten A, Karlsson k. Oral contraception in diabetic women. A cross-over study on seum and high density lipoprotein (HDL) lipids and diabetes control during progestogen and combined estrogen/progestogen contraception. Horm Metab Res 1982;14:61–5.
459. Skouby SO, Jensen BM, Kuhl C, et al. Hormonal contraception in diabetic women: acceptability and influence on diabetes control and ovarian function of a nonalkylated estrogen/progestogen compound. Contraception 1985;32:23–31.
460. Skouby SO, Molsted-Petersen L, Kuhl C, Bennet P. Oral contraceptives in diabetic womne: metabolic effects of four compounds with different estrogen/progestogen profiles. Fertil Steril 1986;46:858–64.
461. Bitton A, Peppercorn MA, Antonioli DA, et al. Clinical, biological, and histologic parameters as predictors of relapse in ulcerative colitis. Gastroenterology 2001;120:13–20.
462. Cosnes J, Carbonnel F, Carrat F, Beaugerie L, Gendre JP. Oral contraceptive use and the clinical course of Crohn's disease: a prospective cohort study. Gut 1999;45:218–22.
463. Sutherland LR, Ramcharan S, Bryant H, Fick G. Effect of oral contraceptive use on reoperation following surgery for Crohn's disease. Dig Dis Sci 1992;37:1377–82.
464. Timmer A, Sutherland LR, Martin F. Oral contraceptive use and smoking are risk factors for relapse in Crohn's disease. The Canadian Mesalamine for Remission of Crohn's Disease Study Group. Gastroenterology 1998;114:1143–50.
465. Wright JP. Factors influencing first relapse in patients with Crohn's disease. J Clin Gastroenterol 1992;15:12–6.
466. Grimmer SF, Back DJ, Orme ML, et al. The bioavailability of ethinyloestradiol and levonorgestrel in patients with an ileostomy. Contraception 1986;33:51–9.

467. Nilsson LO, Victor A, Kral JG, Johansson ED, Kock NG. Absorption of an oral contraceptive gestagen in ulcerative colitis before and after proctocolectomy and construction of a continent ileostomy. Contraception 1985;31:195–204.
468. Bernstein CN, Blanchard JF, Houston DS, Wajda A. The incidence of deep venous thrombosis and pulmonary embolism among patients with inflammatory bowel disease: a population-based cohort study. Thromb Haemost 2001;85:430–4.
469. Di Martino V, Lebray P, Myers RP, et al. Progression of liver fibrosis in women infected with hepatitis C: long-term benefit of estrogen exposure. Hepatology 2004;40:1426–33.
470. Libbrecht L, Craninx M, Nevens F, Desmet V, Roskams T. Predictive value of liver cell dysplasia for development of hepatocellular carcinoma in patients with non-cirrhotic and cirrhotic chronic viral hepatitis. Histopathology 2001;39:66–73.
471. Eisalo A, Konttinen A, Hietala O. Oral contraceptives after liver disease. Br Med J 1971;3:561–2.
472. Peishan Wang, Zemin Lai, Jinlan Tang, et al. Safety of hormonal steroid contraceptive use for hepatitis B virus carrier women. Pharmacoepidemiol Drug Saf 2000;9:245–6.
473. Shaaban MM, Hammad WA, Fathalla MF, et al. Effects of oral contraception on liver function tests and serum proteins in women with past viral hepatitis. Contraception 1982;26:65–74.
474. Schweitzer IL, Weiner JM, McPeak CM, Thursby MW. Oral contraceptives in acute viral hepatitis. JAMA 1975;233:979–80.
475. D'halluin V, Vilgrain V, Pelletier G, et al. Natural history of focal nodular hyperplasia. A retrospective study of 44 cases [in French]. Gastroenterol Clin Biol 2001;25:1008–10.
476. Mathieu D, Kobeiter H, Maison P, et al. Oral contraceptive use and focal nodular hyperplasia of the liver. Gastroenterology 2000;118:560–4.
477. Pietrzak B, Bobrowska K, Jabiry-Zieniewicz Z, et al. Oral and transdermal hormonal contraception in women after kidney transplantation. Transplant Proc 2007;39:2759–62.
478. Pietrzak B, Kaminski P, Wielgos M, Bobrowska K, Durlik M. Combined oral contraception in women after renal transplantation. Neuro Endocrinol Lett 2 006;27:679–82.
479. Jabiry-Zieniewicz Z, Bobrowska K, Kaminski P, et al. Low-dose hormonal contraception after liver transplantation. Transplant Proc 2007;39:1530–2.
480. Fedorkow DM, Corenblum B, Shaffer EA. Cholestasis induced by oestrogen after liver transplantation. BMJ 1989;299:1080–1.
481. Back DJ, Bates M, Bowden A, et al. The interaction of phenobarbital and other anticonvulsants with oral contraceptive steroid therapy. Contraception 1980;22:495–503.
482. Doose DR, Wang S, Padmanabhan M, et al. Effects of topiramate or carbamazepine on the pharmacokinetics of an oral contraceptive containing norethindrone and ethinyl estradiol in healthy obese and nonobese female subjects. Epilepsia 2003;44:540–9.
483. Fattore C, Cipolla G, Gatti G, et al. Induction of ethinylestradiol and levonorgestrel metabolism by oxcarbazepine in healthy women. Epilepsia 1999;40:783–7.
484. Rosenfeld WE, Doose DR, Walker SA, Nayak RK. Effect of topiramate on the pharmacokinetics of an oral contraceptive containing norethindrone and ethinyl estradiol in patients with epilepsy. Epilepsia 1997;38:317–23.
485. Christensen J, Petrenaite V, Atterman J, et al. Oral contraceptives induce lamotrigine metabolism: evidence from a double-blind, placebo-controlled trial. Epilepsia 2007;48:484–9.
486. Contin M, Albani F, Ambrosetto G, et al. Variation in lamotrigine plasma concentrations with hormonal contraceptive monthly cycles in partiens with epilepsy. Epilepsia 2006;47:1573–5.
487. Reimers A, Helde G, Brodtkorb E. Ethinyl estradiol, not progestogens, reduces lamotrigine serum concentrations. Epilepsia 2005;46:1414–7.
488. Sabers A, Buchholt JM, Uldall P, Hansen EL. Lamotrigine plasma levels reduced by oral contraceptives. Epilepsy Res 2001;47:151–4.
489. Sabers A, Ohman I, Christensen J, Tomson T. Oral contraceptives reduce lamotrigine plasma levels. Neurology 2003;61:570–1.
490. Back DJ, Breckenridge AM, MacIver M, et al. The effects of ampicillin on oral contraceptive steroids in women. Br J Clin Pharmacol 1982;14:43–8.
491. Back DJ, Grimmer SF, Orme ML, et al. Evaluation of the Committee on Safety of Medicines yellow card reports on oral contraceptive-drug interactions with anticonvulsants and antibiotics. Br J Clin Pharmacol 1988;25:527–32.
492. Back DJ, Tjia J, Martin C, et al. The lack of interaction between temafloxacin and combined oral contraceptive steroids. Contraception 1991;43:317–23.
493. Bacon JF, Shenfield GM. Pregnancy attributable to interaction between tetracycline and oral contraceptives. BMJ 1980;280:293.
494. Bainton R. Interaction between antibiotic therapy and contraceptive medication. Oral Surg Oral Med Oral Pathol 1986;61:453–5.
495. Bollen M. Use of antibiotics when taking the oral contraceptive pill [comment]. Aust Fam Physician 1995;24:928–9.
496. Bromham DR. Knowledge and use of secondary contraception among patients requesting termination of pregnancy. BMJ 1993;306:556–7.
497. Cote J. Interaction of griseofulvin and oral contraceptives [comment]. J Am Acad Dermatol 1990;22:124–5.
498. Csemiczky G, Alvendal C, Landgren BM. Risk for ovulation in women taking a low-dose oral contraceptive (Microgynon) when receiving antibacterial treatment with a fluoroquinoline (ofloxacin). Adv Contracept 1996;12:101–9.
499. de Groot AC, Eshuis H, Stricker BH. Inefficiency of oral contraception during use of minocycline [in Dutch]. Ned Tijdschr Geneeskd 1990;134:1227–9.
500. DeSano EA Jr, Hurley SC. Possible interactions of antihistamines and antibiotics with oral contraceptive effectiveness. Fertil Steril 1982;37:853–4.
501. Donley TG, Smith RF, Roy B. Reduced oral contraceptive effectiveness with concurrent antibiotic use: a protocol for prescribing antibiotics to women of childbearing age. Compendium 1990;11:392–6.
502. Friedman CI, Huneke AL, Kim MH, Powell J. The effect of ampicillin on oral contraceptive effectiveness. Obstet Gynecol 1980;55:33–7.
503. Grimmer SF, Allen WL, Back DJ, et al. The effect of cotrimoxazole on oral contraceptive steroids in women. Contraception 1983;28:53–9.
504. Helms SE, Bredle DL, Zajic J, et al. Oral contraceptive failure rates and oral antibiotics. J Am Acad Dermatol 1997;36:705–10.
505. Hempel E, Bohm W, Carol W, Klinger G. Enzyme induction by drugs and hormonal contraception [in German]. Zentralbl Gynakol 1973;95:1451–7.
506. Hempel E, Zorn C, Graf K. Effect of chemotherapy agents and antibiotics on hormonal contraception [in German]. Z Arztl Forbild (Jena) 1978;72:924–6.
507. Hetenyi G. Possible interactions between antibiotics and oral contraceptives. Ther Hung 1989;37:86–9.
508. Hughes BR, Cunliffe WJ. Interactions between the oral contraceptive pill and antibiotics [comment]. Br J Dermatol 1990;122:717–8.

509. Joshi JV, Joshi UM, Sankholi GM, et al. A study of interaction of low-dose combination oral contraceptive with ampicillin and metronidazole. Contraception 1980;22:643–52.
510. Kakouris H, Kovacs GT. Pill failure and nonuse of secondary precautions. Br J Fam Plann 1992;18:41–4.
511. Kakouris H, Kovacs GT. How common are predisposing factors to pill failure among pill users? Br J Fam Plann 1994;20:33–5.
512. Kovacs GT, Riddoch G, Duncombe P, et al. Inadvertent pregnancies in oral contraceptive users. Med J Aust 1989;150:549–51.
513. Lequeux A. Pregnancy under oral contraceptives after treatment with tetracycline] [in French]. Louv Med 1980;99:413–4.
514. London BM, Lookingbill DP. Frequency of pregnancy in acne patients taking oral antibiotics and oral contraceptives. Arch Dermatol 1994;130:392–3.
515. Maggiolo F, Puricelli G, Dottorini M, et al. The effects of ciprofloxacin on oral contraceptive steroid treatments. Drugs Exp Clin Res 1991;17:451–4.
516. Murphy AA, Zacur HA, Charache P, Burkman RT. The effect of tetracycline on levels of oral contraceptives. Am J Obstet Gynecol 1991;164:28–33.
517. Neely JL, Abate M, Swinker M, D'Angio R. The effect of doxycycline on serum levels of ethinyl estradiol, noretindrone, and endogenous progesterone. Obstet Gynecol 1991;77:416–20.
518. Pillans PI, Sparrow MJ. Pregnancy associated with a combined oral contraceptive and itraconazole [comment]. N Z Med J 1993;106:436.
519. Scholten PC, Droppert RM, Zwinkels MG, et al. No interaction between ciprofloxacin and an oral contraceptive. Antimicrob Agents Chemother 1998;42:3266–8.
520. Silber TJ. Apparent oral contraceptive failure associated with antibiotic administration. J Adolesc Health Care 1983;4:287–9.
521. Sparrow MJ. Pill method failures. N Z Med J 1987;100:102–5.
522. Sparrow MJ. Pregnancies in reliable pill takers. N Z Med J 1989;102:575–7.
523. Sparrow MJ. Pill method failures in women seeking abortion—fourteen years experience. N Z Med J 1998;111:386–8.
524. van Dijke CP, Weber JC. Interaction between oral contraceptives and griseofulvin. Br Med J (Clin Res Ed) 1984;288:1125–6.
525. Wermeling DP, Chandler MH, Sides GD, Collins D, Muse KN. Dirithromycin increases ethinyl estradiol clearance without allowing ovulation. Obstet Gynecol 1995;86:78–84.
526. Young LK, Farquhar CM, McCowan LM, Roberts HE, Taylor J. The contraceptive practices of women seeking termination of pregnancy in an Auckland clinic. N Z Med J 1994;107:189–92.
527. Abrams LS, Skee D, Natarajan J, Wong FA. Pharmocokinetic overview of Ortho Evra/Evra. Fertil Steril 2002;77:s3–s12.
528. Dogterom P, van den Heuvel MW, Thomsen T. Absence of pharmacokinetic interactions of the combined contraceptive vaginal ring NuvaRing with oral amoxicillin or doxycycline in two randomized trials. Clin Pharmacokinet 2005;44:429–38.
529. Devenport MH, Crook D, Wynn V, Lees LJ. Metabolic effects of low-dose fluconazole in healthy female users and non-users of oral contraceptives. Br J Clin Pharmacol 1989;27:851–9.
530. Hilbert J, Messig M, Kuye O. Evaluation of interaction between fluconazole and an oral contraceptive in healthy women. Obstet Gynecol 2001;98:218–23.
531. Kovacs I, Somos P, Hamori M. Examination of the potential interaction between ketoconazole (Nizoral) and oral contraceptives with special regard to products of low hormone content (Rigevidon, anteovin). Ther Hung 1986;34:167–70.
532. Lunell NO, Pschera H, Zador G, Carlstrom K. Evaluation of the possible interaction of the antifungal triazole SCH 39304 with oral contraceptives in normal health women. Gynecol Obstet Invest 1991;32:91–7.
533. McDaniel PA, Cladroney RD. Oral contraceptives and griseofulvin interactions. Drug Intell Clin Pharm 1986;20:384.
534. Meyboom RH, van Puijenbroek EP, Vinks MH, Lastdrager CJ. Disturbance of withdrawal bleeding during concomitant use of itraconazole and oral contraceptives. N Z Med J 1997;110:300.
535. Rieth H, Sauerbrey N. Interaction studies with fluconazole, a new tirazole antifungal drug [in German]. Wien Med Wochenschr 1989;139:370–4.
536. Sinofsky FE, Pasquale SA. The effect of fluconazole on circulating ethinyl estradiol levels in women taking oral contraceptives. Am J Obstet Gynecol 1998;178:300–4.
537. van Puijenbroek EP, Feenstra J, Meyboom RH. Pill cycle disturbance in simultaneous use of itraconazole and oral contraceptives [in Dutch]. Ned Tijedschr Geneeskd 1998;142:146–9.
538. van Puijenbroek EP, Egberts AC, Meyboom RH, Leufkens HG. Signalling possible drug-drug interactions in a spontaneous reporting system: delay of withdrawal bleeding during concomitant use of oral contraceptives and itraconazole. Br J Clin Pharmacol 1999;47:689–93.
539. Verhoeven CH, van den Heuvel MW, Mulders TM, Dieben TO. The contraceptive vaginal ring, NuvaRing, and antimycotic co-medication. Contraception 2004;69:129–32.
540. Back DJ, Breckenridge AM, Grimmer SF, Orme ML, Purba HS. Pharmacokinetics of oral contraceptive steroids following the administration of the antimalarial drugs primaquine and chloroquine. Contraception 1984;30:289–95.
541. Croft AM, Herxheimer A. Adverse effects of the antimalaria drug, mefloquine: due to primary liver damage with secondary thyroid involvement? BMC Public Health 2002;2:6.
542. Karbwang J, Looareesuwan S, Back DJ, Migasana S, Bunnag D. Effect of oral contraceptive steroids on the clinical course of malaria infection and on the pharmacokinetics of mefloquine in Thai women. Bull World Health Organ 1988;66:763–7.
543. McGready R, Stepniewska K, Seaton E, et al. Pregnancy and use of oral contaceptives reduces the biotransformation of proguanil to cycloguanil. Eur J Clin Pharmacol 2003;59:553–7.
544. Wanwimolruk S, Kaewvichit S, Tanthayaphinant O, Suwannarach C, Oranratnachai A. Lack of effect of oral contraceptive use on the pharmacokinetics of quinine. Br J Clin Pharmacol 1991;31:179–81.
545. Back DJ, Breckenridge AM, Crawford FE, et al. The effect of rifampicin on norethisterone pharmacokinetics. Eur J Clin Pharmacol 1979;15:193–7.
546. Back DJ, Breckenridge AM, Crawford FE, et al. The effect of rifampicin on the pharmacokinetics of ethynylestradiol in women. Contraception 1980;21:135–43.
547. Barditch-Crovo P, Trapnell CB, Ette E, et al. The effects of rifampicin and rifabutin on the pharmacokinetics and pharmacodynamics of a combination oral contraceptive. Clin Pharmacol Ther 1999;65:428–38.
548. Bolt HM, Bolt M, Kappus H. Interaction of rifampicin treatment with pharmacokinetics and metabolism of ethinyloestradiol in man. Acta Endocrinol (Copenh) 1977;85:189–97.
549. Gupta KC, Ali MY. Failure of oral contraceptive with rifampicin. Med J Zambia 1981;15:23.
550. Hirsch A. Sleeping pills [letter] [in French]. Nouv Presse Med 1973;2:2957.

551. Hirsch A, Tillement JP, Chretien J. Effets contrariants de la rifampicine sur les contraceptifs oraux: a propos de trois grossesses non desiree chez trois malades. Rev Fr Mal Respir 1975;2:174–82.
552. Joshi JV, Joshi UM, Sankholi GM, et al. A study of interaction of a low-dose combination oral contraceptive with anti-tubercular drugs. Contraception 1980;21:617–29.
553. Kropp R. Rifampicin and oral cotnraceptives (author's transl) [in German]. Prax Pneumol 1974;28:270–2.
554. LeBel M, Masson E, Guilbert E, et al. Effects of rifabutin and rifampicin on the pharmacokinetics of ethinylestradiol and norethindrone. J Clin Pharmacol 1998;38:1042–50.
555. Meyer B, Muller F, Wessels P, Maree J. A model to detect interactions between roxithromycin and oral contraceptives. Clin Pharmacol Ther 1990;47:671–4.
556. Nocke-Finke L, Breuer H, Reimers D. Effects of rifampicin on the menstrual cycle and on oestrogen excretion in patients taking oral contraceptives [in German]. Deutsche Med Wochenschr 1973;98:1521–3.
557. Piguet B, Muglioni JF, Chaline G. Oral contraception and rifampicin [letter] [in French]. Nouv Presse Med 1975;4:115–6.
558. Reimers D, Jezek A. The simultaneous use of rifampicin and other antitubercular agents with oral contraceptives [in German]. Prax Pneumol 1971;25:255–62.
559. Skolnick JL, Stoler BS, Katz DB, Anderson WH. Rifampicin, oral contraceptives, and pregnancy. JAMA 1976;236:1382.
560. Szoka PR, Edgren RA. Drug interactions with oral contraceptives: compilation and analysis of an adverse experience report database. Fertil Steril 1988;49:s31–s38.

Appendix C
Classifications for Progestin-Only Contraceptives

Classifications for progestin-only contraceptives (POCs) include those for progestin-only pills, depot medroxyprogesterone acetate, and progestin-only implants (Box). POCs do not protect against sexually transmitted infections (STIs) or human immunodeficiency virus (HIV).

BOX. Categories for Classifying Progestin-Only Contraceptives

1 = A condition for which there is no restriction for the use of the contraceptive method.
2 = A condition for which the advantages of using the method generally outweigh the theoretical or proven risks.
3 = A condition for which the theoretical or proven risks usually outweigh the advantages of using the method.
4 = A condition that represents an unacceptable health risk if the contraceptive method is used.

TABLE. Classifications for progestin-only contraceptives, including progestin-only pills, DMPA, and implants*†

Condition	Category			Clarifications/Evidence/Comments
	POP	DMPA	Implants	
Personal Characteristics and Reproductive History				
Pregnancy	Not applicable	Not applicable	Not applicable	**Clarification:** Use of POCs is not required. There is no known harm to the woman, the course of her pregnancy, or the fetus if POCs are inadvertently used during pregnancy. However, the relation between DMPA use during pregnancy and its effects on the fetus remains unclear.
Age				
a. Menarche to <18 yrs	1	2	1	**Evidence:** Most studies have found that women lose BMD while using DMPA but regain BMD after discontinuing DMPA. It is not known whether DMPA use among adolescents affects peak bone mass levels or whether adult women with long duration of DMPA use can regain BMD to baseline levels before entering menopause. The relation between DMPA-associated changes in BMD during the reproductive years and future fracture risk is unknown (*1–41*). Studies find no effect or have inconsistent results about the effects of POCs other than DMPA on BMD (*42–54*).
b. 18–45 yrs	1	1	1	
c. >45 yrs	1	2	1	
Parity				
a. Nulliparous	1	1	1	
b. Parous	1	1	1	
Breastfeeding				**Clarification:** The U.S. Department of Health and Human Services recommends that infants be exclusively breastfed during the first 4–6 months of life, preferably for a full 6 months. Ideally, breastfeeding should continue through the first year of life (*55*).
a. <1 mo postpartum	2	2	2	
b. 1 mo to <6 mos postpartum	1	1	1	
c. ≥6 mos postpartum	1	1	1	**Evidence:** Despite anecdotal clinical reports that POCs might diminish milk production, direct evidence from available clinical studies demonstrates no significant negative effect of POCs on breastfeeding performance (*56–90*) or on the health of the infant (*66,70,72,76–81,91–93*). In general, these studies are of poor quality, lack standard definitions of breastfeeding or outcome measures, and have not included premature or ill infants. Theoretical concerns about effects of progestin exposure on the developing, neonatal brain are based on studies of progesterone effects in animals; whether similar effects occur after progestin exposure in human neonates is not known.

TABLE. (*Continued*) Classifications for progestin-only contraceptives,*† including progestin-only pills, DMPA, and implants

Condition	Category			Clarifications/Evidence/Comments
	POP	DMPA	Implants	
Postpartum (in nonbreastfeeding women)				
a. <21 days	1	1	1	
b. ≥21 days	1	1	1	
Postabortion				**Clarification:** POCs may be started immediately postabortion.
a. First trimester	1	1	1	
b. Second trimester	1	1	1	**Evidence:** Limited evidence suggests that there are no adverse side effects when implants (Norplant) or progestin-only injectables (NET-EN) are initiated after first trimester abortion (*94–97*).
c. Immediate postseptic abortion	1	1	1	
Past ectopic pregnancy	2	1	1	**Comments:** POP users have a higher absolute rate of ectopic pregnancy than do users of other POCs but still less than using no method.
History of pelvic surgery	1	1	1	
Smoking				
a. Age <35 yrs	1	1	1	
b. Age ≥35 yrs				
i. <15 Cigarettes/day	1	1	1	
ii. ≥15 Cigarettes/day	1	1	1	
Obesity				
a. ≥30 kg/m^2 BMI	1	1	1	
b. Menarche to <18 yrs and ≥30 kg/m^2 BMI	1	2	1	**Evidence:** Obese adolescents who used DMPA were more likely than obese nonusers, obese COC users, and nonobese DMPA users to gain weight. These associations were not observed among adult women. One small study did not observe increases in weight gain among adolescent Norplant users by any category of baseline weight (*98–105*).
History of bariatric surgery§				
a. Restrictive procedures: decrease storage capacity of the stomach (vertical banded gastroplasty, laparoscopic adjustable gastric band, laparoscopic sleeve gastrectomy)	1	1	1	**Evidence:** Limited evidence demonstrated no substantial decrease in effectiveness of oral contraceptives among women who underwent laparoscopic placement of an adjustable gastric band (*106*).
b. Malabsorptive procedures: decrease absorption of nutrients and calories by shortening the functional length of the small intestine (Roux-en-Y gastric bypass, biliopancreatic diversion)	3	1	1	**Evidence:** Limited evidence demonstrated no substantial decrease in effectiveness of oral contraceptives among women who underwent a biliopancreatic diversion (*107*); however, evidence from pharmacokinetic studies suggested conflicting results of oral contraceptive effectiveness among women who underwent a jejunoileal bypass (*108,109*). **Comment:** Bariatric surgical procedures involving a malabsorptive component have the potential to decrease oral contraceptive effectiveness, perhaps further decreased by postoperative complications, such as long-term diarrhea and/or vomiting.
Cardiovascular Disease				
Multiple risk factors for arterial cardiovascular disease (such as older age, smoking, diabetes, and hypertension)	2	3	2	**Clarification:** When multiple major risk factors exist, risk for cardiovascular disease might increase substantially. Some POCs might increase the risk for thrombosis, although this increase is substantially less than with COCs. The effects of DMPA might persist for some time after discontinuation.
Hypertension For all categories of hypertension, classifications are based on the assumption that no other risk factors exist for cardiovascular disease. When multiple risk factors do exist, risk for cardiovascular disease might increase substantially. A single reading of blood pressure level is not sufficient to classify a woman as hypertensive.				
a. Adequately controlled hypertension	1	2	1	**Clarification:** Women adequately treated for hypertension are at lower risk for acute myocardial infarction and stroke than are untreated women. Although no data exist, POC users with adequately controlled and monitored hypertension should be at lower risk for acute myocardial infarction and stroke than are untreated hypertensive POC users.

TABLE. (*Continued*) Classifications for progestin-only contraceptives,*† including progestin-only pills, DMPA, and implants

Condition	Category			Clarifications/Evidence/Comments
	POP	DMPA	Implants	
b. Elevated blood pressure levels (properly taken measurements)				
i. Systolic 140–159 mm Hg or diastolic 90–99 mm Hg	1	2	1	**Evidence:** Limited evidence suggests that among women with hypertension, those who used POPs or progestin-only injectables had a small increased risk for cardiovascular events than did women who did not use these methods (*110*).
ii. Systolic ≥160 mm Hg or diastolic ≥100 mm Hg§	2	3	2	
c. Vascular disease	2	3	2	**Comment:** Concern exists about hypo-estrogenic effects and reduced HDL levels, particularly among users of DMPA. However, there is little concern about these effects with regard to POPs. The effects of DMPA might persist for some time after discontinuation
History of high blood pressure during pregnancy (where current blood pressure is measurable and normal)	1	1	1	
Deep venous thrombosis (DVT)/ Pulmonary embolism (PE)				
a. History of DVT/PE, not on anticoagulant therapy				
i. Higher risk for recurrent DVT/PE (≥1 risk factors)	2	2	2	
• History of estrogen-associated DVT/PE				
• Pregnancy-associated DVT/PE				
• Idiopathic DVT/PE				
• Known thrombophilia, including antiphospholipid syndrome				
• Active cancer (metastatic, on therapy, or within 6 mos after clinical remission), excluding non-melanoma skin cancer				
• History of recurrent DVT/PE				
ii Lower risk for recurrent DVT/PE (no risk factors)	2	2	2	
b. Acute DVT/PE	2	2	2	**Evidence:** No direct evidence exists on use of POCs among women with acute DVT/PE. Although findings on the risk for venous thrombosis with use of POCs in otherwise healthy women is inconsistent, any small increased risk is substantially less than that with COCs (*110–112*).
c. DVT/PE and established on anticoagulant therapy for at least 3 mos				**Evidence:** No direct evidence exists on use of POCs among women with DVT/PE on anticoagulant therapy. Although findings on the risk for venous thrombosis with use of POCs are inconsistent in otherwise healthy women, any small increased risk is substantially less than that with COCs (*110–112*).
i. Higher risk for recurrent DVT/PE (≥1 risk factors)	2	2	2	
• Known thrombophilia, including antiphospholipid syndrome				Limited evidence indicates that intramuscular injections of DMPA in women on chronic anticoagulation therapy does not pose a significant risk for hematoma at the injection site or increase the risk for heavy or irregular vaginal bleeding (*113*).
• Active cancer (metastatic, on therapy, or within 6 mos after clinical remission), excluding non-melanoma skin cancer				
• History of recurrent DVT/PE				
ii. Lower risk for recurrent DVT/PE (no risk factors)	2	2	2	
d. Family history (first-degree relatives)	1	1	1	
e. Major surgery				
i. With prolonged immobilization	2	2	2	
ii. Without prolonged immobilization	1	1	1	
f. Minor surgery without immobilization	1	1	1	

TABLE. (*Continued*) Classifications for progestin-only contraceptives,*† including progestin-only pills, DMPA, and implants

Condition	Category			Clarifications/Evidence/Comments
	POP	DMPA	Implants	
Known thrombogenic mutations§ (e.g., factor V Leiden; prothrombin mutation; protein S, protein C, and antithrombin deficiencies)	2	2	2	**Clarification:** Routine screening is not appropriate because of the rarity of the conditions and the high cost of screening.
Superficial venous thrombosis				
a. Varicose veins	1	1	1	
b. Superficial thrombophlebitis	1	1	1	
Current and history of ischemic heart disease§	Initiation: 2 / Continuation: 3	3	Initiation: 2 / Continuation: 3	**Comment:** Concern exists about hypo-estrogenic effects and reduced HDL levels, particularly among users of DMPA. However, there is little concern about these effects with regard to POPs. The effects of DMPA might persist for some time after discontinuation.
Stroke§ (history of cerebrovascular accident)	Initiation: 2 / Continuation: 3	3	Initiation: 2 / Continuation: 3	**Comment:** Concern exists about hypo-estrogenic effects and reduced HDL levels, particularly among users of DMPA. However, there is little concern about these effects with regard to POPs. The effects of DMPA may persist for some time after discontinuation.
Known hyperlipidemias	2	2	2	**Clarification:** Routine screening is not appropriate because of the rarity of the conditions and the high cost of screening. Some types of hyperlipidemias are risk factors for vascular disease.
Valvular heart disease				
a. Uncomplicated	1	1	1	
b. Complicated§ (pulmonary hypertension, risk for atrial fibrillation, history of subacute bacterial endocarditis)	1	1	1	
Peripartum cardiomyopathy§				
a. Normal or mildly impaired cardiac function (New York Heart Association Functional Class I or II: patients with no limitation of activities or patients with slight, mild limitation of activity) (*114*)				**Evidence:** No direct evidence exists on the safety of POCs among women with peripartum cardiomyopathy. Limited indirect evidence from noncomparative studies of women with cardiac disease demonstrated few cases of hypertension, thromboembolism, and heart failure in women with cardiac disease using POPs and DMPA (*115,116*). **Comment:** Progestin-only implants might induce cardiac arrhythmias in healthy women; women with peripartum cardiomyopathy have a high incidence of cardiac arrhythmias.
i. <6 mos	1	1	1	
ii. ≥6 mos	1	1	1	
b. Moderately or severely impaired cardiac function (New York Heart Association Functional Class III or IV: patients with marked limitation of activity or patients who should be at complete rest) (*114*)	2	2	2	**Evidence:** No direct evidence exists on the safety of POCs among women with peripartum cardiomyopathy. Limited indirect evidence from noncomparative studies of women with cardiac disease demonstrated few cases of hypertension, thromboembolism, and heart failure in women with cardiac disease using POPs and DMPA (*115,116*). **Comment:** Progestin-only implants might induce cardiac arrhythmias in healthy women; women with peripartum cardiomyopathy have a high incidence of cardiac arrhythmias.

TABLE. (*Continued*) Classifications for progestin-only contraceptives,*† including progestin-only pills, DMPA, and implants

Condition	Category: POP	Category: DMPA	Category: Implants	Clarifications/Evidence/Comments
Rheumatic Diseases				
Systemic lupus erythematosus (SLE)§				Persons with SLE are at increased risk for ischemic heart disease, stroke, and VTE. Categories assigned to such conditions in the MEC should be the same for women with SLE who present with these conditions. For all categories of SLE, classifications are based on the assumption that no other risk factors for cardiovascular disease are present; these classifications must be modified in the presence of such risk factors. Many women with SLE can be considered good candidates for most contraceptive methods, including hormonal contraceptives (*117–135*).
a. Positive (or unknown) antiphospholipid antibodies	3	Initiation 3 / Continuation 3	3	**Evidence:** Antiphospholipid antibodies are associated with a higher risk for both arterial and venous thrombosis (*136,137*).
b. Severe thrombocytopenia	2	3	2	2	**Comment:** Severe thrombocytopenia increases the risk for bleeding. POCs might be useful in treating menorrhagia in women with severe thrombocytopenia. However, given the increased or erratic bleeding that may be seen on initiation of DMPA and its irreversibility for 11–13 weeks after administration, initiation of this method in women with severe thrombocytopenia should be done with caution.
c. Immunosuppressive treatment	2	2	2	2	
d. None of the above	2	2	2	2	
Rheumatoid arthritis				
a. On immunosuppressive therapy	1	2/3	1	**Clarification:** DMPA use among women on long-term corticosteroid therapy with a history of, or with risk factors for, nontraumatic fractures is classified as Category 3. Otherwise, DMPA use for women with rheumatoid arthritis is classified as Category 2. **Evidence:** Limited evidence shows no consistent pattern of improvement or worsening of rheumatoid arthritis with use of oral contraceptives (*138–143*), progesterone (*144*), or estrogen (*145*).
b. Not on immunosuppressive therapy	1	2	1	
Neurologic Conditions				
Headaches	Initiation / Continuation	Initiation / Continuation	Initiation / Continuation	**Clarification:** Classification depends on accurate diagnosis of severe headaches that are migrainous and headaches that are not. Any new headaches or marked changes in headaches should be evaluated. Classification is for women without any other risk factors for stroke. Risk for stroke increases with age, hypertension, and smoking. **Comment:** Aura is a specific focal neurologic symptom. For more information about this and other diagnostic criteria, see: Headache Classification Subcommittee of the International Headache Society. The international classification of headache disorders. 2nd Ed. Cephalalgia. 2004;24 (Suppl 1):1–150. http://www.i-h-s.org/upload/ct_clas/ihc_II_main_no_print.pdf. Concern exists that severe headaches might increase with use of DMPA and implants. The effects of DMPA may persist for some time after discontinuation.
a. Non-migrainous (mild or severe)	1 / 1	1 / 1	1 / 1	
b. Migraine				
i. Without aura				
• Age <35 yrs	1 / 2	2 / 2	2 / 2	
• Age ≥35 yrs	1 / 2	2 / 2	2 / 2	
ii. With aura, at any age	2 / 3	2 / 3	2 / 3	
Epilepsy§	1	1	1	**Clarification:** If a woman is taking anticonvulsants, refer to the section on drug interactions. Certain anticonvulsants lower POC effectiveness.
Depressive Disorders				
Depressive disorders	1	1	1	**Clarification:** The classification is based on data for women with selected depressive disorders. No data on bipolar disorder or postpartum depression were available. A potential exists for drug interactions between certain antidepressant medications and hormonal contraceptives. **Evidence:** POC use did not increase depressive symptoms in women with depression compared with baseline (*146–149*).

TABLE. (*Continued*) Classifications for progestin-only contraceptives,*† including progestin-only pills, DMPA, and implants

Condition	Category			Clarifications/Evidence/Comments
	POP	DMPA	Implants	
Reproductive Tract Infections and Disorders				
Vaginal bleeding patterns				
a. Irregular pattern without heavy bleeding	2	2	2	**Comment:** Irregular menstrual bleeding patterns are common among healthy women. POC use frequently induces an irregular bleeding pattern. Implant use might induce irregular bleeding patterns, especially during the first 3–6 months, but these patterns may persist longer.
b. Heavy or prolonged bleeding (includes regular and irregular patterns)	2	2	2	**Clarification:** Unusually heavy bleeding should raise the suspicion of a serious underlying condition.
Unexplained vaginal bleeding (suspicious for serious condition)				**Clarification:** If pregnancy or an underlying pathological condition (such as pelvic malignancy) is suspected, it must be evaluated and the category adjusted after evaluation.
Before evaluation	2	3	3	**Comment:** POCs might cause irregular bleeding patterns, which might mask symptoms of underlying pathology. The effects of DMPA might persist for some time after discontinuation.
Endometriosis	1	1	1	
Benign ovarian tumors (including cysts)	1	1	1	
Severe dysmenorrhea	1	1	1	
Gestational trophoblastic disease				
a. Decreasing or undetectable β–hCG levels	1	1	1	
b. Persistently elevated β-hCG levels or malignant disease§	1	1	1	
Cervical ectropion	1	1	1	
Cervical intraepithelial neoplasia	1	2	2	**Evidence:** Among women with persistent HPV infection, long-term DMPA use (≥5 years) might increase the risk for carcinoma in situ and invasive carcinoma (*150*).
Cervical cancer (awaiting treatment)	1	2	2	**Comment:** Theoretical concern exists that POC use might affect prognosis of the existing disease. While awaiting treatment, women may use POCs. In general, treatment of this condition can render a woman sterile.
Breast disease				
a. Undiagnosed mass	2	2	2	**Clarification:** Evaluation should be pursued as early as possible.
b. Benign breast disease	1	1	1	
c. Family history of cancer	1	1	1	
d. Breast cancer§				
i. Current	4	4	4	**Comment:** Breast cancer is a hormonally sensitive tumor, and the prognosis for women with current or recent breast cancer might worsen with POC use.
ii. Past and no evidence of current disease for 5 years	3	3	3	
Endometrial hyperplasia	1	1	1	
Endometrial cancer§	1	1	1	**Comment:** While awaiting treatment, women may use POCs. In general, treatment of this condition renders a woman sterile.
Ovarian cancer§	1	1	1	**Comment:** While awaiting treatment, women may use POCs. In general, treatment of this condition can render a woman sterile.
Uterine fibroids	1	1	1	**Comment:** POCs do not appear to cause growth of uterine fibroids.

TABLE. (*Continued*) Classifications for progestin-only contraceptives,*† including progestin-only pills, DMPA, and implants

Condition	Category			Clarifications/Evidence/Comments
	POP	DMPA	Implants	
Pelvic inflammatory disease (PID)				
a. Past PID (assuming no current risk factors for STIs)				**Comment:** Whether POCs, like COCs, reduce the risk for PID among women with STIs is unknown, but they do not protect against HIV or lower genital tract STI.
i. With subsequent pregnancy	1	1	1	
ii. Without subsequent pregnancy	1	1	1	
b. Current PID	1	1	1	
STIs				
a. Current purulent cervicitis or chlamydial infection or gonorrhea	1	1	1	
b. Other STIs (excluding HIV and hepatitis)	1	1	1	
c. Vaginitis (including *Trichomonas vaginalis* and bacterial vaginosis)	1	1	1	
d. Increased risk for STIs	1	1	1	**Evidence:** Evidence suggests a possible increased risk for chlamydial cervicitis among DMPA users at high risk for STIs. For other STIs, either evidence exists of no association between DMPA use and STI acquisition or evidence is too limited to draw any conclusions. No evidence is available about other POCs (*151–158*)
HIV/AIDS				
High risk for HIV	1	1	1	**Evidence:** The balance of the evidence suggests no association between POC use and HIV acquisition, although findings from studies of DMPA use conducted among higher risk populations have been inconsistent (*159–183*).
HIV infection§	1	1	1	**Evidence:** Most studies suggest no increased risk for HIV disease progression with hormonal contraceptive use, as measured by changes in CD4 cell count, viral load, or survival. Studies observing that women with HIV who use hormonal contraception have increased risks for STIs are generally consistent with reports among uninfected women. One direct study found no association between hormonal contraceptive use and increased risk for HIV transmission to uninfected partners; several indirect studies reported mixed results about whether hormonal contraception is associated with increased risk for HIV-1 DNA or RNA shedding from the genital tract (*171,184–200*).
AIDS§	1	1	1	**Clarification:** Drug interactions might exist between hormonal contraceptives and ARV drugs; refer to the section on drug interactions.
Other Infections				
Schistosomiasis				
a. Uncomplicated	1	1	1	**Evidence:** Among women with uncomplicated schistosomiasis, limited evidence showed that DMPA use had no adverse effects on liver function (*201*).
b. Fibrosis of liver§ (if severe, see cirrhosis)	1	1	1	
Tuberculosis§				**Clarification:** If a woman is taking rifampicin, refer to the section on drug interactions. Rifampicin is likely to decrease the effectiveness of some POCs.
a. Nonpelvic	1	1	1	
b. Pelvic	1	1	1	
Malaria	1	1	1	

TABLE. (*Continued*) Classifications for progestin-only contraceptives,*† including progestin-only pills, DMPA, and implants

Condition	Category			Clarifications/Evidence/Comments
	POP	DMPA	Implants	
Endocrine Conditions				
Diabetes				
a. History of gestational disease	1	1	1	**Evidence:** POCs had no adverse effects on serum lipid levels in women with a history of gestational diabetes in 2 small studies. (*202,203*) Limited evidence is inconsistent about the development of noninsulin-dependant diabetes among users of POCs with a history of gestational diabetes (*204–207*).
b. Nonvascular disease				
i. Noninsulin-dependent	2	2	2	**Evidence:** Among women with insulin- or noninsulin-dependent diabetes, limited evidence on use of POCs (POPs, DMPA, LNG implant) suggests that these methods have little effect on short-term or long-term diabetes control (e.g., glycosylated hemoglobin levels), hemostatic markers, or lipid profile (*208–211*).
ii. Insulin-dependent§	2	2	2	
c. Nephropathy/retinopathy/ neuropathy§	2	3	2	**Comment:** Concern exists about hypo-estrogenic effects and reduced HDL levels, particularly among users of DMPA. The effects of DMPA might persist for some time after discontinuation. Some POCs might increase the risk for thrombosis, although this increase is substantially less than with COCs.
d. Other vascular disease or diabetes of >20 yrs' duration§	2	3	2	**Comment:** Concern exists about hypo-estrogenic effects and reduced HDL levels, particularly among users of DMPA. The effects of DMPA might persist for some time after discontinuation. Some POCs might increase the risk for thrombosis, although this increase is substantially less than with COCs.
Thyroid disorders				
a. Simple goiter	1	1	1	
b. Hyperthyroid	1	1	1	
c. Hypothyroid	1	1	1	
Gastrointestinal Conditions				
Inflammatory bowel disease (IBD) (ulcerative colitis, Crohn disease)	2	2	1	**Evidence:** Risk for disease relapse among women with IBD using oral contraceptives (most studies did not specify formulation) did not increase significantly from that for nonusers (*212–216*). **Comment:** Absorption of POPs among women with IBD might be reduced if the woman has substantial malabsorption caused by severe disease or small bowel surgery. Women with IBD have a higher prevalence than the general population of osteoporosis and osteopenia. Use of DMPA, which has been associated with small changes in BMD, might be of concern.
Gallbladder disease				
a. Symptomatic				
i. Treated by cholecystectomy	2	2	2	
ii. Medically treated	2	2	2	
iii. Current	2	2	2	
b. Asymptomatic	2	2	2	
History of cholestasis				
a. Pregnancy-related	1	1	1	
b. Past COC–related	2	2	2	**Comment:** Theoretically, a history of COC-related cholestasis might predict subsequent cholestasis with POC use. However, this has not been documented.
Viral hepatitis				
a. Acute or flare	1	1	1	
b. Carrier	1	1	1	
c. Chronic	1	1	1	

TABLE. (*Continued*) Classifications for progestin-only contraceptives,*† including progestin-only pills, DMPA, and implants

Condition	Category			Clarifications/Evidence/Comments
	POP	DMPA	Implants	
Cirrhosis				
a. Mild (compensated)	1	1	1	
b. Severe§ (decompensated)	3	3	3	
Liver tumors				
a. Benign				**Evidence:** Limited direct evidence suggests that hormonal contraceptive use does not influence either progression or regression of liver lesions among women with focal nodular hyperplasia (*217*,*218*).
i. Focal nodular hyperplasia	2	2	2	
ii. Hepatocellular adenoma§	3	3	3	**Comment:** No evidence is available about hormonal contraceptive use among women with hepatocellular adenoma. COC use in healthy women is associated with development and growth of hepatocellular adenoma; whether other hormonal contraceptives have similar effects is not known.
b. Malignant§ (hepatoma)	3	3	3	
Anemias				
Thalassemia	1	1	1	
Sickle cell disease§	1	1	1	**Evidence:** Among women with sickle cell disease, POC use did not have adverse effects on hematologic parameters and, in some studies, was beneficial with respect to clinical symptoms (*219–226*).
Iron deficiency anemia	1	1	1	**Comment:** Changes in the menstrual pattern associated with POC use have little effect on hemoglobin levels.
Solid Organ Transplantation				
Solid organ transplantaton§				
a. Complicated: graft failure (acute or chronic), rejection, cardiac allograft vasculopathy	2	2	2	
b. Uncomplicated	2	2	2	
Drug Interactions				
Antiretroviral (ARV) therapy				**Clarification:** ARV drugs have the potential to either decrease or increase the bioavailability of steroid hormones in hormonal contraceptives. Limited data (Appendix M) suggest potential drug interactions between many ARV drugs (particularly some NNRTIs and ritonavir-boosted protease inhibitors) and hormonal contraceptives. These interactions may alter the safety and effectiveness of both the hormonal contraceptive and the ARV drug. Thus, if a woman on ARV treatment decides to initiate or continue hormonal contraceptive use, the consistent use of condoms is recommended to both prevent HIV transmission and compensate for any possible reduction in the effectiveness of the hormonal contraceptive.
a. Nucleoside reverse transcriptase inhibitors (NRTIs)	1	1	1	
b. Non-nucleoside reverse transcriptase inhibitors (NNRTIs)	2	1	2	
c. Ritonavir-boosted protease inhibitors	3	1	2	
Anticonvulsant therapy				
a. Certain anticonvulsants (phenytoin, carbamazepine, barbiturates, primidone, topiramate, oxcarbazepine)	3	1	2	**Clarification:** Although the interaction of certain anticonvulsants with POPs and ETG implants is not harmful to women, it is likely to reduce the effectiveness of POPs and ETG implants. Whether increasing the hormone dose of POPs alleviates this concern remains unclear. Use of other contraceptives should be encouraged for women who are long-term users of any of these drugs. Use of DMPA is a Category 1 because its effectiveness is not decreased by use of certain anticonvulsants. **Evidence:** Use of certain anticonvulsants may decrease the effectiveness of POCs (*227–229*)
b. Lamotrigine	1	1	1	**Evidence:** No drug interactions have been reported among epileptic women taking lamotrigine and using POCs (*230*)

TABLE. (*Continued*) Classifications for progestin-only contraceptives,*† including progestin-only pills, DMPA, and implants

Condition	Category			Clarifications/Evidence/Comments
	POP	DMPA	Implants	
Antimicrobial therapy				
a. Broad-spectrum antibiotics	1	1	1	
b. Antifungals	1	1	1	
c. Antiparasitics	1	1	1	
d. Rifampicin or rifabutin therapy	3	1	2	**Clarification:** Although the interaction of rifampicin or rifabutin with POPs and ETG implants is not harmful to women, it is likely to reduce the effectiveness of POPs and ETG implants. Use of other contraceptives should be encouraged for women who are long-term users of any of these drugs. Use of DMPA is a Category 1 because its effectiveness is not decreased by use of rifampicin or rifabutin. Whether increasing the hormone dose of POPs alleviates this concern remains unclear.

* Abbreviations: STI = sexually transmitted infection; HIV = human immunodeficiency virus; POC = progestin-only contraceptive; DMPA = depot medroxyprogesterone acetate; BMD = bone mineral density; NET-EN = norethisterone enantate; BMI = body mass index; COC = combined oral contraceptive; HDL = high-density lipoprotein; POP = progestin-only pill; DVT = deep venous thrombosis; PE = pulmonary embolism; SLE = systemic lupus erythematosus; VTE = venous thromboembolism; MEC = Medical Eligibility Criteria; hCG = human chorionic gonadotropin; HPV = human papillomavirus; PID = pelvic inflammatory disease; AIDS = acquired immunodeficiency syndrome; IBD = inflammatory bowel disease; ARV = antiretroviral; LNG = levonorgestrel; NRTI = nucleoside reverse transcriptase inhibitor; NNRTI = non-nucleoside reverse transcriptase inhibitor; ETG = etonogestrel.

† POCs do not protect against STI/HIV. If risk exists for STI/HIV (including during pregnancy or postpartum), the correct and consistent use of condoms is recommended, either alone or with another contraceptive method. Consistent and correct use of the male latex condom reduces the risk for STIs and HIV transmission.

§ Condition that exposes a woman to increased risk as a result of unintended pregnancy.

References

1. Albertazzi P, Bottazzi M, Steel SA. Bone mineral density and depot medroxyprogesterone acetate. Contraception 2006;73:577–83.
2. Banks E, Berrington A, Casabonne D. Overview of the relationship between use of progestogen-only contraceptives and bone mineral density. BJOG Br J Obstet Gynaecol 2001;108:1214–21.
3. Beksinska M, Smit J, Kleinschmidt I, Farley T, Mbatha F. Bone mineral density in women aged 40–49 years using depot-medroxyprogesterone acetate, norethisterone enanthate or combined oral contraceptives for contraception. Contraception 2005;71:170–5.
4. Beksinska ME, Kleinschmidt I, Smit JA, Farley TM. Bone mineral density in adolescents using norethisterone enanthate, depot-medroxyprogesterone acetate or combined oral contraceptives for contraception. Contraception 2007;75:438–43.
5. Berenson AB, Breitkopf CR, Grady JJ, Rickert VI, Thomas A. Effects of hormonal contraception on bone mineral density after 24 months of use. Obstet Gynecol 2004;103:899–906.
6. Busen NH, Britt RB, Rianon N. Bone mineral density in a cohort of adolescent women using depot medroxyprogesterone acetate for one to two years. J Adolesc Health 2003;32:257–9.
7. Clark MK, Sowers M, Levy B, Nichols S. Bone mineral density loss and recovery during 48 months in first-time users of depot medroxyprogesterone acetate. Fertil Steril 2006;86:1466–74.
8. Cromer BA, Blair JM, Mahan JD, Zibners L, Naumovski Z. A prospective comparison of bone density in adolescent girls receiving depot medroxyprogesterone acetate (Depo-Provera), levonorgestrel (Norplant), or oral contraceptives. J Pediatr 1996;129:671–6.
9. Cromer BA, Stager M, Bonny A, et al. Depot medroxyprogesterone acetate, oral contraceptives and bone mineral density in a cohort of adolescent girls. J Adolesc Health 2004;35:434–41.
10. Cromer BA, Lazebnik R, Rome E, et al. Double-blinded randomized controlled trial of estrogen supplementation in adolescent girls who receive depot medroxyprogesterone acetate for contraception. Am J Obstet Gynecol 2005;192:42-7.
11. Cromer BA, Bonny AE, Stager M, et al. Bone mineral density in adolescent females using injectable or oral contraceptives: a 24-month prospective study. Fertil Steril 2008.
12. Cundy T, Cornish J, Evans MC, Roberts H, Reid IR. Recovery of bone density in women who stop using medroxyprogesterone acetate. BMJ 1994;308:247–8.
13. Cundy T, Cornish J, Roberts H, Elder H, Reid IR. Spinal bone density in women using depot medroxyprogesterone contraception. Obstet Gynecol 1998;92:569–73.
14. Cundy T, Cornish J, Roberts H, Reid IR. Menopausal bone loss in long-term users of depot medroxyprogesterone acetate contraception. Am J Obstet Gynecol 2002;186:978–83.
15. Cundy T, Ames R, Horne A, et al. A randomized controlled trial of estrogen replacement therapy in long-term users of depot medroxyprogesterone acetate. J Clin Endocrinol Metab 2003;88:78–81.
16. Gbolade B, Ellis S, Murby B, Randall S, Kirkman R. Bone density in long term users of depot medroxyprogesterone acetate. Br J Obstet Gynaecol 1998;105:790–4.
17. Kaunitz AM, Miller PD, Rice VM, Ross D, McClung MR. Bone mineral density in women aged 25–35 years receiving depot medroxyprogesterone acetate: recovery following discontinuation. Contraception 2006;74:90–9.
18. Kaunitz AM, Arias R, McClung M. Bone density recovery after depot medroxyprogesterone acetate injectable contraception use. Contraception 2008;77:67–76.
19. Lappe JM, Stegman MR, Recker RR. The impact of lifestyle factors on stress fractures in female Army recruits. Osteopor Int 2001;12:35–42.
20. Lara-Torre E, Edwards CP, Perlman S, Hertweck SP. Bone mineral density in adolescent females using depot medroxyprogesterone acetate. J Pediatr Adolesc Gynecol 2004;17:17–21.
21. Lopez LM, Grimes DA, Schulz KF, Curtis KM. Steroidal contraceptives: effect on bone fractures in women. Cochrane Database Syst Rev 2006;CD006033.
22. McGough P, Bigrigg A. Effect of depot medroxyprogesterone acetate on bone density in a Scottish industrial city. Eur J Contracept Reprod Health Care 2007;12:253–9.
23. Merki-Feld GS, Neff M, Keller PJ. A 2-year prospective study on the effects of depot medroxyprogesterone acetate on bone mass-response to estrogen and calcium therapy in individual users. Contraception 2003;67:79–86.

24. Orr-Walker BJ, Evans MC, Ames RW, et al. The effect of past use of the injectable contraceptive depot medroxyprogesterone acetate on bone mineral density in normal postmenopausal women. Clini Endocrinol 1998;49:615–8.
25. Ott SM, Scholes D, LaCroix AZ, et al. Effects of contraceptive use on bone biochemical markers in young women. J Clin Endocrinol Metab 2001;86:179–85.
26. Paiva LC, Pinto-Neto AM, Faundes A. Bone density among long-term users of medroxyprogesterone acetate as a contraceptive. Contraception 1998;58:351–5.
27. Perrotti M, Bahamondes L, Petta C, Castro S. Forearm bone density in long-term users of oral combined contraceptives and depot medroxyprogesterone acetate. Fertil Steril 2001;76:469–73.
28. Petitti DB, Piaggio G, Mehta S, Cravioto MC, Meirik O. Steroid hormone contraception and bone mineral density: a cross-sectional study in an international population. The WHO Study of Hormonal Contraception and Bone Health. Obstet Gynecol 2000;95:736–44.
29. Rosenberg L, Zhang Y, Constant D, et al. Bone status after cessation of use of injectable progestin contraceptives. Contraception 2007;76:425–31.
30. Scholes D, LaCroix AZ, Ott SM, Ichikawa LE, Barlow WE. Bone mineral density in women using depot medroxyprogesterone acetate for contraception. Obstet Gynecol 1999;93:233–8.
31. Scholes D, LaCroix AZ, Ichikawa LE, Barlow WE, Ott SM. Injectable hormone contraception and bone density: results from a prospective study. Epidemiology 2002;13:581–7.
32. Scholes D, LaCroix AZ, Ichikawa LE, Barlow WE, Ott SM. The association between depot medroxyprogesterone acetate contraception and bone mineral density in adolescent women. Contraception 2004;69:99–104.
33. Scholes D, LaCroix AZ, Ichikawa LE, Barlow WE, Ott SM. Change in bone mineral density among adolescent women using and discontinuing depot medroxyprogesterone acetate contraception. Arch Pediatr Adolesc Med 2005;159:139–44.
34. Shaarawy M, El-Mallah SY, Seoudi S, Hassan M, Mohsen IA. Effects of the long-term use of depot medroxyprogesterone acetate as hormonal contraceptive on bone mineral density and biochemical markers of bone remodeling. Contraception 2006;74:297–302.
35. Tang OS, Tang G, Yip P, Li B, Fan S. Long-term depot-medroxyprogesterone acetate and bone mineral density. Contraception 1999;59:25–9.
36. Tang OS, Tang G, Yip PS, Li B. Further evaluation on long-term depot-medroxyprogesterone acetate use and bone mineral density: a longitudinal cohort study. Contraception 2000;62:161–4.
37. Tharnprisarn W, Taneepanichskul S. Bone mineral density in adolescent and young Thai girls receiving oral contraceptives compared with depot medroxyprogesterone acetate: a cross-sectional study in young Thai women. Contraception 2002;66:101–3.
38. Virutamasen P, Wangsuphachart S, Reinprayoon D, et al. Trabecular bone in long-term depot-medroxyprogesterone acetate users. Asia-Oceania J Obstet Gynaecol 1994;20:269–74.
39. Walsh JS, Eastell R, Peel NF. Effects of depot medroxyprogesterone acetate on bone density and bone metabolism before and after peak bone mass: a case-control study. J Clin Endocrinol Metab 2008.
40. Wanichsetakul P, Kamudhamas A, Watanaruangkovit P, Siripakarn Y, Visutakul P. Bone mineral density at various anatomic bone sites in women receiving combined oral contraceptives and depot-medroxyprogesterone acetate for contraception. Contraception 2002;65:407–10.
41. Wetmore CM, Ichikawa L, LaCroix AZ, Ott SM, Scholes D. Association between caffeine intake and bone mass among young women: potential effect modification by depot medroxyprogesterone acetate use. Osteoporos Int 200819:519–27.
42. Bahamondes L, Perrotti M, Castro S, et al. Forearm bone density in users of Depo-Provera as a contraceptive method. Fertil Steril 1999;71:849–52.
43. Bahamondes L, Monteiro-Dantas C, Espejo-Arce X, et al. A prospective study of the forearm bone density of users of etonorgestrel- and levonorgestrel-releasing contraceptive implants. Hum Reprod 2006;21:466–70.
44. Bahamondes L, Espejo-Arce X, Hidalgo MM, et al. A cross-sectional study of the forearm bone density of long-term users of levonorgestrel-releasing intrauterine system. Hum Reprod 2006;21:1316–9.
45. Beerthuizen R, van Beek A, Massai R, et al. Bone mineral density during long-term use of the progestagen contraceptive implant Implanon compared to a non-hormonal method of contraception. Hum Reprod 2000;15:118–22.
46. Caird LE, Reid-Thomas V, Hannan WJ, Gow S, Glasier AF. Oral progestogen-only contraception may protect against loss of bone mass in breast-feeding women. Clin Endocrinol (Oxf) 1994;41:739–45.
47. Di X, Li Y, Zhang C, Jiang J, Gu S. Effects of levonorgestrel-releasing subdermal contraceptive implants on bone density and bone metabolism. Contraception 1999;60:161–6.
48. Diaz S, Reyes MV, Zepeda A, et al. Norplant((R)) implants and progesterone vaginal rings do not affect maternal bone turnover and density during lactation and after weaning. Hum Reprod 1999;14:2499–505.
49. Intaraprasert S, Taneepanichskul S, Theppisai U, Chaturachinda K. Bone density in women receiving Norplant implants for contraception. J Med Assoc Thai 1997;80:738–41.
50. Monteiro-Dantas C, Espejo-Arce X, Lui-Filho JF, et al. A three-year longitudinal evaluation of the forearm bone density of users of etonogestrel- and levonorgestrel-releasing contraceptive implants. Reprod Health 2007;4:11.
51. Naessen T, Olsson SE, Gudmundson J. Differential effects on bone density of progestogen-only methods for contraception in premenopausal women. Contraception 1995;52:35–9.
52. Taneepanichskul S, Intaraprasert S, Theppisai U, Chaturachinda K. Bone mineral density during long-term treatment with Norplant implants and depot medroxyprogesterone acetate. A cross-sectional study of Thai women. Contraception 1997;56:153–5.
53. Taneepanichskul S, Intaraprasert S, Theppisai U, Chaturachinda K. Bone mineral density in long-term depot medroxyprogesterone acetate acceptors. Contraception 1997;56:1–3.
54. Vanderjagt DJ, Sagay AS, Imade GE, Farmer SE, Glew RH. Effect of Norplant contraceptive on the bones of Nigerian women as assessed by quantitative ultrasound and serum markers of bone turnover. Contraception 2005;72:212–6.
55. Office on Women's Health, US Department of Health and Human Services. HHS blueprint for action on breastfeeding. Washington, DC: US Department of Health and Human Services, Office on Women's Health; 2000.
56. Guiloff E, Ibarrapo A, Zanartu J, et al. Effect of contraception on lactation. Am J Obstet Gynecol 1974;118:42–5.
57. World Health Organization Special Programme of Research Development and Research Training in human reproduction. Effects of hormonal contraceptives on milk volume and infant growth. Contraception 1984;30:505–22.
58. Heikkila M, Luukkainen T. Duration of breast-feeding and development of children after insertion of a levonorgestrel-releasing intrauterine contraceptive device. Contraception 1982;25:279–92.
59. Giner VJ, Cortes G, V, Sotelo LA, Bondani G. Effect of daily oral administration of 0.350 mg of norethindrone on lactation and on the composition of milk [in Spanish]. Ginecol Obstet Mex 1976;40:31–9.
60. Zacharias S, Aguilera E, Assenzo JR, Zanartu J. Effects of hormonal and nonhormonal contraceptives on lactation and incidence of pregnancy. Contraception 1986;33:203–13.
61. Kamal I, Hefnawi F, Ghoneim M, Abdallah M, Abdel RS. Clinical, biochemical, and experimental studies on lactation. V. Clinical effects of steroids on the initiation of lactation. Am J Obstet Gynecol 1970;108:655–8.

62. Hannon PR, Duggan AK, Serwint JR, et al. The influence of medroxyprogesterone on the duration of breast-feeding in mothers in an urban community. Arch Pediatr Adolesc Med 1997;151:490–6.
63. Halderman LD, Nelson AL. Impact of early postpartum administration of progestin-only hormonal contraceptives compared with nonhormonal contraceptives on short-term breast-feeding patterns. Am J Obstet Gynecol 2002;186:1250–6.
64. Narducci U, Piatti N. Use of Depo Provera as a contraceptive in the puerperium [in Italian]. Minerva Ginecol 1973;25:107–11.
65. Melis GB, Strigini F, Fruzzetti F, et al. Norethisterone enanthate as an injectable contraceptive in puerperal and non-puerperal women. Contraception 1981;23:77–88.
66. Karim M, Ammar R, El-mahgoub S, et al. Injected progestogen and lactation. Br Med J 1971;1:200–3.
67. Shaaban MM. Contraception with progestogens and progesterone during lactation. J Steroid Biochem Mol Biol 1991;40:705–10.
68. Zanartu J, Aguilera E, Munoz-Pinto G. Maintenance of lactation by means of continuous low-dose progestogen given post-partum as a contraceptive. Contraception 1976;13:313–8.
69. McEwan JA, Joyce DN, Tothill AU, Hawkins DF. Early experience in contraception with a new progestogen. Contraception 1977;16:339–50.
70. McCann MF, Moggia AV, Higgins JE, Potts M, Becker C. The effects of a progestin-only oral contraceptive (levonorgestrel 0.03 mg) on breast-feeding. Contraception 1989;40:635–48.
71. West CP. The acceptability of a progestagen-only contraceptive during breast-feeding. Contraception 1983;27:563–9.
72. Bjarnadottir RI, Gottfredsdottir H, Sigurdardottir K, Geirsson RT, Dieben TO. Comparative study of the effects of a progestogen-only pill containing desogestrel and an intrauterine contraceptive device in lactating women. BJOG 2001;108:1174–80.
73. Taneepanichskul S, Reinprayoon D, Thaithumyanon P, et al. Effects of the etonogestrel-releasing implant Implanon and a nonmedicated intrauterine device on the growth of breast-fed infants. Contraception 2006;73:368–71.
74. Reinprayoon D, Taneepanichskul S, Bunyavejchevin S, et al. Effects of the etonogestrel-releasing contraceptive implant (Implanon(R)) on parameters of breastfeeding compared to those of an intrauterine device. Contraception 2000;62:239–46.
75. Seth U, Yadava HS, Agarwal N, Laumas KR, Hingorani V. Effect of a subdermal silastic implant containing norethindrone acetate on human lactation. Contraception 1977;16:383–98.
76. Shaaban MM, Salem HT, Abdullah KA. Influence of levonorgestrel contraceptive implants, NORPLANT, initiated early postpartum upon lactation and infant growth. Contraception 1985;32:623–35.
77. Abdel-Aleem H, Abol-Oyoun SM, Shaaban MM, et al. The use of nomegestrol acetate subdermal contraceptive implant, uniplant, during lactation. Contraception 1996;54:281–6.
78. Croxatto HB, Diaz S, Peralta O, et al. Fertility regulation in nursing women. II. Comparative performance of progesterone implants versus placebo and copper T. Am J Obstet Gynecol 1982;144:201–8.
79. Diaz S, Peralta O, Juez G, et al. Fertility regulation in nursing women. VI. Contraceptive effectiveness of a subdermal progesterone implant. Contraception 1984;30:311–25.
80. Sivin I, Diaz S, Croxatto HB, et al. Contraceptives for lactating women: a comparative trial of a progesterone-releasing vaginal ring and the copper T 380A IUD. Contraception 1997;55:225–32.
81. Massai R, Miranda P, Valdes P, et al. Preregistration study on the safety and contraceptive efficacy of a progesterone-releasing vaginal ring in Chilean nursing women. Contraception 1999;60:9–14.
82. Shaamash AH, Sayed GH, Hussien MM, Shaaban MM. A comparative study of the levonorgestrel-releasing intrauterine system Mirena(R) versus the Copper T380A intrauterine device during lactation: breast-feeding performance, infant growth and infant development. Contraception 2005;72:346–51.
83. WHO Special Programme of Research Development and Research Training in Human Reproduction. Progestogen-only contraceptives during lactation: II. Infant development. World Health Organization, Task Force for Epidemiological Research on Reproductive Health; Special Programme of Research, Development, and Research Training in Human Reproduction. Contraception 1994;50:55–68.
84. WHO Special Programme of Research Development and Research Training in Human Reproduction. Progestogen-only contraceptives during lactation: I. Infant growth. World Health Organization Task force for Epidemiological Research on Reproductive Health; Special Programme of Research, Development and Research Training in Human Reproduction. Contraception 1994;50:35–53.
85. Diaz S, Zepeda A, Maturana X, et al. Fertility regulation in nursing women: IX. Contraceptive performance, duration of lactation, infant growth, and bleeding patterns during use of progesterone vaginal rings, progestin-only pills, Norplant(R) implants, and Copper T 380-A intrauterine devices. Contraception 1997;56:223–32.
86. Coutinho EM, Athayde C, Dantas C, Hirsch C, Barbosa I. Use of a single implant of elcometrine (ST-1435), a nonorally active progestin, as a long acting contraceptive for postpartum nursing women. Contraception 1999;59:115–22.
87. Massai MR, Diaz S, Quinteros E, et al. Contraceptive efficacy and clinical performance of Nestorone implants in postpartum women. Contraception 2001;64:369–76.
88. Schiappacasse V, Diaz S, Zepeda A, Alvarado R, Herreros C. Health and growth of infants breastfed by Norplant contraceptive implants users: a six-year follow-up study. Contraception 2002;66:57–65.
89. Diaz S, Herreros C, Juez G, et al. Fertility regulation in nursing women: VII. Influence of NORPLANT levonorgestrel implants upon lactation and infant growth. Contraception 1985;32:53–74.
90. Massai R, Quinteros E, Reyes MV, et al. Extended use of a progesterone-releasing vaginal ring in nursing women: a phase II clinical trial. Contraception 2005;72:352–7.
91. Jimenez J, Ochoa M, Soler MP, Portales P. Long-term follow-up of children breast-fed by mothers receiving depot-medroxyprogesterone acetate. Contraception 1984;30:523–33.
92. Abdulla KA, Elwan SI, Salem HS, Shaaban MM. Effect of early postpartum use of the contraceptive implants, NORPLANT, on the serum levels of immunoglobulins of the mothers and their breastfed infants. Contraception 1985;32:261–6.
93. Shikary ZK, Betrabet SS, Toddywala WS, et al. Pharmacodynamic effects of levonorgestrel (LNG) administered either orally or subdermally to early postpartum lactating mothers on the urinary levels of follicle stimulating hormone (FSH), luteinizing hormone (LH) and testosterone (T) in their breast-fed male infants. Contraception 1986;34:403–12.
94. Kurunmaki H. Contraception with levonorgestrel-releasing subdermal capsules, Norplant, after pregnancy termination. Contraception 1983;27:473–82.
95. Kurunmaki H, Toivonen J, Lähteenmäki PL, Luukkainen T. Immediate postabortal contraception with Norplant: levonorgestrel, gonadotropin, estradiol, and progesterone levels over two postaboral months and return of fertility after removal of Norplant capsules. Contraception 1984;30:431-42.
96. Lahteenmaki P, Toivonen J, Lahteenmaki PL. Postabortal contraception with norethisterone enanthate injections. Contraception 1983;27:553–62.
97. Ortayli N, Bulut A, Sahin T, Sivin I. Immediate postabortal contraception with the levonorgestrel intrauterine device, Norplant, and traditional methods. Contraception 2001;63:309–14.
98. Bonny AE, Ziegler J, Harvey R, et al. Weight gain in obese and nonobese adolescent girls initiating depot medroxyprogesterone, oral contraceptive pills, or no hormonal contraceptive method. Arch Pediatr Adolesc Med 2006;160:40–5.

99. Clark MK, Dillon JS, Sowers M, Nichols S. Weight, fat mass, and central distribution of fat increase when women use depot-medroxyprogesterone acetate for contraception. Int J Obes (Lond) 2005;29:1252–8.
100. Jain J, Jakimiuk AJ, Bode FR, Ross D, Kaunitz AM. Contraceptive efficacy and safety of DMPA-SC. Contraception 2004;70:269–75.
101. Kozlowski KJ, Rickert VI, Hendon A, Davis P. Adolescents and Norplant: preliminary findings of side effects. J Adolesc Health 1995;16:373–8.
102. Leiman G. Depo-medroxyprogesterone acetate as a contraceptive agent: its effect on weight and blood pressure. Am J Obstet Gynecol 1972;114:97–102.
103. Mangan SA, Larsen PG, Hudson S. Overweight teens at increased risk for weight gain while using depot medroxyprogesterone acetate. J Pediatr Adolesc Gynecol 2002;15:79–82.
104. Risser WL, Gefter LR, Barratt MS, Risser JM. Weight change in adolescents who used hormonal contraception. J Adolesc Health 1999;24:433–6.
105. Westhoff C, Jain JK, Milsom I, Ray A. Changes in weight with depot medroxyprogesterone acetate subcutaneous injection 104 mg/0.65 mL 1. Contraception 2007;75:261–7.
106. Weiss HG, Nehoda H, Labeck B, et al. Pregnancies after adjustable gastric banding. Obes Surg 2001;11:303–6.
107. Gerrits EG, Ceulemans R, van HR, Hendrickx L, Totte E. Contraceptive treatment after biliopancreatic diversion needs consensus. Obes Surg 2003;13:378–82.
108. Victor A, Odlind V, Kral JG. Oral contraceptive absorption and sex hormone binding globulins in obese women: effects of jejunoileal bypass. Gastroenterol Clin North Am 1987;16:483–91.
109. Andersen AN, Lebech PE, Sorensen TI, Borggaard B. Sex hormone levels and intestinal absorption of estradiol and D-norgestrel in women following bypass surgery for morbid obesity. Int J Obes 1982;6:91–6.
110. World Health Organization. Cardiovascular disease and use of oral and injectable progestogen-only contraceptives and combined injectable contraceptives. Results of an international, multicenter, case-control study. Contraception 1998;57:315–24.
111. Heinemann LA, Assmann A, DoMinh T, et al. Oral progestogen-only contraceptives and cardiovascular risk: results from the Transnational Study on Oral Contraceptives and the Health of Young Women. Eur J Contracept Reprod Health Care 1999;4:67–73.
112. Vasilakis C, Jick H, Mar Melero-Montes M. Risk of idiopathic venous thromboembolism in users of progestogens alone. Lancet 1999;354:1610–1.
113. Sonmezer M, Atabekoglu C, Cengiz B, et al. Depot-medroxyprogesterone acetate in anticoagulated patients with previous hemorrhagic corpus luteum. European J Contracept Reprod Health Care 2005;10:9–14.
114. The Criteria Committee of the New York Heart Association. Nomenclature and criteria for diagnosis of diseases of the heart and great vessels. 9th ed. Boston, MA: Little, Brown & Co; 1994.
115. Avila WS, Grinberg M, Melo NR, Aristodemo PJ, Pileggi F. Contraceptive use in women with heart disease [in Portuguese]. Arq Bras Cardiol 1996;66:205–11.
116. Taurelle R, Ruet C, Jaupart F, Magnier S. Contraception using a progestagen-only minipill in cardiac patients [in French]. Arch Mal Coeur Vaiss 1979;72:98–106.
117. Bernatsky S, Ramsey-Goldman R, Gordon C, et al. Factors associated with abnormal Pap results in systemic lupus erythematosus. Rheumatology (Oxford) 2004;43:1386–9.
118. Bernatsky S, Clarke A, Ramsey-Goldman R, et al. Hormonal exposures and breast cancer in a sample of women with systemic lupus erythematosus. Rheumatology (Oxford) 2004;43:1178–81.
119. Chopra N, Koren S, Greer WL, et al. Factor V Leiden, prothrombin gene mutation, and thrombosis risk in patients with antiphospholipid antibodies. J Rheumatol 2002;29:1683–8.
120. Esdaile JM, Abrahamowicz M, Grodzicky T, et al. Traditional Framingham risk factors fail to fully account for accelerated atherosclerosis in systemic lupus erythematosus. Arthritis Rheum 2001;44:2331–7.
121. Julkunen HA. Oral contraceptives in systemic lupus erythematosus: side-effects and influence on the activity of SLE. Scand J Rheumatol 1991;20:427–33.
122. Julkunen HA, Kaaja R, Friman C. Contraceptive practice in women with systemic lupus erythematosus. Br J Rheumatol 1993;32:227–30.
123. Jungers P, Dougados M, Pelissier C, et al. Influence of oral contraceptive therapy on the activity of systemic lupus erythematosus. Arthritis Rheum 1982;25:618–23.
124. Manzi S, Meilahn EN, Rairie JE, et al. Age-specific incidence rates of myocardial infarction and angina in women with systemic lupus erythematosus: comparison with the Framingham Study. Am J Epidemiol 1997;145:408–15.
125. McAlindon T, Giannotta L, Taub N, et al. Environmental factors predicting nephristis in systemic lupus erythematosus. Ann Rheum Dis 1993;52:720–4.
126. McDonald J, Stewart J, Urowitz MB, et al. Peripheral vascular disease in patients with systemic lupus erythematosus. Ann Rheum Dis 1992;51:56–60.
127. Mintz G, Gutierrez G, Deleze M, et al. Contraception with progestogens in systemic lupus erythematosus. Contraception 1984;30:29–38.
128. Petri M. Musculoskeletal complications of systemic lupus erythematosus in the Hopkins Lupus Cohort: an update. Arthritis Care Res 1995;8:137–45.
129. Petri M, Kim MY, Kalunian KC, et al. Combined oral contraceptives in women with systemic lupus erythematosus. N Engl J Med 2005;353:2550–8.
130. Petri M. Lupus in Baltimore: evidence-based 'clinical perarls' from the Hopkins Lupus Cohort. Lupus 2005;14:970–3.
131. Sanchez-Guerrero J, Uribe AG, Jimenez-Santana L, et al. A trial of contraceptive methods in women with systemic lupus erythematosus. N Engl J Med 2005;353:2539–49.
132. Sarabi ZS, Chang E, Bobba R, et al. Incidence rates of arterial and venous thrombosis after diagnosis of systemic lupus erythematosus. Arthritis Rheum 2005;53:609–12.
133. Schaedel ZE, Dolan G, Powell MC. The use of the levonorgestrel-releasing intrauterine system in the management of menorrhagia in women with hemostatic disorders. Am J Obstet Gynecol 2005;193:1361–3.
134. Somers E, Magder LS, Petri M. Antiphospholipid antibodies and incidence of venous thrombosis in a cohort of patients with systemic lupus erythematosus. J Rheumatol 2002;29:2531–6.
135. Urowitz MB, Bookman AA, Koehler BE, et al. The bimodal mortality pattern of systemic lupus erythematosus. Am J Med 1976;60:221–5.
136. Choojitarom K, Verasertniyom O, Totemchokchyakarn K, et al. Lupus nephritis and Raynaud's phenomenon are significant risk factors for vascular thrombosis in SLE patients with positive antiphospholipid antibodies. Clini Rheumatol 2008;27:345–51.
137. Wahl DG, Guillemin F, de Maistre E, et al. Risk for venous thrombosis related to antiphospholipid antibodies in systemic lupus erythematosus—a meta-analysis. Lupus 1997;6:467–73.

138. Demers R, Blais JA, Pretty H. Rheumatoid arthritis treated by norethynodrel associated with mestranol: clinical aspects and laboratory tests [in French]. Can Med Assoc J 1966;95:350–4.
139. Drossaers-Bakker KW, Zwinderman AH, Van ZD, Breedveld FC, Hazes JM. Pregnancy and oral contraceptive use do not significantly influence outcome in long term rheumatoid arthritis. Ann Rheum Dis 2002;61:405–8.
140. Gilbert M, Rotstein J, Cunningham C, et al. Norethynodrel with mestranol in treatment of rheumatoid arthritis. JAMA 1964;190:235.
141. Gill D. Rheumatic complaints of women using anti-ovulatory drugs. An evaluation. J Chronic Dis 1968;21:435–44.
142. Hazes JM, Dijkmans BA, Vandenbroucke JP, Cats A. Oral contraceptive treatment for rheumatoid arthritis: an open study in 10 female patients. Br J Rheumatol 1989;28 Suppl 1:28–30.
143. Ostensen M, Aune B, Husby G. Effect of pregnancy and hormonal changes on the activity of rheumatoid arthritis. Scand J Rheumatol 1983;12:69–72.
144. Vignos PJ, Dorfman RI. Effect of large doses of progesterone in rheumatoid arthritis. Am J Med Sci 1951;222:29–34.
145. Bijlsma JW, Huber-Bruning O, Thijssen JH. Effect of oestrogen treatment on clinical and laboratory manifestations of rheumatoid arthritis. Ann Rheum Dis 1987;46:777–9.
146. Cromer BA, Smith RD, Blair JM, Dwyer J, Brown RT. A prospective study of adolescents who choose among levonorgestrel implant (Norplant), medroxyprogesterone acetate (Depo-Provera), or the combined oral contraceptive pill as contraception. Pediatrics 1994;94 :687–94.
147. Gupta N, O'Brien R, Jacobsen LJ, et al. Mood changes in adolescents using depo-medroxyprogesterone acetate for contraception: a prospective study. Am J Obstet Gynecol 2001;14:71–6.
148. Westoff C, Truman C. Depressive symptoms and Depo-Provera. Contraception 1998;57:237–40.
149. Westoff C, Truman C, Kalmuss D, et al. Depressive symptoms and Norplant contraceptive implants. Contraception 1998;57:241–5.
150. Smith JS. Cervical cancer and use of hormonal conraceptives: a systematic review. Lancet 2003;361:1159–67.
151. Baeten JM, Nyange PM, Richardson BA, et al. Hormonal contraception and risk of sexually transmitted disease acquisition: results from a prospective study. Am J Obstet Gynecol 2001;185:380–5.
152. Giuliano AR, Papenfuss M, Abrahamsen M, et al. Human papillomavirus infection at the United States–Mexico border: implications for cervical cancer prevention and control. Cancer Epidemiol Biomarkers Prev 2001;10:1129–36.
153. Jacobson DL, Peralta L, Farmer M, et al. Relationship of hormonal contraception and cervical ectopy as measured by computerized planimetry to chlamydial infection in adolescents. Sex Transm Dis 2000;27:313–9.
154. Lavreys L, Chohan B, Ashley R, et al. Human herpesvirus 8: seroprevalence and correlates in prostitutes in Mombasa, Kenya. J Infect Dis 2003;187:359–63.
155. Morrison CS, Bright P, Wong EL, et al. Hormonal contraceptive use, cervical ectopy, and the acquisition of cervical infections. Sex Transm Dis 2004;31:561–7.
156. Moscicki AB, Hills N, Shiboski S, et al. Risks for incident human papillomavirus infection and low-grade squamous intraepithelial lesion development in young females. JAMA 2001;285:2995–3002.
157. Nsofor BI, Bello CS, Ekwempu CC. Sexually transmitted disease among women attending a family planning clinic in Zaria, Nigeria. Int J Gynaecol Obstet 1989;28:365–7.
158. Ruijs GJ, Kauer FM, van Gijssel PM, Schirm J, Schroder FP. Direct immunofluorescence for *Chlamydia trachomatis* on urogenital smears for epidemiological purposes. Eur J Obstet Gyneco Reprod Biol 1988;27:289–97.
159. Aklilu M, Messele T, Tsegaye A, et al. Factors associated with HIV-1 infection among sex workers of Addis Ababa, Ethiopia. AIDS 2001;15:87–96.
160. Allen S, Serufilira A, Gruber V, et al. Pregnancy and contraception use among urban Rwandan women after HIV testing and counseling. Am J Public Health 1993;83:705–10.
161. Baeten JM, Benki S, Chohan V, et al. Hormonal contraceptive use, herpes simplex virus infection, and risk of HIV-1 acquisition among Kenyan women. AIDS 2007;21:1771–7.
162. Bulterys M, Chao A, Habimana P, et al. Incident HIV-1 infection in a cohort of young women in Butare, Rwanda. AIDS 1994;8:1585–91.
163. Carael M, Van de Perre PH, Lepage PH, et al. Human immunodeficiency virus transmission among heterosexual couples in Central Africa. AIDS 1988;2:201–5.
164. Cohen CR, Duerr A, Pruithithada N, et al. Bacterial vaginosis and HIV seroprevalence among female commercial sex workers in Chiang Mai, Thailand. AIDS 1995;9:1093–7.
165. Criniti A, Mwachari CW, Meier AS, et al. Association of hormonal contraception and HIV-seroprevalence in Nairobi, Kenya. AIDS 2003;17:2667–9.
166. Kapiga SH, Shao JF, Lwihula GK, Hunter DJ. Risk factors for HIV infection among women in Dar-es-Salaam, Tanzania. J Acquir Immune Defic Syndr 1994;7:301–9.
167. Kapiga SH, Lyamuya EF, Lwihula GK, Hunter DJ. The incidence of HIV infection among women using family planning methods in Dar es Salaam, Tanzania. AIDS 1998;12:75–84.
168. Kiddugavu M, Makumbi F, Wawer MJ, et al. Hormonal contraceptive use and HIV-1 infection in a population-based cohort in Rakai, Uganda. AIDS 2003;17:233–40.
169. Kilmarx PH, Limpakarnjanarat K, Mastro TD, et al. HIV-1 seroconversion in a prospective study of female sex workers in northern Thailand: continued high incidence among brothel-based women. AIDS 1998;12:1889–98.
170. Kleinschmidt I, Rees H, Delany S, et al. Injectable progestin contraceptive use and risk of HIV infection in a South African family planning cohort. Contraception 2007;75:461–7.
171. Lavreys L, Chohan V, Overbaugh J, et al. Hormonal contraception and risk of cervical infections among HIV-1-seropositive Kenyan women. AIDS 2004;18:2179–84.
172. Limpakarnjanarat K, Mastro TD, Saisorn S, et al. HIV-1 and other sexually transmitted infections in a cohort of female sex workers in Chiang Rai, Thailand. Sex Transm Infect 1999;75:30–5.
173. Martin HL, Jr., Nyange PM, Richardson BA, et al. Hormonal contraception, sexually transmitted diseases, and risk of heterosexual transmission of human immunodeficiency virus type 1. J Infect Dis 1998;178:1053–9.
174. Mati JK, Hunter DJ, Maggwa BN, Tukei PM. Contraceptive use and the risk of HIV infection in Nairobi, Kenya. Int J Gynaecol Obstet 1995;48:61–7.
175. Morrison CS, Richardson BA, Mmiro F, et al. Hormonal contraception and the risk of HIV acquisition. AIDS 2007;21:85–95.
176. Myer L, Denny L, Wright TC, Kuhn L. Prospective study of hormonal contraception and women's risk of HIV infection in South Africa. Int J Epidemiol 2007;36:166–74.

177. Nagachinta T, Duerr A, Suriyanon V, et al. Risk factors for HIV-1 transmission from HIV-seropositive male blood donors to their regular female partners in northern Thailand. AIDS 1997;11:1765–72.
178. Nzila N, Laga M, Thiam MA, et al. HIV and other sexually transmitted diseases among female prostitutes in Kinshasa. AIDS 1991;5:715–21.
179. Plourde PJ, Plummer FA, Pepin J, et al. Human immunodeficiency virus type 1 infection in women attending a sexually transmitted diseases clinic in Kenya [comment]. J Infect Dis 1992;166:86–92.
180. Rehle T, Brinkmann UK, Siraprapasiri T, et al. Risk factors of HIV-1 infection among female prostitutes in Khon Kaen, northeast Thailand. Infection 1992;20:328–31.
181. Siraprapasiri T, Thanprasertsuk S, Rodklay A, et al. Risk factors for HIV among prostitutes in Chiangmai, Thailand. AIDS 1991;5:579–82.
182. Taneepanichskul S, Phuapradit W, Chaturachinda K. Association of contraceptives and HIV-1 infection in Thai female commercial sex workers. Aust N Z J Obstet Gynaecol 1997;37:86–8.
183. Ungchusak K, Rehle T, Thammapornpilap P, et al. Determinants of HIV infection among female commercial sex workers in northeastern Thailand: results from a longitudinal study. J Acquir Immune Defic Syndr Hum Retrovirol 1996;12:500–7. Erratum in: J Acquir Immune Defic Syndr Hum Retrovirol 1998;18:192.
184. Allen S, Stephenson R, Weiss H, et al. Pregnancy, hormonal contraceptive use, and HIV-related death in Rwanda. J Womens Health (Larchmt) 2007;16:1017–27.
185. Cejtin HE, Jacobson L, Springer G, et al. Effect of hormonal contraceptive use on plasma HIV-1-RNA levels among HIV-infected women. AIDS 2003;17:1702–4.
186. Clark RA, Kissinger P, Williams T. Contraceptive and sexually transmitted diseases protection among adult and adolescent women infected with human immunodeficiency virus. Int J STD AIDS 1996;7:439–42.
187. Clark RA, Theall KP, Amedee AM, et al. Lack of association between genital tract HIV-1 RNA shedding and hormonal contraceptive use in a cohort of Louisiana women. Sex Transm Dis 2007;34:870–2.
188. Clemetson DB, Moss GB, Willerford DM, et al. Detection of HIV DNA in cervical and vaginal secretions. Prevalence and correlates among women in Nairobi, Kenya. JAMA 1993;269:2860–4.
189. European Study Group on Heterosexual Transmission of HIV. Comparison of female to male and male to female transmission of HIV in 563 stable couples. BMJ 1992;304:809–13.
190. Kaul R, Kimani J, Nagelkerke NJ, et al. Risk factors for genital ulcerations in Kenyan sex workers. The role of human immunodeficiency virus type 1 infection. Sex Transm Dis 1997;24:387–92.
191. Kilmarx PH, Limpakarnjanarat K, Kaewkungwal J, et al. Disease progression and survival with human immunodeficiency virus type 1 subtype E infection among female sex workers in Thailand. J Infect Dis 2000;181:1598–606.
192. Kovacs A, Wasserman SS, Burns D, et al. Determinants of HIV-1 shedding in the genital tract of women. Lancet 2001;358:1593–601.
193. Kreiss J, Willerford DM, Hensel M, et al. Association between cervical inflammation and cervical shedding of human immunodeficiency virus DNA. J Infect Dis 1994;170:1597-601.
194. Mostad SB, Overbaugh J, DeVange DM, et al. Hormonal contraception, vitamin A deficiency, and other risk factors for shedding of HIV-1 infected cells from the cervix and vagina. Lancet 1997;350:922–7.
195. Richardson BA, Otieno PA, Mbori-Ngacha D, et al. Hormonal contraception and HIV-1 disease progression among postpartum Kenyan women. AIDS 2007;21:749–53.
196. Seck K, Samb N, Tempesta S, et al. Prevalence and risk factors of cervicovaginal HIV shedding among HIV-1 and HIV-2 infected women in Dakar, Senegal. Sex Transm Infect 2001;77:190–3.
197. Stringer EM, Kaseba C, Levy J, et al. A randomized trial of the intrauterine contraceptive device vs hormonal contraception in women who are infected with the human immunodeficiency virus. Am J Obstet Gynecol 2007;197:144–8.
198. Taneepanichskul S, Intaraprasert S, Phuapradit W, Chaturachinda K. Use of Norplant implants in asymptomatic HIV-1 infected women. Contraception 1997;55:205–7.
199. Taneepanichskul S, Tanprasertkul C. Use of Norplant implants in the immediate postpartum period among asymptomatic HIV-1-positive mothers. Contraception 2001;64:39–41.
200. Wang CC, McClelland RS, Overbaugh J, et al. The effect of hormonal contraception on genital tract shedding of HIV-1. AIDS 2004;18:205–9.
201. Tagy AH, Saker ME, Moussa AA, Kolgah A. The effect of low-dose combined oral contraceptive pills versus injectable contracetpive (Depot Provera) on liver function tests of women with compensated bilharzial liver fibrosis. Contraception 2001;64:173–6.
202. Pyorala T, Vahapassi J, Huhtala M. The effect of lynestrenol and norethindrone on the carbohydrate and lipid metabolism in subjects with gestational diabetes. Ann Chir Gynaecol 1979;68:69–74.
203. Radberg T, Gustafson A, Skryten A, Karlsson K. Metabolic studies in women with previous gestational diabetes during contraceptive treatment: effects on serum lipids and high density lipoproteins. Acta Endocrinol (Copenh) 1982;101:134–9.
204. Xiang AH, Kawakubo M, Kjos SL, Buchanan TA. Long-acting injectable progestin contraception and risk of type 2 diabetes in Latino women with prior gestational diabetes mellitus. Diabetes Care 2006;29:613–7.
205. Xiang AH, Kawakubo M, Buchanan TA, Kjos SL. A longitudinal study of lipids and blood pressure in relation to method of contraception in Latino women with prior gestational diabetes mellitus. Diabetes Care 2007;30:1952–8.
206. Kjos SL, Peters RK, Xiang A, et al. Contraception and the risk of type 2 diabetes mellitus in Latina women with prior gestational diabetes mellitus. JAMA 1998;280:533–8.
207. Nelson AL, Le MH, Musherraf Z, Vanberckelaer A. Intermediate-term glucose tolerance in women with a history of gestational diabetes: natural history and potential associations with breastfeeding and contraception. Am J Obstet Gynecol 2008;198:699–7.
208. Diab KM, Zaki MM. Contraception in diabetic women: comparative metabolic study of norplant, depot medroxyprogesterone acetate, low dose oral contraceptive pill and CuT380A. J Obstet Gynecol Res 2000;26:17–26.
209. Lunt H, Brown LJ. Self-reported changes in capillary glucose and insulin requirements during the menstrual cycle. Diabetic Med 1995;13:525–30.
210. Radberg T, Gustafson A, Skryten A, Karlsson K. Oral contraception in diabetic women. A cross-over study on seum and high density lipoprotein (HDL) lipids and diabetes control during progestogen and combined estrogen/progestogen contraception. Horm Metab Res 1982;14:61–5.
211. Skouby SO, Molsted-Petersen L, Kuhl C, Bennet P. Oral contraceptives in diabetic womne: metabolic effects of four compounds with different estrogen/progestogen profiles. Fertil Steril 1986;46:858–64.

212. Bitton A, Peppercorn MA, Antonioli DA, et al. Clinical, biological, and histologic parameters as predictors of relapse in ulcerative colitis. Gastroenterology 2001;120:13–20.
213. Cosnes J, Carbonnel F, Carrat F, Beaugerie L, Gendre JP. Oral contraceptive use and the clinical course of Crohn's disease: a prospective cohort study. Gut 1999;45:218–22.
214. Sutherland LR, Ramcharan S, Bryant H, Fick G. Effect of oral contraceptive use on reoperation following surgery for Crohn's disease. Dig Dis Sci 1992;37:1377–82.
215. Timmer A, Sutherland LR, Martin F. Oral contraceptive use and smoking are risk factors for relapse in Crohn's disease. The Canadian Mesalamine for Remission of Crohn's Disease Study Group. Gastroenterology 1998;114:1143–50.
216. Wright JP. Factors influencing first relapse in patients with Crohn's disease. J Clin Gastroenterol 1992;15:12–6.
217. D'halluin V, Vilgrain V, Pelletier G, et al. Natural history of focal nodular hyperplasia. A retrospective study of 44 cases [in French]. Gastroenterol Clin Biol 2001;25:1008–10.
218. Mathieu D, Kobeiter H, Maison P, et al. Oral contraceptive use and focal nodular hyperplasia of the liver. Gastroenterology 2000;118:560–4.
219. Adadevoh BK, Isaacs WA. The effect of megestrol acetate on sickling. Am J Med Sci 1973;265:367–70.
220. Barbosa IC, Ladipo OA, Nascimento ML, et al. Carbohydrate metabolism in sickle cell patients using subdermal implant containing nomegesterol acetate (Uniplant). Contraception 2001;63:263–5.
221. de Abood M, de Castillo Z, Guerrero F, Espino M, Austin KL. Effects of Depo-Provera or Microgynon on the painful crises of sickle cell anemia patients. Contraception 1997;56:313–6.
222. De Ceulaer K, Gruber C, Hayes R, Serjeant GR. Medroxyprogesterone acetate and homozygous sickle-cell disease. Lancet 1982;2:229–31.
223. Howard RJ, Lillis C, Tuck SM. Contraceptives, counseling, and pregnancy in women with sickle cell disease. BMJ 1993;306:1735–7.
224. Ladipo OA, Falusi AG, Feldblum PJ, Osotimehin BO, Otolorin EO, Ojengbede OA Norplant use by women with sickle cell disease. Int J Gynaecol Obstet 1993;41:85–7.
225. Nascimento ML, Ladipo OA, Coutinho E. Nomogestrol acetate contraceptive implant use by women with sickle cell disease. Clin Pharmacol Ther 1998;64:433–8.
226. Yoong WC, Tuck SM, Yardumian A. Red cell deformability in oral contraceptive pill users with sickle cell anaemia. Br J Haematol 1999;104:868–70.
227. Odlind V, Olsson S-E. Enhanced metabolism of levonorgestrel during phenytoin treatment in a woman with Norplant implants. Contraception 1986;33:257–61.
228. Schindlbeck C, Janni W, Friese K. Failure of Implanon contraception in a patient taking carbamazepine for epilepsia. Arch Gynecol Obstet 2006;273:255–6.
229. Shane-McWhorter L, Cerven JD, MacFarlane LL, Osborn C. Enhanced metabolism of levonorgestrel during phenobarbital treatment and resultant pregnancy. Pharmacotherapy 1998;18:1360–4.
230. Reimers A, Helde G, Brodtkorb E. Ethinyl estradiol, not progestogens, reduces lamotrigine serum concentrations. Epilepsia 2005;46:1414–7.

Appendix D
Classifications for Emergency Contraceptive Pills

Classifications for emergency contraceptive pills (ECPs) are for both levonorgestrel and combined oral contraceptive pills. ECPs do not protect against sexually transmitted infections (STIs) or human immunodeficiency virus (HIV).

BOX. Categories for Classifying Emergency Contraceptive Pills

1 = A condition for which there is no restriction for the use of the contraceptive method.
2 = A condition for which the advantages of using the method generally outweigh the theoretical or proven risks.
3 = A condition for which the theoretical or proven risks usually outweigh the advantages of using the method.
4 = A condition that represents an unacceptable health risk if the contraceptive method is used.

TABLE. Classifications for emergency contraceptive pills, including levonorgestrel contraceptive pills and combined oral contraceptive pills*†

Condition	Category	Clarifications/Evidence/Comments
Personal Characteristics and Reproductive History		
Pregnancy	Not applicable	**Clarification:** Although this method is not indicated for a woman with a known or suspected pregnancy, no harm to the woman, the course of her pregnancy, or the fetus if ECPs are inadvertently used is known to exist.
Breastfeeding	1	
Past ectopic pregnancy	1	
History of bariatric surgery§		
a. Restrictive procedures: decrease storage capacity of the stomach (vertical banded gastroplasty, laparoscopic adjustable gastric band, laparoscopic sleeve gastrectomy)	1	
b. Malabsorptive procedures: decrease absorption of nutrients and calories by shortening the functional length of the small intestine (Roux-en-Y gastric bypass, biliopancreatic diversion)	1	**Comment:** Bariatric surgical procedures involving a malabsorptive component have the potential to decrease oral contraceptive effectiveness, perhaps further decreased by postoperative complications such as long-term diarrhea and/or vomiting. Because of these malabsorptive concerns, an emergency IUD might be more appropriate than ECPs.
Cardiovascular Disease		
History of severe cardiovascular complications§ (ischemic heart disease, cerebrovascular attack, or other thromboembolic conditions)	2	**Comment:** The duration of ECP use is less than that of regular use of COCs or POPs and thus would be expected to have less clinical impact.
Angina pectoris	2	**Comment:** The duration of ECP use is less than that of regular use of COCs or POPs and thus would be expected to have less clinical impact.
Rheumatic Diseases		
Rheumatoid arthritis		
a. On immunosuppressive therapy	1	
b. Not on immunosuppressive therapy	1	
Neurologic Conditions		
Migraine	2	**Comment:** The duration of ECP use is less than that of regular use of COCs or POPs and thus would be expected to have less clinical impact.
Gastrointestinal Conditions		
Inflammatory bowel disease (ulcerative colitis, Crohn disease)	1	
Severe liver disease§ (including jaundice)	2	**Comment:** The duration of ECP use is less than that of regular use of COCs or POPs and thus would be expected to have less clinical impact.
Solid Organ Transplantation		
Solid organ transplantation§		
a. Complicated: graft failure (acute or chronic), rejection, cardiac allograft vasculopathy	1	
b. Uncomplicated	1	

TABLE. (*Continued*) Classifications for emergency contraceptive pills, including levonorgestrel contraceptive pills and combined oral contraceptive pills*†

Condition	Category	Clarifications/Evidence/Comments
Other		
Repeated ECP use	1	**Clarification:** Recurrent ECP use is an indication that the woman requires further counseling about other contraceptive options. Frequently repeated ECP use may be harmful for women with conditions classified as 2, 3, or 4 for CHC or POC use.
Rape	1	**Comment:** Use of ECPs in cases of rape has no restrictions.

* Abbreviations: STI = sexually transmitted infection; HIV = human immunodeficiency virus; ECP, emergency contraceptive pill; IUD = intrauterine device; COC = combined oral contraceptive; POP = progestin-only pill; CHC = combined hormonal contraceptive; POC = progestin-only contraceptive

† ECPs do not protect against STI/HIV. If risk exists for STI/HIV (including during pregnancy or postpartum), the correct and consistent use of condoms is recommended, either alone or with another contraceptive method. Consistent and correct use of the male latex condom reduces the risk for STIs and HIV transmission.

§ Condition that exposes a woman to increased risk as a result of unintended pregnancy.

Appendix E

Classifications for Intrauterine Devices

Classifications for intrauterine devices (IUDs) are for the levonorgestrel-releasing (20 μg/24 hours) IUD and the copper-bearing IUD (Box). IUDs do not protect against sexually transmitted infections (STIs) or human immunodeficiency virus (HIV).

BOX. Categories for Classifying Intrauterine Devices

1 = A condition for which there is no restriction for the use of the contraceptive method.
2 = A condition for which the advantages of using the method generally outweigh the theoretical or proven risks.
3 = A condition for which the theoretical or proven risks usually outweigh the advantages of using the method.
4 = A condition that represents an unacceptable health risk if the contraceptive method is used.

TABLE. Classifications for intrauterine devices, including the LNG-IUD and the Cu-IUD*†

Condition	Category		Clarifications/Evidence/Comments
	LNG-IUD	Cu-IUD	
Personal Characteristics and Reproductive History			
Pregnancy	4	4	**Clarification:** The IUD is not indicated during pregnancy and should not be used because of the risk for serious pelvic infection and septic spontaneous abortion.
Age			
a. Menarche to <20 yrs	2	2	**Comment:** Concern exists about both the risk for expulsion from nulliparity and for STIs from sexual behaviour in younger age groups.
b. ≥20 yrs	1	1	
Parity			
a. Nulliparous	2	2	**Evidence:** Data conflict about whether IUD use is associated with infertility among nulliparous women, although well-conducted studies suggest no increased risk (*1–9*).
b Parous	1	1	
Postpartum (breastfeeding or nonbreastfeeding women, including post-Cesarean section)			
a. <10 minutes after delivery of the placenta	2	1	**Evidence:** Immediate postpartum Cu-IUD insertion, particularly when insertion occurs immediately after delivery of the placenta, is associated with lower expulsion rates than is delayed postpartum insertion up to 72 hours postpartum; no data exist that examine times >72 hours postpartum. In addition, postplacental placement at the time of Cesarean section has lower expulsion rates than does postplacental vaginal insertions. Insertion complications of perforation and infection are not increased by Cu-IUD placement at any time during the postpartum period (*10–23*). No evidence is available that compares different insertion times for the LNG-IUD.
b. 10 minutes after delivery of the placenta to <4 wks	2	2	
c. ≥4 wks	1	1	
d. Puerperal sepsis	4	4	**Comment:** Insertion of an IUD might substantially worsen the condition.
Postabortion			
a. First trimester	1	1	**Clarification:** IUDs can be inserted immediately after first trimester spontaneous or induced abortion.
b. Second trimester	2	2	**Evidence:** Risk for complications from immediate versus delayed insertion of an IUD after abortion did not differ. Expulsion was greater when an IUD was inserted after a second trimester abortion than when inserted after a first trimester abortion. Safety or expulsion for postabortion insertion of an LNG-IUD did not differ from that of a Cu-IUD (*24–37*).
c. Immediate postseptic abortion	4	4	**Comment:** Insertion of an IUD might substantially worsen the condition.

TABLE. (*Continued*) Classifications for intrauterine devices,*† including the LNG-IUD and the Cu-IUD

Condition	Category		Clarifications/Evidence/Comments
	LNG-IUD	Cu-IUD	
Past ectopic pregnancy	1	1	**Comment:** The absolute risk for ectopic pregnancy is extremely low because of the high effectiveness of IUDs. However, when a woman becomes pregnant during IUD use, the relative likelihood of ectopic pregnancy increases greatly.
History of pelvic surgery (see Postpartum, including post-Cesarean section)	1	1	
Smoking			
a. Age <35 yrs	1	1	
b. Age ≥35 yrs			
i. <15 Cigarettes/day	1	1	
ii. ≥15 Cigarettes/day	1	1	
Obesity			
a. ≥30 kg/m^2 BMI	1	1	
b. Menarche to <18 yrs and ≥30 kg/m^2 BMI	1	1	
History of bariatric surgery§			
a. Restrictive procedures: decrease storage capacity of the stomach (vertical banded gastroplasty, laparoscopic adjustable gastric band, laparoscopic sleeve gastrectomy)	1	1	
b. Malabsorptive procedures: decrease absorption of nutrients and calories by shortening the functional length of the small intestine (Roux-en-Y gastric bypass, biliopancreatic diversion)	1	1	
Cardiovascular Disease			
Multiple risk factors for arterial cardiovascular disease (such as older age, smoking, diabetes, and hypertension)	2	1	
Hypertension			
For all categories of hypertension, classifications are based on the assumption that no other risk factors for cardiovascular disease exist. When multiple risk factors do exist, risk for cardiovascular disease might increase substantially. A single reading of blood pressure level is not sufficient to classify a woman as hypertensive.			
a. Adequately controlled hypertension	1	1	
b. Elevated blood pressure levels (properly taken measurements)			
i. Systolic 140–159 mm Hg or diastolic 90–99 mm Hg	1	1	
ii. Systolic ≥160 mm Hg or diastolic ≥100 mm Hg§	2	1	**Comment:** Theoretical concern exists about the effect of LNG on lipids. Use of Cu-IUDs has no restrictions.
c. Vascular disease	2	1	**Comment:** Theoretical concern exists about the effect of LNG on lipids. Use of Cu-IUDs has no restrictions.
History of high blood pressure during pregnancy (where current blood pressure is measurable and normal)	1	1	
Deep venous thrombosis (DVT)/ pulmonary embolism (PE)			
a. History of DVT/PE, not on anticoagulant therapy			
i. Higher risk for recurrent DVT/PE (≥1 risk factors)	2	1	
• History of estrogen-associated DVT/PE			
• Pregnancy-associated DVT/PE			
• Idiopathic DVT/PE			
• Known thrombophilia, including antiphospholipid syndrome			
• Active cancer (metastatic, on therapy, or within 6 mos after clinical remission), excluding non-melanoma skin cancer			
• History of recurrent DVT/PE			
ii. Lower risk for recurrent DVT/PE (no risk factors)	2	1	

TABLE. (*Continued*) Classifications for intrauterine devices,*† including the LNG-IUD and the Cu-IUD

Condition	Category		Clarifications/Evidence/Comments
	LNG-IUD	Cu-IUD	
b. Acute DVT/PE	2	2	**Evidence:** No direct evidence exists on the use of POCs among women with acute DVT/PE. Although findings on the risk for venous thrombosis with the use of POCs in otherwise healthy women are inconsistent, any small increased risk is substantially less than that with COCs (*38–40*).
c. DVT/PE and established on anticoagulant therapy for at least 3 mos			**Evidence:** No direct evidence exists on the use of POCs among women with acute DVT/PE. Although findings on the risk for venous thrombosis with the use of POCs in otherwise healthy women are inconsistent, any small increased risk is substantially less than that with COCs (*38–40*). **Evidence:** Limited evidence indicates that insertion of the LNG-IUD does not pose major bleeding risks in women on chronic anticoagulant therapy. (*41–44*) **Comment:** The LNG-IUD might be a useful treatment for menorrhagia in women on long-term chronic anticoagulation therapy.
i. Higher risk for recurrent DVT/PE (≥1 risk factors) • Known thrombophilia, including antiphospholipid syndrome • Active cancer (metastatic, on therapy, or within 6 mos after clinical remission), excluding nonmelanoma skin cancer • History of recurrent DVT/PE	2	2	
ii. Lower risk for recurrent DVT/PE (no risk factors)	2	2	
d. Family history (first-degree relatives)	1	1	
e. Major surgery			
i. With prolonged immobilization	2	1	
ii. Without prolonged immobilization	1	1	
f. Minor surgery without immobilization	1	1	
Known thrombogenic mutations§ (e.g., factor V Leiden; prothrombin mutation; protein S, protein C, and antithrombin deficiencies)	2	1	**Clarification:** Routine screening is not appropriate because of the rarity of the conditions and the high cost of screening.
Superficial venous thrombosis			
a. Varicose veins	1	1	
b. Superficial thrombophlebitis	1	1	
Current and history of ischemic heart disease§	Initiation: 2 / Continuation: 3	1	**Comment:** Theoretical concern exists about the effect of LNG on lipids. Use of Cu-IUDs has no restrictions.
Stroke§ (history of cerebrovascular accident)	2	1	**Comment:** Theoretical concern exists about the effect of LNG on lipids. Use of Cu-IUDs has no restrictions.
Known hyperlipidemias	2	1	**Clarification:** Routine screening is not appropriate because of the rarity of the condition and the high cost of screening.
Valvular heart disease			
a. Uncomplicated	1	1	**Comment**: According to the American Heart Association, administration of prophylactic antibiotics solely to prevent endocarditis is not recommended for patients who undergo genitourinary tract procedures, including insertion or removal of IUDs (*45*).
b. Complicated§ (pulmonary hypertension, risk for atrial fibrillation, history of subacute bacterial endocarditis)	1	1	**Comment**: According to the American Heart Association, administration of prophylactic antibiotics solely to prevent endocarditis is not recommended for patients who undergo genitourinary tract procedures, including insertion or removal of IUDs *(45)*.
Peripartum cardiomyopathy§			
a. Normal or mildly impaired cardiac function (New York Heart Association Functional Class I or II: patients with no limitation of activities or patients with slight, mild limitation of activity) (*46*)			**Evidence:** No direct evidence exists on the safety of IUDs among women with peripartum cardiomyopathy. Limited indirect evidence from noncomparative studies did not demonstrate any cases of arrhythmia or infective endocarditis in women with cardiac disease who used IUDs (*47,48*).
i. <6 mos	2	2	**Comment:** IUD insertion might induce cardiac arrhythmias in healthy women; women with peripartum cardiomyopathy have a high incidence of cardiac arrhythmias.
ii. ≥6 mos	2	2	

TABLE. (*Continued*) Classifications for intrauterine devices,*† including the LNG-IUD and the Cu-IUD

Condition	Category: LNG-IUD	Category: Cu-IUD	Clarifications/Evidence/Comments
b. Moderately or severely impaired cardiac function (New York Heart Association Functional Class III or IV: patients with marked limitation of activity or patients who should be at complete rest) (*46*)	2	2	**Evidence:** There is no direct evidence on the safety of IUDs among women with peripartum cardiomyopathy. Limited indirect evidence from noncomparative studies did not demonstrate any cases of arrhythmia or infective endocarditis in women with cardiac disease who used IUDs (*47,48*). **Comment:** IUD insertion might induce cardiac arrhythmias in healthy women; women with peripartum cardiomyopathy have a high incidence of cardiac arrhythmias.
Rheumatic Diseases			
Systemic lupus erythematosus (SLE)§ Persons with SLE are at increased risk for ischemic heart disease, stroke, and VTE. Categories assigned to such conditions in the MEC should be the same for women with SLE who have these conditions. For all categories of SLE, classifications are based on the assumption that no other risk factors for cardiovascular disease are present; these classifications must be modified in the presence of such risk factors. Many women with SLE can be considered good candidates for most contraceptive methods, including hormonal contraceptives (*43,49–66*).			
		Initiation / Continuation	
a. Positive (or unknown) antiphospholipid antibodies	3	1 / 1	**Evidence:** Antiphospholipid antibodies are associated with a higher risk for both arterial and venous thrombosis (*67,68*).
b. Severe thrombocytopenia	2	3 / 2	**Clarification:** Severe thrombocytopenia increases the risk for bleeding. The category should be assessed according to the severity of thrombocytopenia and its clinical manifestations. In women with very severe thrombocytopenia who are at risk for spontaneous bleeding, consultation with a specialist and certain pretreatments might be warranted. **Evidence:** The LNG-IUD might be a useful treatment for menorrhagia in women with severe thrombocytopenia (*43*).
c. Immunosuppressive treatment	2	2 / 1	
d. None of the above	2	1 / 1	
Rheumatoid arthritis	Initiation / Continuation	Initiation / Continuation	
a. On immunosuppressive therapy	2 / 1	2 / 1	
b. Not on immunosuppressive therapy	1	1	
Neurologic Conditions			
Headaches	Initiation / Continuation		**Clarification:** Any new headaches or marked changes in headaches should be evaluated.
a. Non-migrainous (mild or severe)	1 / 1	1	
b. Migraine			
i. Without aura			**Comment:** Aura is a specific focal neurologic symptom. For more information about this and other diagnostic criteria, see: Headache Classification Subcommittee of the International Headache Society. The international classification of headache disorders. 2nd ed. Cephalalgia 2004;24(Suppl 1):1– 150. Available from http://www.i-h-s.org/upload/ct_clas/ihc_II_main_no_print.pdf.
• Age <35 yrs	2 / 2	1	
• Age ≥35 yrs	2 / 2	1	
ii. With aura, at any age	2 / 3	1	
Epilepsy§	1	1	
Depressive Disorders			
Depressive disorders	1	1	**Clarification**: The classification is based on data for women with selected depressive disorders. No data were available on bipolar disorder or postpartum depression. Drug interactions potentially can occur between certain antidepressant medications and hormonal contraceptives.
Reproductive Tract Infections and Disorders			
Vaginal bleeding patterns	Initiation / Continuation		
a. Irregular pattern without heavy bleeding	1 / 1	1	
b. Heavy or prolonged bleeding (includes regular and irregular patterns)	1 / 2	2	**Clarification:** Unusually heavy bleeding should raise suspicion of a serious underlying condition. **Evidence:** Evidence from studies examining the treatment effects of the LNG-IUD among women with heavy or prolonged bleeding reported no increase in adverse effects and found the LNG-IUD to be beneficial in treating menorrhagia (*69–76*).
Unexplained vaginal bleeding (suspicion for serious condition)	Initiation / Continuation	Initiation / Continuation	**Clarification:** If pregnancy or an underlying pathological condition (such as pelvic malignancy) is suspected, it must be evaluated and the category adjusted after evaluation. The IUD does not need to be removed before evaluation.
Before evaluation	4 / 2	4 / 2	

TABLE. (*Continued*) Classifications for intrauterine devices,*† including the LNG-IUD and the Cu-IUD

Condition	Category: LNG-IUD	Category: Cu-IUD	Clarifications/Evidence/Comments
Endometriosis	1	2	**Evidence:** LNG-IUD use among women with endometriosis decreased dysmenorrhea, pelvic pain, and dyspareunia (*77–81*).
Benign ovarian tumors (including cysts)	1	1	
Severe dysmenorrhea	1	2	**Comment:** Dysmenorrhea might intensify with Cu-IUD use. LNG-IUD use has been associated with reduction of dysmenorrhea.
Gestational trophoblastic disease			
a. Decreasing or undetectable β–hCG levels	3	3	**Evidence:** Limited evidence suggests that women using an IUD after uterine evacuation for a molar pregnancy are not at greater risk for postmolar trophoblastic disease than are women using other methods of contraception (*82–84*).
b. Persistently elevated β-hCG levels or malignant disease§	4	4	**Evidence:** Limited evidence suggests that women using an IUD after uterine evacuation for a molar pregnancy are not at greater risk for postmolar trophoblastic disease than are women using other methods of contraception (*82–84*)
Cervical ectropion	1	1	
Cervical intraepithelial neoplasia	2	1	**Comment:** Theoretical concern exists that LNG-IUDs might enhance progression of cervical intraepithelial neoplasia.
Cervical cancer (awaiting treatment)	Initiation 4 / Continuation 2	Initiation 4 / Continuation 2	**Comment:** Concern exists about the increased risk for infection and bleeding at insertion. The IUD most likely will need to be removed at the time of treatment, but until then, the woman is at risk for pregnancy.
Breast disease			
a. Undiagnosed mass	2	1	
b. Benign breast disease	1	1	
c. Family history of cancer	1	1	
d. Breast cancer§			**Comment:** Breast cancer is a hormonally sensitive tumor. Concerns about progression of the disease might be less with LNG-IUDs than with COCs or higher-dose POCs.
i. Current	4	1	
ii. Past and no evidence of current disease for 5 yrs	3	1	
Endometrial hyperplasia	1	1	**Evidence:** Among women with endometrial hyperplasia, no adverse health events occurred with LNG-IUD use; most women experienced disease regression (*85–93*).
Endometrial cancer§	Initiation 4 / Continuation 2	Initiation 4 / Continuation 2	**Comment:** Concern exists about the increased risk for infection, perforation, and bleeding at insertion. The IUD most likely will need to be removed at the time of treatment, but until then, the woman is at risk for pregnancy.
Ovarian cancer§	1	1	**Comment:** Women with ovarian cancer who undergo fertility sparing treatment and need contraception may use an IUD.
Uterine fibroids	2	2	**Evidence:** Among women with uterine fibroids using an LNG-IUD, most experienced improvements in serum levels of hemoglobin, hematocrit, and ferritin (*73,94–100*) and menstrual blood loss (*73,75,94–101*). Rates of LNG-IUD expulsion were higher in women with uterine fibroids (11%) than in women without fibroids (0%–3%); these findings were not statistically significant or significance testing was not conducted (*75,101*). Rates of expulsion from noncomparative studies ranged from 0%–20% (*94,96–100*). **Comment:** Women with heavy or prolonged bleeding should be assigned the category for that condition.
Anatomical abnormalities			
a. Distorted uterine cavity (any congenital or acquired uterine abnormality distorting the uterine cavity in a manner that is incompatible with IUD insertion)	4	4	**Comment:** An anatomic abnormality that distorts the uterine cavity might preclude proper IUD placement.
b. Other abnormalities (including cervical stenosis or cervical lacerations) not distorting the uterine cavity or interfering with IUD insertion	2	2	

TABLE. (*Continued*) Classifications for intrauterine devices,*† including the LNG-IUD and the Cu-IUD

Condition	Category: LNG-IUD		Category: Cu-IUD		Clarifications/Evidence/Comments
Pelvic inflammatory disease (PID)	Initiation	Continuation	Initiation	Continuation	
a. Past PID (assuming no known current risk factors for STIs)					**Comment:** IUDs do not protect against STI/HIV/PID. In women at low risk for STIs, IUD insertion poses little risk for PID. Current risk for STIs and desire for future pregnancy are relevant considerations.
i. With subsequent pregnancy	1	1	1	1	
ii. Without subsequent pregnancy	2	2	2	2	
b. Current PID	4	2	4	2	**Clarification for continuation:** Treat the PID using appropriate antibiotics. The IUD usually does not need to be removed if the woman wishes to continue using it. Continued use of an IUD depends on the woman's informed choice and her current risk factors for STIs and PID. **Evidence:** Among IUD users treated for PID, clinical course did not differ regardless of whether the IUD was removed or left in place (*102–104*).
STIs	Initiation	Continuation	Initiation	Continuation	
a. Current purulent cervicitis or chlamydial infection or gonorrhea	4	2	4	2	**Clarification for continuation:** Treat the STI using appropriate antibiotics. The IUD usually does not need to be removed if the woman wishes to continue using it. Continued use of an IUD depends on the woman's informed choice and her current risk factors for STIs and PID. **Evidence:** No evidence exists about whether IUD insertion among women with STIs increases the risk for PID over that of women with no IUD insertion. Among women who had an IUD inserted, the absolute risk for subsequent PID was low among women with STI at the time of insertion but greater than among women with no STI at the time of IUD insertion (*105–111*).
b. Other STIs (excluding HIV and hepatitis)	2	2	2	2	
c. Vaginitis (including *Trichomonas vaginalis* and bacterial vaginosis)	2	2	2	2	
d. Increased risk for STIs	2/3	2	2/3	2	**Clarification for initiation**: If a woman has a very high individual likelihood of exposure to gonorrhea or chlamydial infection, the condition is a Category 3. **Evidence:** Using an algorithm to classify STI risk status among IUD users, 1 study reported that 11% of women at high risk for STIs experienced IUD-related complications compared with 5% of those not classified as high risk (*107*).
HIV/AIDS					
High risk for HIV	Initiation	Continuation	Initiation	Continuation	
	2	2	2	2	**Evidence:** Among women at risk for HIV, Cu-IUD use did not increase risk for HIV acquisition (*112–122*).
HIV infection§	2	2	2	2	**Evidence:** Among IUD users, limited evidence shows no higher risk for overall complications or for infectious complications in HIV-infected than in HIV-uninfected women. IUD use did not adversely affect progression of HIV when compared with hormonal contraceptive use among HIV-infected women. Furthermore, IUD use among HIV-infected women was not associated with increased risk for transmission to sex partners (*112,123–130*).
AIDS§	3	2	3	2	**Clarification for continuation:** IUD users with AIDS should be closely monitored for pelvic infection.
Clinically well on ARV therapy	2	2	2	2	
Other Infections					
Schistosomiasis					
a. Uncomplicated		1		1	
b. Fibrosis of the liver§ (if severe, see cirrhosis)		1		1	
Tuberculosis§	Initiation	Continuation	Initiation	Continuation	
a. Nonpelvic	1	1	1	1	
b. Pelvic	4	3	4	3	**Comment:** Insertion of an IUD may substantially worsen the condition.
Malaria		1		1	

TABLE. (*Continued*) Classifications for intrauterine devices,*† including the LNG-IUD and the Cu-IUD

Condition	Category: LNG-IUD	Category: Cu-IUD	Clarifications/Evidence/Comments
Endocrine Conditions			
Diabetes			
a. History of gestational disease	1	1	
b. Nonvascular disease			**Evidence:** Limited evidence on the use of the LNG-IUD among women with insulin-dependent or noninsulin-dependent diabetes suggests that these methods have little effect on short-term or long-term diabetes control (e.g., glycosylated hemoglobin levels), hemostatic markers, or lipid profile (*131,132*).
i. Noninsulin-dependent	2	1	
ii. Insulin-dependent§	2	1	
c. Nephropathy/retinopathy/neuropathy§	2	1	
d. Other vascular disease or diabetes of >20 yrs' duration§	2	1	
Thyroid disorders			
a. Simple goiter	1	1	
b. Hyperthyroid	1	1	
c. Hypothyroid	1	1	
Gastrointestinal Conditions			
Inflammatory bowel disease (IBD) (ulcerative colitis, Crohn disease)	1	1	**Evidence:** Although two case reports described three women with IBD who experienced exacerbation of disease 5 days–25 months after LNG-IUD insertion (*133,134*), no comparative studies have examined the safety of IUD use among women with IBD.
Gallbladder disease			
a. Symptomatic			
i. Treated by cholecystectomy	2	1	
ii. Medically treated	2	1	
iii. Current	2	1	
b. Asymptomatic	2	1	
History of cholestasis			
a. Pregnancy-related	1	1	
b. Past COC-related	2	1	**Comment:** Concern exists that history of COC-related cholestasis might predict subsequent cholestasis with LNG use. Whether risk exists with use of LNG-IUD is unclear.
Viral hepatitis			
a. Acute or flare	1	1	
b. Carrier	1	1	
c. Chronic	1	1	
Cirrhosis			
a. Mild (compensated)	1	1	
b. Severe§ (decompensated)	3	1	
Liver tumors			
a. Benign	2	1	
i. Focal nodular hyperplasia			
ii. Hepatocellular adenoma§	3	1	**Comment:** No evidence is available about hormonal contraceptive use in women with hepatocellular adenoma. COC use in healthy women is associated with development and growth of hepatocellular adenoma; whether other hormonal contraceptives have similar effects is not known.
b. Malignant§ (hepatoma)	3	1	
Anemias			
Thalassemia	1	2	**Comment:** Concern exists about an increased risk for blood loss with Cu-IUDs.
Sickle cell disease§	1	2	**Comment:** Concern exists about an increased risk for blood loss with Cu-IUDs.
Iron deficiency anemia	1	2	**Comment:** Concern exists about an increased risk for blood loss with Cu-IUDs.
Solid Organ Transplantation			
Solid organ transplantation§	Initiation / Continuation	Initiation / Continuation	**Evidence:** No comparative studies have examined IUD use among transplant patients. Four case reports of transplant patients using IUDs provided inconsistent results, including beneficial effects and contraceptive failures (*135–138*).
a. Complicated: graft failure (acute or chronic), rejection, cardiac allograft vasculopathy	3 / 2	3 / 2	
b. Uncomplicated	2 / 2	2 / 2	

TABLE. (*Continued*) Classifications for intrauterine devices,*† including the LNG-IUD and the Cu-IUD

Condition	Category				Clarifications/Evidence/Comments
	LNG-IUD		Cu-IUD		
Drug Interactions					
Antiretroviral (ARV) therapy	Initiation	Continuation	Initiation	Continuation	**Clarification:** No known interaction exists between ARV therapy and IUD use. However, AIDS as a condition is classified as Category 3 for insertion and Category 2 for continuation unless the woman is clinically well on ARV therapy, in which case, both insertion and continuation are classified as Category 2 (see AIDS condition).
a. Nucleoside reverse transcriptase inhibitors (NRTIs)	2/3	2	2/3	2	
b. Non-nucleoside reverse transcriptase inhibitors (NNRTIs)	2/3	2	2/3	2	
c. Ritonavir-boosted protease inhibitors	2/3	2	2/3	2	
Anticonvulsant therapy					
a. Certain anticonvulsants (phenytoin, carbamazepine, barbiturates, primidone, topiramate, oxcarbazepine)		1		1	**Evidence:** Limited evidence suggests use of certain anticonvulsants does not interfere with the contraceptive effectiveness of the LNG-IUD (*139*).
b Lamotrigine		1		1	**Evidence:** No drug interactions have been reported among epileptic women taking lamotrigine and using the LNG-IUD (*140*).
Antimicrobial therapy					
a. Broad-spectrum antibiotics		1		1	
b. Antifungals		1		1	
c. Antiparasitics		1		1	
d. Rifampicin or rifabutin therapy		1		1	**Evidence:** One cross-sectional survey found that rifabutin had no impact on the effectiveness of the LNG-IUD (*139*).

* Abbreviations: LNG-IUD = levonorgestrel-releasing intrauterine device; Cu-IUD = copper IUD; STI = sexually transmitted infection; HIV = human immunodeficiency virus; BMI = body mass index; DVT = deep venous thrombosis; PE = pulmonary embolism; POC = progestin-only contraceptive; COC = combined oral contraceptive; SLE = systemic lupus erythematosus; MEC = Medical Eligibility Criteria; hCG = human chorionic gonadotropin; PID = pelvic inflammatory disease; AIDS = acquired immunodeficiency syndrome; ARV = antiretroviral; IBD = inflammatory bowel disease; NRTI = nucleoside reverse transcriptase inhibitor; NNRTI = non-nucleoside reverse transcriptase inhibitor.
† IUDs do not protect against STI/HIV. If risk exists for STI/HIV (including during pregnancy or postpartum), the correct and consistent use of condoms is recommended, either alone or with another contraceptive method. Consistent and correct use of the male latex condom reduces the risk for STIs and HIV transmission
§ Condition that exposes a woman to increased risk as a result of unintended pregnancy.

References

1. Cramer DW, Schiff I, Schoenbaum SC, Gibson M, Belisle S, Albrecht B, et al. Tubal infertility and the intrauterine device. N Engl J Med 1985;312:941–7.
2. Daling JR, Weiss NS, Metch BJ, Chow WH, Soderstrom RM, Moore DE, et al. Primary tubal infertility in relation to the use of an intrauterine device. N Engl J Med 1985;312:937–41.
3. Daling JR, Weiss NS, Voigt LF, McKnight B, Moore DE. The intrauterine device and primary tubal infertility. N Eng J Med 1992;326:203–4.
4. Delbarge W, Bátár I, Bafort M, Bonnivert J, Colmant C, Dhont M, et al. Return to fertility in nulliparous and parous women after removal of the GyneFix intrauterine contraceptive system. Eur J Contracept Reprod Health Care 2002;7:24–30.
5. Doll H, Vessey M, Painter R. Return of fertility in nulliparous women after discontinuation of the intrauterine device: comparison with women discontinuing other methods of contraception. BJOG 2001;108:304–14.
6. Hubacher D, et al. Use of copper intrauterine devices and the risk of tubal infertility among nulligravid women. N Engl J Med 2001;345:561–7.
7. Skjeldestad FE, Bratt H. Return of fertility after use of IUDs (Nova-T, MLCu250 and MLCu375). Adv Contracep 1987;3:139–45.
8. Urbach DR, Marrett LD, Kung R, Cohen MM. Association of perforation of the appendix with female tubal infertility. Am J Epidemiol 2001;153:566–71.
9. Wilson JC. A prospective New Zealand study of fertility after removal of copper intrauterine contraceptive devices for conception and because of complications: a four-year study. Am J Obstet Gynecol 1989;160:391–6.
10. Thiery M, Vanderpas H, Delbeke L, Vankets H. Comparative performance of 2 copper-wired IUDs (Ml-Cu-250 and T-Cu-200): immediate postpartum and interval insertion. Contracept Deliv Syst 1980;1:27–35.
11. Thiery M, Van Kets H, Van der PH, van Os W, Dombrowicz N. The ML Cu250; clinical experience in Belgium and the Netherlands. Br J Obstet Gynaecol 1982;89:51–3.
12. Brenner PF. A clinical trial of the Delta-T intrauterine device: immediate postpartum insertion. Contraception 1983;28:135–47.
13. Chi IC, Wilkens L, Rogers S. Expulsions in immediate postpartum insertions of Lippes loop D and copper T IUDs and their counterpart Delta devices—an epidemiological analysis. Contraception 1985;32:119–34.
14. Morrison C, Waszak C, Katz K, Diabate F, Mate EM. Clinical outcomes of two early postpartum IUD insertion programs in Africa. Contraception 1996;53:17–21.
15. El-Shafei MM, Mashali A, Hassan EO, El-Boghdadi, El-Lakkany N. Postpartum and postabortion intrauterine device insertion unmet needs of safe reproductive health: three years experience of a Mansoura University Hospital. Egypt Soc Obstet Gynecol 2000;26:253–62.
16. Muller ALL, Ramos JGL, Martins-Costa SH, et al. Transvaginal ultrasonographic assessment of the expulsion rate of intrauterine devices inserted in the immediate postpartum period: a pilot study. Contraception 2005;72:192–5.
17. Zhou SW, Chi IC. Immediate postpartum IUD insertions in a Chinese hospital—a two year follow-up. Int J Gynaecol Obstet 1991;35:157–64.
18. Bonilla Rosales F, Aguilar Zamudio ME, Cazares Montero Mde L, Hernandez Ortiz ME, Luna Ruiz MA. Factors for expulsion of intrauterine device Tcu380A applied immediately postpartum and after a delayed period [in Spanish]. Rev Med Inst Mex Seguro Soc 2005;43:5–10.
19. Lara R, Sanchez RA, Aznar R. Application of intreauterine device through the incision of the Cesarean section [in Spanish]. Ginecol Obstet Mex 1989;57:23–7.
20. Welkovic S, Costa LO, Faundes A, de Alencar Ximenes R, Costa CF. Post-partum bleeding and infection after post-placental IUD insertion. Contraception 2001;63:155–8.
21. Celen S, Moroy P, Sucak A, Aktulay A, Danisman N. Clinical outcomes of early postplacental insertion of intrauterine contraceptive devices. Contraception 2004;69:279–82.
22. Eroglu K, Akkuzu G, Vural G, et al. Comparison of efficacy and complications of IUD insertion in immediate postplacental/early postpartum period with interval period: 1 year follow-up. Contraception 2006;74:376–81.

23. Mishell DR, Jr., Roy S. Copper intrauterine contraceptive device event rates following insertion 4 to 8 weeks post partum. Am J Obstet Gynecol 1982;143:29–35.
24. World Health Organization's Special Programme of Research DaRTiHR. Task Force on Intrauterine Devices for Fertility Regulation. IUD insertion following spontaneous abortion: a clinical trial of the TCu 220C, Lippes loop D, and copper 7. Stud Fam Plann 1983;14:109–14.
25. World Health Organization's Special Programme of Research DaRTiHR. Task Force on Intrauterine Devices for Fertility Regulation. IUD insertion following termination of pregnancy: a clinical trial of the TCu 220C, Lippes loop D, and copper 7. Stud Fam Plann 1983;14:99–108.
26. World Health Organization's Special Programme of Research DaRTiHR. Task Force on Intrauterine Devices for Fertility Regulation. The Alza T IPCS 52, a longer acting progesterone IUD: safety and efficacy compared to the TCu220C and multiload 250 in two randomized multicentre trials. Clin Reprod Fertil 1983;2:113–28.
27. El Tagy A, Sakr E, Sokal DC, Issa AH. Safety and acceptability of postabortal IUD insertion and the importance of counseling. Contraception 2003;67:229–34.
28. Gillett PG, Lee NH, Yuzpe AA, Cerskus I. A comparison of the efficacy and acceptability of the copper-7 intrauterine device following immediate or delayed insertion after first-trimester therapeutic abortion. Fertil Steril 1980;34:121–4.
29. Grimes D, Schulz K, Stanwood N. Immediate postabortal insertion of intrauterine devices. [update of Cochrane Database Syst Rev. 2000;(2):CD001777; PMID:10796820]. [Review]. Cochrane Database Syst Rev 2002;CD001777.
30. Gupta I, Devi PK. Studies on immediate post-abortion copper 'T' device. Indian J Med Res 1975;63:736–9.
31. Moussa A. Evaluation of postabortion IUD insertion in Egyptian women. Contraception 2001;63:315–7.
32. Pakarinen P, Toivonen J, Luukkainen T. Randomized comparison of levonorgestrel- and copper-releasing intrauterine systems immediately after abortion, with 5 years' follow-up. Contraception 2003;68:31–4.
33. Stanwood NL, Grimes DA, Schulz KF. Insertion of an intrauterine contraceptive device after induced or spontaneous abortion: a review of the evidence. BJOG 2001;108:1168–73.
34. Suvisaari J, Lahteenmaki P. Detailed analysis of menstrual bleeding patterns after postmensstrual and postabortal insertion of a copper IUD or a levonorgestrel-releasing intrauterine system. Contraception 1996;54:201–8.
35. Timonen H, Luukkainen T. Immediate postabortion insertion of the copper-T (TCu-200) with eighteen months follow-up. Contraception 1974;9:153–60.
36. Tuveng JM, Skjeldestad FE, Iverson T. Postabortal insertion of IUD. Adv Contracept 1986;2:387–92.
37. Zhang PZ. Five years experience with the copper T 200 in Shanghai—856 cases. Contraception 1980;22:561–71.
38. Cardiovascular disease and use of oral and injectable progestogen-only contraceptives and combined injectable contraceptives. Results of an international, multicenter, case-control study. World Health Organization Collaborative Study of Cardiovascular Disease and Steroid Hormone Contraception. Contraception 1998;57:315–24.
39. Heinemann LA, Assmann A, DoMinh T, et al. Oral progestogen-only contraceptives and cardiovascular risk: results from the Transnational Study on Oral Contraceptives and the Health of Young Women. Eur J Contracept Reprod Health Care 1999;4:67–73.
40. Vasilakis C, Jick H, Mar Melero-Montes M. Risk of idiopathic venous thromboembolism in users of progestogens alone. Lancet 1999;354:1610–1.
41. Kingman CE, Kadir RA, Lee CA, et al. The use of the levonorgestrel-releasing intrauterine system for treatment of menorrhagia in women withinherited bleeding disorders. BJOG 2004;111:1425–8.
42. Pisoni CN, Cuadrado MJ, Khamashta MA, et al. Treatment of menorrhagia associated with oral anticoagulation: efficacy and safety of the levonorgestrel releasing intrauterine device (Mirena coil). Lupus 2006;15:877–80.
43. Schaedel ZE, Dolan G, Powell MC. The use of the levonorgestrel-releasing intrauterine system in the management of menorrhagia in women with hemostatic disorders. Am J Obstet Gynecol 2005;193:1361–3.
44. Lukes AS, Reardon B, Arepally G. Use of the levonorgestrel-releasing intrauterine system in women with hemostatic disorders. Fertil Steril 2008;90:673–7.
45. Wilson W, Taubert KA, Gewitz M et al. Prevention of infective endocarditis: guidelines from the American Heart Association: a guideline from the American Heart Association Rheumatic Fever, Endocarditis, and Kawasaki Disease Committee, Council on Cardiovascular Disease in the Young, and the Council on Clinical Cardiology, Council on Cardiovascular Surgery and Anesthesia, and the Quality of Care and Outcomes Research Interdisciplinary Working Group. Circulation. 2007;116:1736–1754.
46. The Criteria Committee of the New York Heart Association. Nomenclature and criteria for diagnosis of diseases of the heart and great vessels. 9th ed. Boston, MA: Little, Brown & Co; 1994.
47. Avila WS, Grinberg M, Melo NR, Aristodemo PJ, Pileggi F. Contraceptive use in women with heart disease [in Portuguese]. Arq Bras Cardiol 1996;66:205–11.
48. Suri V, Aggarwal N, Kaur R, et al. Safety of intrauterine contraceptive device (copper T 200 B) in women with cardiac disease. Contraception 2008;78:315–8.
49. Bernatsky S, Ramsey-Goldman R, Gordon C, et al. Factors associated with abnormal Pap results in systemic lupus erythematosus. Rheumatology (Oxford) 2004;43:1386–9.
50. Bernatsky S, Clarke A, Ramsey-Goldman R, et al. Hormonal exposures and breast cancer in a sample of women with systemic lupus erythematosus. Rheumatology (Oxford) 2004;43:1178–81.
51. Chopra N, Koren S, Greer WL, et al. Factor V Leiden, prothrombin gene mutation, and thrombosis risk in patients with antiphospholipid antibodies. J Rheumatol 2002;29:1683–8.
52. Esdaile JM, Abrahamowicz M, Grodzicky T, et al. Traditional Framingham risk factors fail to fully account for accelerated atherosclerosis in systemic lupus erythematosus. Arthritis Rheum 2001;44:2331–7.
53. Julkunen HA. Oral contraceptives in systemic lupus erythematosus: side-effects and influence on the activity of SLE. Scand J Rheumatol 1991;20:427–33.
54. Julkunen HA, Kaaja R, Friman C. Contraceptive practice in women with systemic lupus erythematosus. Br J Rheumatol 1993;32:227–30.
55 Jungers P, Dougados M, Pelissier C, et al. Influence of oral contraceptive therapy on the activity of systemic lupus erythematosus. Arthritis Rheum 1982;25:618–23.
56. Manzi S, Meilahn EN, Rairie JE, et al. Age-specific incidence rates of myocardial infarction and angina in women with systemic lupus erythematosus: comparison with the Framingham Study. Am J Epidemiol 1997;145:408–15.
57. McAlindon T, Giannotta L, Taub N, et al. Environmental factors predicting nephristis in systemic lupus erythematosus. Ann Rheum Dis 1993;52:720–4.
58. McDonald J, Stewart J, Urowitz MB, et al. Peripheral vascular disease in patients with systemic lupus erythematosus. Ann Rheum Dis 1992;51:56–60.
59. Mintz G, Gutierrez G, Deleze M, et al. Contraception with progestogens in systemic lupus erythematosus. Contraception 1984;30:29–38.
60. Petri M. Musculoskeletal complications of systemic lupus erythematosus in the Hopkins Lupus Cohort: an update. Arthritis Care Res 1995;8:137–45.
61. Petri M, Kim MY, Kalunian KC, et al. Combined oral contraceptives in women with systemic lupus erythematosus. N Engl J Med 2005;353:2550–8.

62. Petri M. Lupus in Baltimore: evidence-based 'clinical perarls' from the Hopkins Lupus Cohort. Lupus 2005;14:970–3.
63. Sanchez-Guerrero J, Uribe AG, Jimenez-Santana L, et al. A trial of contraceptive methods in women with systemic lupus erythematosus. N Eng J Med 2005;353:2539–49.
64. Sarabi ZS, Chang E, Bobba R, et al. Incidence rates of arterial and venous thrombosis after diagnosis of systemic lupus erythematosus. Arthritis Rheum 2005;53:609–12.
65. Somers E, Magder LS, Petri M. Antiphospholipid antibodies and incidence of venous thrombosis in a cohort of patients with systemic lupus erythematosus. J Rheumatol 2002;29:2531–6.
66. Urowitz MB, Bookman AA, Koehler BE, et al. The bimodal mortality pattern of systemic lupus erythematosus. Am J Med 1976;60:221–5.
67. Choojitarom K, Verasertniyom O, Totemchokchyakarn K, et al. Lupus nephritis and Raynaud's phenomenon are significant risk factors for vascular thrombosis in SLE patients with positive antiphospholipid antibodies. Clin Rheumatol 2008;27:345–51.
68. Wahl DG, Guillemin F, de Maistre E, et al. Risk for venous thrombosis related to antiphospholipid antibodies in systemic lupus erythematosus—a meta-analysis. Lupus 1997;6:467–73.
69. Barrington JW, Arunkalaivanan AS, bdel-Fattah M. Comparison between the levonorgestrel intrauterine system (LNG-IUS) and thermal balloon ablation in the treatment of menorrhagia. Eur J Obstet Gynecol Reprod Biol 2003;108:72–4.
70. Gupta B, Mittal S, Misra R, Deka D, Dadhwal V. Levonorgestrel-releasing intrauterine system vs. transcervical endometrial resection for dysfunctional uterine bleeding. Int J Gynaecol Obstet 2006;95:261–6.
71. Hurskainen R, Teperi J, Rissanen P, et al. Quality of life and cost-effectiveness of levonorgestrel-releasing intrauterine system versus hysterectomy for treatment of menorrhagia: a randomised trial [see comment]. Lancet 2001;357:273–7.
72. Istre O, Trolle B. Treatment of menorrhagia with the levonorgestrel intrauterine system versus endometrial resection. Fertil Steril 2001;76:304–9.
73. Koh SC, Singh K. The effect of levonorgestrel-releasing intrauterine system use on menstrual blood loss and the hemostatic, fibrinolytic/inhibitor systems in women with menorrhagia. J Thromb Haemost 2007;5:133–8.
74. Lethaby AE, Cooke I, Rees M. Progesterone/progestogen releasing intrauterine systems versus either placebo or any other medication for heavy menstrual bleeding. Cochrane Database Syst Rev 2000;CD002126.
75. Magalhaes J, Aldrighi JM, de Lima GR. Uterine volume and menstrual patterns in users of the levonorgestrel-releasing intrauterine system with idiopathic menorrhagia or menorrhagia due to leiomyomas. Contraception 2007;75:193–8.
76. Stewart A, Cummins C, Gold L, Jordan R, Phillips W. The effectiveness of the levonorgestrel-releasing intrauterine system in menorrhagia: a systematic review.
77. Fedele L, Bianchi S, Zanconato G, Portuese A, Raffaelli R. Use of a levonorgestrel-releasing intrauterine device in the treatment of rectovaginal endometriosis. Fertil Steril 2001;75:485–8.
78. Lockhat FBE. The effect of a levonorgestrel intrauterine system (LNG-IUS) on symptomatic endometriosis. Fertil Steril 2002;77 Suppl 1:S24.
79. Petta CA, Ferriani RA, Abrao MS, et al. Randomized clinical trial of a levonorgestrel-releasing intrauterine system and a depot GnRH analogue for the treatment of chronic pelvic pain in women with endometriosis. Hum Reprod 2005;20:1993–8.
80. Vercellini P, Aimi G, Panazza S, et al. A levonorgestrel-releasing intrauterine system for the treatment of dysmenorrhea associated with endometriosis: a pilot study. Fertil Steril 1999;72:505–8.
81. Vercellini P, Frontino G, De Giorgi O, et al. Comparison of a levonorgestrel-releasing intrauterine device versus expectant management after conservative surgery for symptomatic endometriosis: a pilot study. Fertil Steril 2003;80:305–9.
82. Deicas RE, Miller DS, Rademaker AW, Lurain JR. The role of contraception in the development of postmolar trophoblastic tumour. Obstet Gynecol 1991;78:221–6.
83. Adewole IF, Oladokun A, Fawole AO, Olawuyi JF, Adeleye JA. Fertility regulatory methods and development of complications after evacuation of complete hydatidiform mole. J Obstet Gynecol 2000;20:68–9.
84. Ho Yuen B, Burch P. Relationship of oral contraceptives and the intrauterine contraceptive devices to the regression of concentration of the beta subunit of human chorionic gonadotropin and invasive complications after molar pregnancy. Am J Obstet Gynecoy 1983;145:214–7.
85. Haimovich S, Checa MA, Mancebo G, Fuste P, Carreras R. Treatment of endometrial hyperplasia without atypia in peri- and postmenopausal women with a levonorgestrel intrauterine device. Menopause 2008;15:1002–7.
86. Varma R, Soneja H, Bhatia K, et al. The effectiveness of a levonorgestrel-releasing intrauterine system (LNG-IUS) in the treatment of endometrial hyperplasia—a long-term follow-up study. Eur J Obstet Gynecol Reprod Biol 2008;139:169–75.
87. Wheeler DT, Bristow RE, Kurman RJ. Histologic alterations in endometrial hyperplasia and well-differentiated carcinoma treated with progestins. Am J Surg Pathol 2007;31:988–98.
88. Wildemeersch D, Janssens D, Pylyser K, et al. Management of patients with non-atypical and atypical endometrial hyperplasia with a levonorgestrel-releasing intrauterine system: long-term follow-up. Maturitas 2007;57:210–3.
89. Clark TJ, Neelakantan D, Gupta JK. The management of endometrial hyperplasia: an evaluation of current practice. Eur J Obstet Gynecol Reprod Biol 2006;125:259–64.
90. Vereide AB, Arnes M, Straume B, Maltau JM, Orbo A. Nuclear morphometric changes and therapy monitoring in patients with endometrial hyperplasia: a study comparing effects of intrauterine levonorgestrel and systemic medroxyprogesterone. Gynecol Oncol 2003;91:526–33.
91. Perino A, Quartararo P, Catinella E, Genova G, Cittadini E. Treatment of endometrial hyperplasia with levonorgestrel releasing intrauterine devices. Acta Eur Fertil 1987;18:137–40.
92. Scarselli G, Mencaglia L, Tantini C, Colafranceschi M, Taddei G. Hysteroscopic evaluation of intrauterine progesterone contraceptive system as a treatment for abnormal uterine bleeding. Acta Eur Fertil 1984;15:279–82.
93. Orbo A, Arnes M, Hancke C, et al. Treatment results of endometrial hyperplasia after prospective D-score classification: a follow-up study comparing effect of LNG-IUD and oral progestins versus observation only. Gynecol Oncol 2008;111:68–73.
94. Jindabanjerd K, Taneepanichskul S. The use of levonorgestrel–IUD in the treatment of uterine myoma in Thai women. J Med Assoc Thai 2006;89 Suppl 4:S147–51.
95. Tasci Y, Caglar GS, Kayikcioglu F, Cengiz H, Yagci B, Gunes M. Treatment of menorrhagia with the levonorgestrel releasing intrauterine system: effects on ovarian function and uterus. Arch Gynecol Obstet 2009;280:39–42
96. Rosa E Silva JC, de Sa Rosa e Silva AC, Candido dos Reis FJ, et al. Use of a levonorgestrel-releasing intrauterine device for the symptomatic treatment of uterine myomas. J Reprod Med 2005;50:613–7.
97. Mercorio F, De SR, Di Spiezio SA, et al. The effect of a levonorgestrel-releasing intrauterine device in the treatment of myoma-related menorrhagia. Contraception 2003;67:277–80.

98. Grigorieva V, Chen-Mok M, Tarasova M, Mikhailov A. Use of a levonorgestrel-releasing intrauterine system to treat bledding related to uterine leiomyomas. Fertil Steril 2003;79:1194–8.
99. Starczewski A, Iwanicki M. Intrauterine therapy with levonorgestrel releasing IUD of women with hypermenorrhea secondary to uterine fibroids [in Polish]. Ginekol Pol 2000;71:1221-5.
100. Soysal S, Soysal ME. The efficacy of levonorgestrel-releasing intrauterine device in selected cases of myoma-related menorrhagia: a prospective controlled trial. Gynecol Obstet Invest 2005;59:29–35.
101. Ikomi A, Mansell E, Spence-Jones C, Singer A. Treatment of menorrhagia with the levonorgestrel intrauterine system: can we learn from our failures? J Obstet Gynaecol 2000;20:630–1.
102. Larsson B, Wennergren M. Investigation of a copper-intrauterine device (Cu-IUD) for possible effect on frequency and healing of pelvic inflammatory disease. Contraception 1977;15:143–9.
103. Soderberg G, Lindgren S. Influence of an intrauterine device on the course of an acute salpingitis. Contraception 1981;24:137–43.
104. Teisala K. Removal of an intrauterine device and the treatment of acute pelvic inflammatory disease. Ann Med 1989;21:63–5.
105. Faúndes A, Telles E, Cristofoletti ML, Faúndes D, Castro S, Hardy E. The risk of inadvertent intrauterine device insertion in women carriers of endocervical *Chlamydia trachomatis*. Contraception 1998;58:105–9.
106. Ferraz do Lago R, Simões JA, Bahamondes L, et al. Follow-up of users of intrauterine device with and without bacterial vaginosis and other cervicovaginal infections. Contraception 2003;68:105–9.
107. Morrison CS, Sekadde-Kigondu C, Miller WC, Weiner DH, Sinei SK. Use of sexually transmitted disease risk assessment algorithms for selection of intrauterine device candidates. Contraception 1999;59:97–106.
108. Pap-Akeson M, Solheim F, Thorbert G, Akerlund M. Genital tract infections associated with the intrauterine contraceptive device can be reduced by inserting the threads into the uterine cavity. Br J Obstet Gynaecol 1992;99:676–9.
109. Sinei SK, Schulz KF, Lamptey PR, Grimes DA, Mati JK, Rosenthal SM, et al. Preventing IUDC-related pelvic infection: the efficacy of prophylactic doxycycline at insertion. Br J Obstet Gynaecol 1990;97:412–9.
110. Skjeldestad FE, Halvorsen LE, Kahn H, Nordbø SA, Saake K. IUD users in Norway are at low risk of for genital *C. trachomatis* infection. Contraception 1996;54:209–12.
111. Walsh TL, Bernstein GS, Grimes DA, Frezieres R, Bernstein L, Coulson AH. Effect of prophylactic antibiotics on morbidity associated with IUD insertion: results of a pilot randomized controlled trial. Contraception 1994;50:319–27.
112. European Study Group on Heterosexual Transmission of HIV. Comparison of female to male and male to female transmission of HIV in 563 stable couples. BMJ 1992;304:809–13.
113. Carael M, Van de Perre PH, Lepage PH, et al. Human immunodeficiency virus transmission among heterosexual couples in Central Africa. AIDS 1988;2:201–5.
114. Kapiga SH, Shao JF, Lwihula GK, Hunter DJ. Risk factors for HIV infection among women in Dar-es-Salaam, Tanzania. J Acquir Immune Defic Syndr 1994;7:301–9.
115. Kapiga SH, Lyamuya EF, Lwihula GK, Hunter DJ. The incidence of HIV infection among women using family planning methods in Dar es Salaam, Tanzania. AIDS 1998;12:75–84.
116. Mann JM, Nzilambi N, Piot P, et al. HIV infection and associated risk factors in female prostitutes in Kinshasa, Zaire. AIDS 1998;2:249–54.
117. Martin HL, Jr., Nyange PM, Richardson BA, et al. Hormonal contraception, sexually transmitted diseases, and risk of heterosexual transmission of human immunodeficiency virus type 1. J Infect Dis 1998;178:1053–9.
118. Mati JK, Hunter DJ, Maggwa BN, Tukei PM. Contraceptive use and the risk of HIV infection in Nairobi, Kenya. Int J Gynaecol Obstet 1995;48:61–7.
119. Nicolosi A, Correa Leite ML, Musicco M, et al. The efficiency of male-to-female and female-to-male sexual transmission of the human immunodeficiency virus: a study of 730 stable couples. Italian Study Group on HIV Heterosexual Transmission [comment]. Epidemiology 1994;5:570–5.
120. Plourde PJ, Plummer FA, Pepin J, et al. Human immunodeficiency virus type 1 infection in women attending a sexually transmitted diseases clinic in Kenya [comment]. J Infect Dis 1992;166:86–92.
121. Sinei SK, Fortney JA, Kigondu CS, et al. Contraceptive use and HIV infection in Kenyan family planning clinic attenders. Int J STD AIDS 1996;7:65–70.
122. Spence MR, Robbins SM, Polansky M, Schable CA. Seroprevalence of human immunodeficiency virus type I (HIV-1) antibodies in a family-planning population. Sex Transm Dis 1991;18:143–5.
123. Morrison CS, Sekadde-Kigondu C, Sinei SK, et al. Is the intrauterine device appropriate contraception for HIV-1–infected women? BJOG 2001;108:784–90.
124. Richardson BA, Morrison CS, Sekadde-Kigondu C, et al. Effect of intrauterine device use on cervical shedding of HIV-1 DNA. AIDS 1999;13:2091–7.
125. Sinei SK, Morrison CS, Sekadde-Kigondu C, Allen M, Kokonya D. Complications of use of intrauterine devices among HIV-1–infected women. Lancet 1998;351:1238–41.
126. Mostad SB, Overbaugh J, DeVange DM, et al. Hormonal contraception, vitamin A deficiency, and other risk factors for shedding of HIV-1 infected cells from the cervix and vagina. Lancet 1997;350:922–7.
127. Kovacs A, Wasserman SS, Burns D, et al. Determinants of HIV-1 shedding in the genital tract of women. Lancet 2001;358:1593–601.
128. Stringer EM, Kaseba C, Levy J, et al. A randomized trial of the intrauterine contraceptive device vs hormonal contraception in women who are infected with the human immunodeficiency virus. Am J Obstet Gynecol 2007;197:144–8.
129. Heikinheimo O, Lehtovirta P, Suni J, Paavonen J. The levonorgestrel-releasing intrauterine system (LNG-IUS) in HIV-infected women—effects on bleeding patterns, ovarian function and genital shedding of HIV. Hum Reprod 2006;21:2857–61.
130. Lehtovirta P, Paavonen J, Heikinheimo O. Experience with the levonorgestrel-releasing intrauterine system among HIV-infected women. Contraception 2007;75:37–9.
131. Grigoryan OR, Grodnitskaya EE, Andreeva EN, et al. Contraception in perimenopausal women with diabetes mellitus. Gynecol Endocrinol 2006;22:198–206.
132. Rogovskaya S, Rivera R, Grimes DA, et al. Effect of a levonorgestrel intrauterine system on women with type 1 diabetes: a randomized trial. Obstet Gynecol 2005;105:811–5.
133. Cox M, Tripp J, Blacksell S. Clinical performance of the levonorgestrel intrauterine system in routine use by the UK Family Planning and Reproductive Health Research Network: 5-year report. J Fam Plann Reprod Health Care 2002;28:73–7.
134. Wakeman J. Exacerbation of Crohn's disease after insertion of a levonorgestrel intrauterine system: a case report. J Fam Plann Reprod Health Care 2003;29:154.

135. Fong YF, Singh K. Effect of the levonorgestrel-releasing intrauterine system on uterine myomas in a renal transplant patient. Contraception 1999;60:51–3.
136. Zerner J, Doil KL, Drewry J, Leeber DA. Intrauterine contraceptive device failures in renal transplant patients. J Reprod Med 1981;26:99–102.
137. Lessan-Pezeshki M, Ghazizadeh S, Khatami MR, et al. Fertility and contraceptive issues after kidney transplantation in women. Transplant Proc 2004;36:1405–6.
138. O'Donnell D. Contraception in the female transplant recipient. Dialysis & Transplantation 1986;15:610,612.
139. Bounds W, Guillebaud J. Observational series on women using the contraceptive Mirena concurrently with anti-epileptic and other enzyme-inducing drugs. J Fam Plann Reprod Health Care 2002;28:78–80.
140. Reimers A, Helde G, Brodtkorb E. Ethinyl estradiol, not progestogens, reduces lamotrigine serum concentrations. Epilepsia 2005;46:1414–7.

Appendix F

Classifications for Copper Intrauterine Devices for Emergency Contraception

A copper IUD (Cu-IUD) can be used within 5 days of unprotected intercourse as an emergency contraceptive. However, when the time of ovulation can be estimated, the Cu-IUD can be inserted beyond 5 days after intercourse, if necessary, as long as the insertion does not occur >5 days after ovulation.

The eligibility criteria for interval Cu-IUD insertion also apply for the insertion of Cu-IUDs as emergency contraception (Box). Cu-IUDs for emergency contraception do not protect against sexually transmitted infections (STIs) or human immunodeficiency virus (HIV).

BOX. Categories for Classifying Cu-IUDs as Emergency Contraception

1 = A condition for which there is no restriction for the use of the contraceptive method.
2 = A condition for which the advantages of using the method generally outweigh the theoretical or proven risks.
3 = A condition for which the theoretical or proven risks usually outweigh the advantages of using the method.
4 = A condition that represents an unacceptable health risk if the contraceptive method is used.

TABLE. Classifications for copper intrauterine devices for emergency contraception*†

Condition	Category	Clarifications/Evidence/Comments
Pregnancy	4	**Clarification**: IUD use is not indicated during pregnancy and should not be used because of the risk for serious pelvic infection and septic spontaneous abortion.
Rape		
a. High risk for STI	3	**Comment:** IUDs do not protect against STI/HIV or PID. Among women with chlamydial infection or gonorrhea, the potential increased risk for PID with IUD insertion should be avoided. The concern is less for other STIs.
b. Low risk for STI	1	

* Abbreviations: IUD = intrauterine device; Cu-IUD = copper IUD; STI = sexually transmitted infection; HIV = human immunodeficiency virus; PID = pelvic inflammatory disease

† Cu-IUDs for emergency contraception do not protect against STI/HIV. If risk exists for STI/HIV (including during pregnancy or postpartum), the correct and consistent use of condoms is recommended, either alone or with another contraceptive method. Consistent and correct use of the male latex condom reduces the risk for STIs and HIV transmission.

Appendix G
Classifications for Barrier Methods

Classifications for barrier contraceptive methods include those for condoms, which include male latex condoms, male polyurethane condoms, and female condoms; spermicides; and diaphragm with spermicide or cervical cap (Box). Consistent and correct use of the male latex condom reduces the risk for STI/HIV transmission.

Women with conditions that make pregnancy an unacceptable risk should be advised that barrier methods for pregnancy prevention may not be appropriate for those who cannot use them consistently and correctly because of the relatively higher typical-use failure rates of these methods.

BOX. Categories for Classifying Barrier Methods

1 = A condition for which there is no restriction for the use of the contraceptive method.
2 = A condition for which the advantages of using the method generally outweigh the theoretical or proven risks.
3 = A condition for which the theoretical or proven risks usually outweigh the advantages of using the method.
4 = A condition that represents an unacceptable health risk if the contraceptive method is used.

TABLE. Classifications for barrier methods,*† including condoms, spermicides, and diaphragms/caps

Condition	Category: Condom	Category: Spermicide	Category: Diaphragm/cap	Clarifications/Evidence/Comments
Personal Characteristics and Reproductive History				
Pregnancy	Not applicable	Not applicable	Not applicable	**Clarification:** None of these methods are relevant for contraception during known pregnancy. However, for women who remain at risk for STI/HIV during pregnancy, the correct and consistent use of condoms is recommended.
Age				
a. Menarche to <40 yrs	1	1	1	
b. ≥40 yrs	1	1	1	
Parity				
a. Nulliparous	1	1	1	
b. Parous	1	1	2	**Clarification:** Risk for cervical cap failure is higher in parous women than in nulliparous women.
Postpartum				
a. <6 wks postpartum	1	1	Not applicable	**Clarification:** Diaphragm and cap are unsuitable until uterine involution is complete.
b. ≥6 wks postpartum	1	1	1	
Postabortion				
a. First trimester	1	1	1	
b. Second trimester	1	1	1	**Clarification:** Diaphragm and cap are unsuitable until 6 weeks after second trimester abortion.
c. Immediate postseptic abortion	1	1	1	
Past ectopic pregnancy	1	1	1	
History of pelvic surgery	1	1	1	
Smoking				
a. Age <35 yrs	1	1	1	
b. Age ≥35 yrs				
i. <15 Cigarettes/day	1	1	1	
ii. ≥15 Cigarettes/day	1	1	1	
Obesity				**Comment:** Severe obesity might make diaphragm and cap placement difficult.
a. ≥30 kg/m^2 BMI	1	1	1	
b. Menarche to <18 yrs and ≥30 kg/m^2 BMI	1	1	1	
History of bariatric surgery§				
a. Restrictive procedures: decrease storage capacity of the stomach (vertical banded gastroplasty, laparoscopic adjustable gastric band, laparoscopic sleeve gastrectomy)	1	1	1	

TABLE. (*Continued*) Classifications for barrier methods,*† including condoms, spermicides, and diaphragms/caps

Condition	Category: Condom	Category: Spermicide	Category: Diaphragm/cap	Clarifications/Evidence/Comments
b. Malabsorptive procedures: decrease absorption of nutrients and calories by shortening the functional length of the small intestine (Roux-en-Y gastric bypass, biliopancreatic diversion)	1	1	1	
Cardiovascular Disease				
Multiple risk factors for arterial cardiovascular disease (such as older age, smoking, diabetes, and hypertension)	1	1	1	
Hypertension				
a. Adequately controlled hypertension	1	1	1	
b. Elevated blood pressure levels (properly taken measurements)				
i. Systolic 140–159 mm Hg or diastolic 90–99 mm Hg	1	1	1	
ii. Systolic ≥160 mm Hg or diastolic ≥100 mm Hg§	1	1	1	
c. Vascular disease	1	1	1	
History of high blood pressure during pregnancy (where current blood pressure is measurable and normal)	1	1	1	
Deep venous thrombosis (DVT)/pulmonary embolism (PE)				
a. History of DVT/PE, not on anticoagulant therapy				
i. Higher risk for recurrent DVT/PE (≥1 risk factors)	1	1	1	
• History of estrogen-associated DVT/PE				
• Pregnancy-associated DVT/PE				
• Idiopathic DVT/PE				
• Known thrombophilia, including antiphospholipid syndrome				
• Active cancer (metastatic, on therapy, or within 6 mos after clinical remission), excluding non-melanoma skin cancer				
• History of recurrent DVT/PE				
ii. Lower risk for recurrent DVT/PE (no risk factors)	1	1	1	
b. Acute DVT/PE	1	1	1	
c. DVT/PE and established on anticoagulant therapy for at least 3 mos				
i. Higher risk for recurrent DVT/PE (≥1 risk factors)	1	1	1	
• Known thrombophilia, including antiphospholipid syndrome				
• Active cancer (metastatic, on therapy, or within 6 mos after clinical remission), excluding non-melanoma skin cancer				
• History of recurrent DVT/PE				
ii. Lower risk for recurrent DVT/PE (no risk factors)	1	1	1	
d. Family history (first-degree relatives)	1	1	1	
e. Major surgery				
i. With prolonged immobilization	1	1	1	
ii. Without prolonged immobilization	1	1	1	
f. Minor surgery without immobilization	1	1	1	
Known thrombogenic mutations§ (e.g., factor V Leiden; prothrombin mutation; protein S, protein C, and antithrombin deficiencies)	1	1	1	**Clarification:** Routine screening is not appropriate because of the rarity of the conditions and the high cost of screening.

TABLE. (*Continued*) Classifications for barrier methods,*† including condoms, spermicides, and diaphragms/caps

Condition	Category: Condom	Category: Spermicide	Category: Diaphragm/cap	Clarifications/Evidence/Comments
Superficial venous thrombosis				
a. Varicose veins	1	1	1	
b. Superficial thrombophlebitis	1	1	1	
Current and history of ischemic heart disease§	1	1	1	
Stroke§ (history of cerebrovascular accident)	1	1	1	
Known hyperlipidemias	1	1	1	**Clarification:** Routine screening is not appropriate because of the rarity of the conditions and the high cost of screening.
Valvular heart disease				
a. Uncomplicated	1	1	1	
b. Complicated§ (pulmonary hypertension, risk for atrial fibrillation, history of subacute bacterial endocarditis)	1	1	2	
Peripartum cardiomyopathy§				
a. Normal or mildly impaired cardiac function (New York Heart Association Functional Class I or II: patients with no limitation of activities or patients with slight, mild limitation of activity) (*1*)				
i. <6 mos	1	1	1	
ii. ≥6 mos	1	1	1	
b. Moderately or severely impaired cardiac function (New York Heart Association Functional Class III or IV: patients with marked limitation of activity or patients who should be at complete rest) (*1*)	1	1	1	
Rheumatic Diseases				
Systemic lupus erythematosus§				
a. Positive (or unknown) antiphospholipid antibodies	1	1	1	
b. Severe thrombocytopenia	1	1	1	
c. Immunosuppressive treatment	1	1	1	
d. None of the above	1	1	1	
Rheumatoid arthritis				
a. On immunosuppressive therapy	1	1	1	
b. Not on immunosuppressive therapy	1	1	1	
Neurologic Conditions				
Headaches				
a. Non-migrainous (mild or severe)	1	1	1	
b. Migraine				
i. Without aura				
• Age <35 yrs	1	1	1	
• Age ≥35 yrs	1	1	1	
ii. With aura, at any age	1	1	1	
Epilepsy§	1	1	1	
Depressive Disorders				
Depressive disorders	1	1	1	
Reproductive Tract Infections and Disorders				
Unexplained vaginal bleeding (suspicious for serious condition) Before evaluation	1	1	1	**Clarification:** If pregnancy or an underlying pathological condition (such as pelvic malignancy) is suspected, it must be evaluated and the category adjusted after evaluation.
Endometriosis	1	1	1	
Benign ovarian tumors (including cysts)	1	1	1	
Severe dysmenorrhea	1	1	1	

TABLE. (*Continued*) Classifications for barrier methods,*† including condoms, spermicides, and diaphragms/caps

Condition	Category: Condom	Category: Spermicide	Category: Diaphragm/cap	Clarifications/Evidence/Comments
Gestational trophoblastic disease				
a. Decreasing or undetectable β–hCG levels	1	1	1	
b. Persistently elevated β-hCG levels or malignant disease§	1	1	1	
Cervical ectropion	1	1	1	
Cervical intraepithelial neoplasia	1	1	1	**Clarification: The cap should not be used.** Diaphragm use has no restrictions.
Cervical cancer (awaiting treatment)	1	2	1	**Clarification: The cap should not be used.** Diaphragm use has no restrictions. **Comment:** Repeated and high-dose use of nonoxynol-9 can cause vaginal and cervical irritation or abrasions.
Breast disease				
a. Undiagnosed mass	1	1	1	
b. Benign breast disease	1	1	1	
c. Family history of cancer	1	1	1	
d. Breast cancer§				
i. Current	1	1	1	
ii. Past and no evidence of current disease for 5 yrs	1	1	1	
Endometrial hyperplasia	1	1	1	
Endometrial cancer§	1	1	1	
Ovarian cancer§	1	1	1	
Uterine fibroids	1	1	1	
Anatomical abnormalities	1	1	Not applicable	**Clarification:** The diaphragm cannot be used in certain cases of prolapse. Cap use is not appropriate for a woman with markedly distorted cervical anatomy.
Pelvic inflammatory disease (PID)				
a. Past PID (assuming no current risk factors of STIs)				
i. With subsequent pregnancy	1	1	1	
ii. Without subsequent pregnancy	1	1	1	
b. Current PID	1	1	1	
STIs				
a. Current purulent cervicitis or chlamydial infection or gonorrhea	1	1	1	
b. Other STIs (excluding HIV and hepatitis)	1	1	1	
c. Vaginitis (including *Trichomonas vaginalis* and bacterial vaginosis)	1	1	1	
d. Increased risk for STIs	1	1	1	
HIV/AIDS				
High risk for HIV	1	4	4	**Evidence:** Repeated and high-dose use of the spermicide nonoxynol-9 was associated with increased risk for genital lesions, which might increase the risk for HIV infection (*2*). **Comment:** Diaphragm use is assigned Category 4 because of concerns about the spermicide, not the diaphragm.
HIV infection§	1	3	3	**Comment:** Use of spermicides and/or diaphragms (with spermicide) can disrupt the cervical mucosa, which may increase viral shedding and HIV transmission to uninfected sex partners.
AIDS§	1	3	3	**Comment:** Use of spermicides and/or diaphragms (with spermicide) can disrupt the cervical mucosa, which may increase viral shedding and HIV transmission to uninfected sex partners
Other Infections				
Schistosomiasis				
a. Uncomplicated	1	1	1	
b. Fibrosis of liver§	1	1	1	
Tuberculosis§				
a. Nonpelvic	1	1	1	
b. Pelvic	1	1	1	

TABLE. (*Continued*) Classifications for barrier methods,*† including condoms, spermicides, and diaphragms/caps

Condition	Category			Clarifications/Evidence/Comments
	Condom	Spermicide	Diaphragm/ cap	
Malaria	1	1	1	
History of toxic shock syndrome	1	1	3	**Comment:** Toxic shock syndrome has been reported in association with contraceptive sponge and diaphragm use.
Urinary tract infection	1	1	2	**Comment:** Use of diaphragms and spermicides might increase risk for urinary tract infection.
Endocrine Conditions				
Diabetes				
a. History of gestational disease	1	1	1	
b. Nonvascular disease				
i. Noninsulin-dependent	1	1	1	
ii. Insulin-dependent§	1	1	1	
c. Nephropathy/retinopathy/neuropathy§	1	1	1	
d. Other vascular disease or diabetes of >20 yrs' duration§	1	1	1	
Thyroid disorders				
a. Simple goiter	1	1	1	
b. Hyperthyroid	1	1	1	
c. Hypothyroid	1	1	1	
Gastrointestinal Conditions				
Inflammatory bowel disease (ulcerative colitis, Crohn disease)	1	1	1	
Gallbladder disease				
a. Symptomatic				
i. Treated by cholecystectomy	1	1	1	
ii. Medically treated	1	1	1	
iii. Current	1	1	1	
b. Asymptomatic	1	1	1	
History of cholestasis				
a. Pregnancy-related	1	1	1	
b. Past COC-related	1	1	1	
Viral hepatitis				
a. Acute or flare	1	1	1	
b. Carrier	1	1	1	
c. Chronic	1	1	1	
Cirrhosis				
a. Mild (compensated)	1	1	1	
b. Severe§ (decompensated)	1	1	1	
Liver tumors				
a. Benign				
i. Focal nodular hyperplasia	1	1	1	
ii. Hepatocellular adenoma§	1	1	1	
b. Malignant§ (hepatoma)	1	1	1	
Anemias				
Thalassemia	1	1	1	
Sickle cell disease§	1	1	1	
Iron deficiency anemia	1	1	1	
Solid Organ Transplantation				
Solid organ transplantation§				
a. Complicated: graft failure (acute or chronic), rejection, cardiac allograft vasculopathy	1	1	1	
b. Uncomplicated	1	1	1	

TABLE. (*Continued*) Classifications for barrier methods,*† including condoms, spermicides, and diaphragms/caps

Condition	Category: Condom	Category: Spermicide	Category: Diaphragm/cap	Clarifications/Evidence/Comments
Drug Interactions				
Antiretroviral (ARV) therapy				**Clarification:** No drug interaction between ARV therapy and barrier method use is known. However, HIV infection and AIDS are classified as Category 3 for spermicides and diaphragms (see HIV/AIDS condition above).
a. Nucleoside reverse transcriptase inhibitors (NRTIs)	1	3	3	
b. Non-nucleoside reverse transcriptase inhibitors (NNRTIs)	1	3	3	
c. Ritonavir-boosted protease inhibitors	1	3	3	
Anticonvulsant therapy				
a. Certain anticonvulsants (phenytoin, carbamazepine, barbiturates, primidone, topiramate, oxcarbazepine)	1	1	1	
b. Lamotrigine	1	1	1	
Antimicrobial therapy				
a. Broad-spectrum antibiotics	1	1	1	
b. Antifungals	1	1	1	
c. Antiparasitics	1	1	1	
d. Rifampicin or rifabutin therapy	1	1	1	
Allergy to latex	3	1	3	**Clarification:** The condition of allergy to latex does not apply to plastic condoms/diaphragms.

* Abbreviations: STI = sexually transmitted infection; HIV = human immunodeficiency virus; BMI, body mass index; DVT = deep venous thrombosis; PE = pulmonary embolism; ARV = antiretroviral; hCG = human chorionic gonadotropin; PID = pelvic inflammatory disease; AIDS = acquired immunodeficiency syndrome; COC = combined oral contraceptive; NRTI = nucleoside reverse transcriptase inhibitor; NNRTI = non-nucleoside reverse transcriptase inhibitor.

† If risk exists for STI/HIV (including during pregnancy or postpartum), the correct and consistent use of condoms is recommended, either alone or with another contraceptive method. Consistent and correct use of the male latex condom reduces the risk for STIs and HIV transmission. Women with conditions that make pregnancy an unacceptable risk should be advised that barrier methods for pregnancy prevention may not be appropriate for those who cannot use them consistently and correctly because of the relatively higher typical-use failure rates of these methods.

§ Condition that exposes a woman to increased risk as a result of unintended pregnancy.

References

1. The Criteria Committee of the New York Heart Association. Nomenclature and criteria for diagnosis of diseases of the heart and great vessels. 9th ed. Boston, MA: Little, Brown & Co; 1994.

2. Wilkinson D, Ramjee G, Tholandi M, Rutherford G. Nonoxynol-9 for preventing vaginal acquisition of HIV infection by women from men. Cochrane Database Syst Rev 2002;4:CD003939.

Appendix H
Classifications for Fertility Awareness–Based Methods

Fertility awareness–based (FAB) methods of family planning involve identifying the fertile days of the menstrual cycle, whether by observing fertility signs such as cervical secretions and basal body temperature or by monitoring cycle days (Box). FAB methods can be used in combination with abstinence or barrier methods during the fertile time. If barrier methods are used, refer to Appendix G.

No medical conditions become worse because of use of FAB methods. In general, FAB methods can be used without concern for health effects to persons who choose them. However, a number of conditions make their use more complex. The existence of these conditions suggests that 1) use of these methods should be delayed until the condition is corrected or resolved or 2) persons using FAB methods will require special counseling, and a more highly trained provider is generally necessary to ensure correct use.

Women with conditions that make pregnancy an unacceptable risk should be advised that FAB methods might not be appropriate for them because of the relatively higher typical-use failure rates of these methods. FAB methods do not protect against sexually transmitted infections (STIs) or human immunodeficiency virus (HIV).

Box. Definitions for terms associated with fertility awareness–based methods

- **Symptoms-based methods**: FAB methods based on observation of fertility signs (e.g., cervical secretions, basal body temperature) such as the Cervical Mucus Method, the Symptothermal Method, and the TwoDay Method.
- **Calendar-based methods**: FAB methods based on calendar calculations such as the Calendar Rhythm Method and the Standard Days Method.
- **Acccept (A)**: There is no medical reason to deny the particular FAB method to a woman in this circumstance.
- **Caution (C)**: The method is normally provided in a routine setting but with extra preparation and precautions. For FAB methods, this usually means that special counselling might be needed to ensure correct use of the method by a woman in this circumstance.
- **Delay (D)**: Use of this method should be delayed until the condition is evaluated or corrected. Alternative temporary methods of contraception should be offered.

TABLE. Fertility awareness–based methods,*† including symptoms-based and calendar-based methods

Condition	Category: Symptom-based method	Category: Calendar-based method	Clarifications/Evidence/Comments
Personal Characteristics and Reproductive History			
Pregnancy	Not applicable		**Clarification:** FAB methods are not relevant during pregnancy.
Life stage			**Clarification:** Menstrual irregularities are common in postmenarche and perimenopause and might complicate the use of FAB methods.
a. Postmenarche	C	C	
b. Perimenopause	C	C	
Breastfeeding			**Comment:** Use of FAB methods when breastfeeding might be less effective than when not breastfeeding.
a. <6 wks postpartum	D	D	**Comment:** Women who are primarily breastfeeding and are amenorrheic are unlikely to have sufficient ovarian function to produce detectable fertility signs and hormonal changes during the first 6 months postpartum. However, the likelihood of resumption of fertility increases with time postpartum and with substitution of breast milk with other foods.
b. ≥6 wks	C	D	
c. After menses begin	C	C	**Comment:** When the woman notices fertility signs, particularly cervical secretions, she can use a symptoms-based method. First postpartum menstrual cycles in breastfeeding women vary significantly in length. Return to regularity takes several cycles. When she has had at least 3 postpartum menses and her cycles are regular again, she can use a calendar-based method. When she has had at least 4 postpartum menses and her most recent cycle lasted 26–32 days, she can use the Standard Days Method. Before that time, a barrier method should be offered if the woman plans to use a FAB method later.

TABLE. (*Continued*) Fertility awareness–based methods,*† including symptoms-based and calendar-based methods

Condition	Category: Symptom-based method	Category: Calendar-based method	Clarifications/Evidence/Comments
Postpartum (in nonbreastfeeding women)			
a. <4 wks	D	D	**Comment:** Nonbreastfeeding women are not likely to have sufficient ovarian function to either require a FAB method or to have detectable fertility signs or hormonal changes before 4 weeks postpartum. Although the risk for pregnancy is low, a method appropriate for the postpartum period should be offered.
b. ≥4 wks	A	D	**Comment:** Nonbreastfeeding women are likely to have sufficient ovarian function to produce detectable fertility signs and/or hormonal changes at this time; likelihood increases rapidly with time postpartum. Women can use calendar-based methods as soon as they have completed three postpartum menses. Methods appropriate for the postpartum period should be offered before that time.
Postabortion	C	D	**Comment:** Postabortion women are likely to have sufficient ovarian function to produce detectable fertility signs and/or hormonal changes; likelihood increases with time postabortion. Women can start using calendar-based methods after they have had at least 1 postabortion menses (e.g., women who before this pregnancy had most cycles of 26–32 days can then use the Standard Days Method). Methods appropriate for the postabortion period should be offered before that time.
Reproductive Tract Infections and Disorders			
Irregular vaginal bleeding	D	D	**Comment:** Presence of this condition makes FAB methods unreliable. Therefore, barrier methods should be recommended until the bleeding pattern is compatible with proper method use. The condition should be evaluated and treated as necessary.
Vaginal discharge	D	A	**Comment:** Because vaginal discharge makes recognition of cervical secretions difficult, the condition should be evaluated and treated if needed before providing methods based on cervical secretions.
Other			
Use of drugs that affect cycle regularity, hormones, and/or fertility signs	C/D	C/D	**Comment:** Use of certain mood-altering drugs such as lithium, tricyclic antidepressants, and antianxiety therapies, and certain antibiotics and anti-inflammatory drugs, might alter cycle regularity or affect fertility signs. The condition should be carefully evaluated and a barrier method offered until the degree of effect has been determined or the drug is no longer being used.
Diseases that elevate body temperature			
a. Chronic diseases	C	A	**Comment:** Elevated temperature levels might make basal body temperature difficult to interpret but have no effect on cervical secretions. Thus, use of a method that relies on temperature should be delayed until the acute febrile disease abates. Temperature-based methods are not appropriate for women with chronically elevated temperatures. In addition, some chronic diseases interfere with cycle regularity, making calendar-based methods difficult to interpret.
b. Acute diseases	D	A	

* Abbreviations: FAB = fertility awareness–based; A = accept; C = caution; D = delay; STI = sexually transmitted infection; HIV = human immunodeficiency infection.
† Fertility awareness–based methods do not protect against STI/HIV. If risk exists for STI/HIV (including during pregnancy or postpartum), the correct and consistent use of condoms is recommended, either alone or with another contraceptive method. Consistent and correct use of the male latex condom reduces the risk for STIs and HIV transmission.

Appendix I
Lactational Amenorrhea Method

The Bellagio Consensus provided the scientific basis for defining the conditions under which breastfeeding can be used safely and effectively for birth-spacing purposes, and programmatic guidelines were developed for use of lactational amenorrhea in family planning (*1,2*). These guidelines include the following three criteria, all of which must be met to ensure adequate protection from an unplanned pregnancy: 1) amenorrhea; 2) fully or nearly fully breastfeeding, and 3) <6 months postpartum.

The main indications for breastfeeding are to provide an ideal food for the infant and protect against disease. No medical conditions exist for which use of the lactational amenorrhea method for contraception is restricted. However, breastfeeding might not be recommended for women or infants with certain conditions.

Women with conditions that make pregnancy an unacceptable risk should be advised that the lactational amenorrhea method might not be appropriate for them because of its relatively higher typical-use failure rates. The lactational amenorrhea method does not protect against sexually transmitted infections (STIs) and human immunodeficiency virus (HIV). If risk exists for STI/HIV (including during pregnancy or postpartum), the correct and consistent use of condoms is recommended, either alone or with another contraceptive method. Consistent and correct use of the male latex condom reduces the risk for STIs and HIV transmission.

HIV Infection

HIV can be transmitted from mother to infant through breastfeeding. Therefore, in the United States, where replacement feeding is affordable, feasible, acceptable, sustainable, and safe, breastfeeding for women with HIV is not recommended (*3,4*).

Other Medical Conditions

The American Academy of Pediatrics also recommends against breastfeeding for women with active untreated tuberculosis disease, who are positive for human T-cell lymphotropic virus types I or II, or who have herpes simplex lesions on a breast (infant can feed from the other breast). In addition, infants with classic galactosemia should not breastfeed (*4*).

Medication Used during Breastfeeding

To protect infant health, the American Academy of Pediatrics does not recommend breastfeeding for women receiving certain drugs, including diagnostic or therapeutic radioactive isotopes or exposure to radioactive materials, antimetabolites or chemotherapeutic agents, and current use of drugs of abuse (*4*).

References

1. Kennedy KI, Rivera R, McNeilly AS. Consensus statement on the use of breastfeeding as a family planning method. Contraception 1989;39:477–96.
2. Labbok M, Cooney K, Coly S. Guidelines: breastfeeding, family planning, and the Lactational Amenorrhea Method-LAM. Washington, DC: Institute for Reproductive Health; 1994.
3. Perinatal HIV Guidelines Working Group. Public Health Service Task Force recommendations for use of antiretroviral drugs in pregnant HIV-infected women for maternal health and interventions to reduce perinatal HIV transmission in the United States. Rockville, MD: Public Health Service Task Force; 2009.
4. Gartner LM, Morton J, Lawrence RA, et al. Breastfeeding and the use of human milk. Pediatrics 2005;115:496–506.

Appendix J

Coitus Interruptus (Withdrawal)

Coitus interruptus (CI), also known as withdrawal, is a traditional family planning method in which the man completely removes his penis from the vagina, and away from the external genitalia of the female partner, before he ejaculates. CI prevents sperm from entering the woman's vagina, thereby preventing contact between spermatozoa and the ovum.

This method might be appropriate for couples

- who are highly motivated and able to use this method effectively;
- with religious or philosophical reasons for not using other methods of contraception;
- who need contraception immediately and have entered into a sexual act without alternative methods available;
- who need a temporary method while awaiting the start of another method; or
- who have intercourse infrequently.

Some benefits of CI are that the method, if used correctly, does not affect breastfeeding and is always available for primary use or use as a back-up method. In addition, CI involves no economic cost or use of chemicals. CI has no directly associated health risks. CI does not protect against sexually transmitted infections (STIs) and human immunodeficiency virus (HIV). If risk exists for STI/HIV (including during pregnancy or postpartum), the correct and consistent use of condoms is recommended, either alone or with another contraceptive method. Consistent and correct use of the male latex condom reduces the risk for STIs and HIV transmission.

CI is unforgiving of incorrect use, and its effectiveness depends on the willingness and ability of the couple to use withdrawal with every act of intercourse. Women with conditions that make pregnancy an unacceptable risk should be advised that CI might not be appropriate for them because of its relatively higher typical-use failure rates.

Appendix K
Female and Male Sterilization

Tubal sterilization for women and vasectomy for men are permanent, safe, and highly effective methods of contraception. In general, no medical conditions would absolutely restrict a person's eligibility for sterilization (with the exception of known allergy or hypersensitivity to any materials used to complete the sterilization method). However, certain conditions place a woman at high surgical risk; in these cases, careful consideration should be given to the risks and benefits of other acceptable alternatives, including long-acting, highly effective, reversible methods and vasectomy. Female and male sterilization do not protect against sexually transmitted infections (STIs) or human immunodeficiency virus (HIV). If risk exists for STI/HIV (including during pregnancy or postpartum), the correct and consistent use of condoms is recommended, either alone or with another contraceptive method. Consistent and correct use of the male latex condom reduces the risk for STIs and HIV transmission.

Because these methods are intended to be irreversible, persons who choose sterilization should be certain that they want to prevent pregnancy permanently. Most persons who choose sterilization remain satisfied with their decision. However, a small proportion of women regret this decision (1%–26% from different studies, with higher rates of regret reported by women who were younger at sterilization) (*1,2*). Regret among men about vasectomy has been reported to be approximately 5% (*3*), similar to the proportion of women who report regretting their husbands' vasectomy (6%) (*4*). Therefore, all persons should be appropriately counseled about the permanency of sterilization and the availability of highly effective, reversible methods of contraception.

References

1. Peterson HB. Sterilization. Obstet Gynecol 2008;111:189–203.
2. Hillis SD, Marchbanks PA, Tylor LR, Peterson HB. Poststerilization regret: findings from the United States Collaborative Review of Sterilization. Obstet Gynecol 1999;93:889–95.
3. Ehn BE, Liljestrand J. A long-term follow-up of 108 vasectomized men. Good counselling routines are important. Scand J Urol Nephrol 1995;29:477–81.
4. Jamieson DJ, Kaufman SC, Costello C, et al. A comparison of women's regret after vasectomy versus tubal sterilization. Obstet Gynecol 2002;99:1073–9.

Appendix L

Summary of Classifications for Hormonal Contraceptive Methods and Intrauterine Devices

Health-care providers can use the summary table as a quick reference guide to the classifications for hormonal contraceptive methods and intrauterine contraception and to compare classifications across these methods. See the full appendix for each method for clarifications to the numeric categories, as well as for summaries of the evidence and additional comments.

BOX. Categories for Classifying Hormonal Contraceptives and IUDs

1 = A condition for which there is no restriction for the use of the contraceptive method.
2 = A condition for which the advantages of using the method generally outweigh the theoretical or proven risks.
3 = A condition for which the theoretical or proven risks usually outweigh the advantages of using the method.
4 = A condition that represents an unacceptable health risk if the contraceptive method is used.

TABLE. Summary of classifications for hormonal contraceptive methods and intrauterine devices*

Condition	COC/P/R	POP	DMPA	Implants	LNG-IUD	Cu-IUD
Personal Characteristics and Reproductive History						
Pregnancy	Not applicable†	Not applicable†	Not applicable†	Not applicable†	4†	4†
Age	Menarche to <40 yrs = 1 ≥40 yrs = 2	Menarche to <18 yrs = 1 18–45 yrs = 1 >45 yrs = 1	Menarche to <18 yrs = 2 18–45 yrs = 1 >45 yrs = 1	Menarche to <18 yrs =1 18–45 yrs = 1 >45 yrs = 1	Menarche to <20 yrs = 2 ≥20 yrs = 1	Menarche to <20 yrs = 2 ≥20 yrs = 1
Parity						
a. Nulliparous	1	1	1	1	2	2
b. Parous	1	1	1	1	1	1
Breastfeeding						
a. <1 mo postpartum	3†	2†	2†	2†		
b. 1 mo to <6 mos	2†	1†	1†	1†		
c. ≥6 mos postpartum	2†	1†	1†	1†		
Postpartum (nonbreastfeeding women)						
a. <21 days	3	1	1	1		
b. ≥21 days	1	1	1	1		
Postpartum (breastfeeding or nonbreastfeeding women, including post-Cesarean section)						
a. <10 min after delivery of the placenta					2	1
b. 10 min after delivery of the placenta to <4 wks					2	2
c. ≥4 wks					1	1
d. Puerperal sepsis					4	4
Postabortion						
a. First trimester	1†	1†	1†	1†	1†	1†
b. Second trimester	1†	1†	1†	1†	2	2
c. Immediate postseptic abortion	1†	1†	1†	1†	4	4
Past ectopic pregnancy	1	2	1	1	1	1
History of pelvic surgery (see postpartum, including Cesarean section)	1	1	1	1	1	1
Smoking						
a. Age <35 yrs	2	1	1	1	1	1
b. Age ≥35 yrs						
i. <15 Cigarettes/day	3	1	1	1	1	1
ii. ≥15 Cigarettes/day	4	1	1	1	1	1

TABLE. (*Continued*) Summary of classifications for hormonal contraceptive methods and intrauterine devices*

Condition	COC/P/R	POP	DMPA	Implants	LNG-IUD	Cu-IUD
Obesity						
a. ≥30 kg/m² BMI	2	1	1	1	1	1
b. Menarche to <18 yrs and ≥30 kg/m² BMI	2	1	2	1	1	1
History of bariatric surgery§						
a. Restrictive procedures: decrease storage capacity of the stomach (vertical banded gastroplasty, laparoscopic adjustable gastric band, laparoscopic sleeve gastrectomy)	1	1	1	1	1	1
b. Malabsorptive procedures: decrease absorption of nutrients and calories by shortening the functional length of the small intestine (Roux-en-Y gastric bypass, biliopancreatic diversion)	COCs: 3 P/R: 1	3	1	1	1	1
Cardiovascular Disease						
Multiple risk factors for arterial cardiovascular disease (such as older age, smoking, diabetes, and hypertension)	3/4†	2†	3†	2†	2	1
Hypertension						
a. Adequately controlled hypertension	3†	1†	2†	1†	1	1
b. Elevated blood pressure levels (properly taken measurements)						
i. Systolic 140–159 mm Hg or diastolic 90–99 mm Hg	3	1	2	1	1	1
ii. Systolic ≥160 mm Hg or diastolic ≥100 mm Hg§	4	2	3	2	2	1
c. Vascular disease	4	2	3	2	2	1
History of high blood pressure during pregnancy (where current blood pressure is measurable and normal)	2	1	1	1	1	1
Deep venous thrombosis (DVT)/ pulmonary embolism (PE)						
a. History of DVT/PE, not on anticoagulant therapy						
i. Higher risk for recurrent DVT/PE (≥1 risk factors) • History of estrogen-associated DVT/PE • Pregnancy-associated DVT/PE • Idiopathic DVT/PE • Known thrombophilia, including antiphospholipid syndrome • Active cancer (metastatic, on therapy, or within 6 mos after clinical remission), excluding non-melanoma skin cancer • History of recurrent DVT/PE	4	2	2	2	2	1
ii. Lower risk for recurrent DVT/PE (no risk factors)	3	2	2	2	2	1
b. Acute DVT/PE	4	2	2	2	2	2
c. DVT/PE and established on anticoagulant therapy for at least 3 mos						
i. Higher risk for recurrent DVT/PE (≥1 risk factors) • Known thrombophilia, including antiphospholipid syndrome • Active cancer (metastatic, on therapy, or within 6 mos after clinical remission), excluding non-melanoma skin cancer • History of recurrent DVT/PE	4†	2	2	2	2	2
ii. Lower risk for recurrent DVT/PE (no risk factors)	3†	2	2	2	2	2

TABLE. (*Continued*) Summary of classifications for hormonal contraceptive methods and intrauterine devices*

Condition	COC/P/R	POP	DMPA	Implants	LNG-IUD	Cu-IUD
d. Family history (first-degree relatives)	2	1	1	1	1	1
e. Major surgery						
i. With prolonged immobilization	4	2	2	2	2	1
ii. Without prolonged immobilization	2	1	1	1	1	1
f. Minor surgery without immobilization	1	1	1	1	1	1
Known thrombogenic mutations§ (e.g. factor V Leiden; prothrombin mutation; protein S, protein C, and antithrombin deficiencies)	4†	2†	2†	2†	2†	1†
Superficial venous thrombosis						
a. Varicose veins	1	1	1	1	1	1
b. Superficial thrombophlebitis	2	1	1	1	1	1
Current and history of ischemic heart disease§		Initiation Continuation		Initiation Continuation	Initiation Continuation	
	4	2 3	3	2 3	2 3	1
Stroke§ (history of cerebrovascular accident)		Initiation Continuation		Initiation Continuation		
	4	2 3	3	2 3	2	1
Known hyperlipidemias	2/3†	2†	2†	2†	2†	1†
Valvular heart disease						
a. Uncomplicated	2	1	1	1	1	1
b. Complicated§ (pulmonary hypertension, risk for atrial fibrillation, history of subacute bacterial endocarditis)	4	1	1	1	1	1
Peripartum cardiomyopathy§						
a. Normal or mildly impaired cardiac function (New York Heart Association Functional Class I or II: patients with no limitation of activities or patients with slight, mild limitation of activity) (*1*)						
i. <6 mos	4	1	1	1	2	2
ii. ≥6 mos	3	1	1	1	2	2
b. Moderately or severely impaired cardiac function (New York Heart Association Functional Class III or IV: patients with marked limitation of activity or patients who should be at complete rest) (*1*)	4	2	2	2	2	2
Rheumatic Diseases						
Systemic lupus erythematosus§			Initiation Continuation			Initiation Continuation
a. Positive (or unknown) antiphospholipid antibodies	4	3	3 3	3	3	1 1
b. Severe thrombocytopenia	2	2	3 2	2	2†	3† 2†
c. Immunosuppressive treatment	2	2	2 2	2	2	2 1
d. None of the above	2	2	2 2	2	2	1 1
Rheumatoid arthritis					Initiation Continuation	Initiation Continuation
a. On immunosuppressive therapy	2	1	2/3†	1	2 1	2 1
b. Not on immunosuppressive therapy	2	1	2	1	1	1
Neurologic Conditions						
Headaches	Initiation Continuation	Initiation Continuation	Initiation Continuation	Initiation Continuation	Initiation Continuation	
a. Non-migrainous (mild or severe)	1† 2†	1† 1†	1† 1†	1† 1†	1† 1†	1†
b. Migraine						
i. Without aura						
• Age <35 yrs	2† 3†	1† 2†	2† 2†	2† 2†	2† 2†	1†
• Age ≥35 yrs	3† 4†	1† 2†	2† 2†	2† 2†	2† 2†	1†
ii. With aura (at any age)	4† 4†	2† 3†	2† 3†	2† 3†	2† 3†	1†
Epilepsy§	1†	1†	1†	1†	1	1

If on treatment, see Drug Interactions section below

TABLE. (*Continued*) Summary of classifications for hormonal contraceptive methods and intrauterine devices*

Condition	COC/P/R	POP	DMPA	Implants	LNG-IUD		Cu-IUD	
Depressive Disorders								
Depressive disorders	1†	1†	1†	1†	1†		1†	
Reproductive Tract Infections and Disorders								
Vaginal bleeding patterns					Initiation	Continuation		
a. Irregular pattern without heavy bleeding	1	2	2	2	1	1	1	
b. Heavy or prolonged bleeding (includes regular and irregular patterns)	1†	2†	2†	2†	1†	2†	2†	
Unexplained vaginal bleeding (suspicious for serious condition)					Initiation	Continuation	Initiation	Continuation
Before evaluation	2†	2†	3†	3†	4†	2†	4†	2†
Endometriosis	1	1	1	1	1		2	
Benign ovarian tumors (including cysts)	1	1	1	1	1		1	
Severe dysmenorrhea	1	1	1	1	1		2	
Gestational trophoblastic disease								
a. Decreasing or undetectable ß-hCG levels	1	1	1	1	3		3	
b. Persistently elevated ß-hCG levels or malignant disease§	1	1	1	1	4		4	
Cervical ectropion	1	1	1	1	1		1	
Cervical intraepithelial neoplasia	2	1	2	2	2		1	
Cervical cancer (awaiting treatment)					Initiation	Continuation	Initiation	Continuation
	2	1	2	2	4	2	4	2
Breast disease								
a. Undiagnosed mass	2†	2†	2†	2†	2		1	
b. Benign breast disease	1	1	1	1	1		1	
c. Family history of cancer	1	1	1	1	1		1	
d. Breast cancer§								
i. Current	4	4	4	4	4		1	
ii. Past and no evidence of current disease for 5 yrs	3	3	3	3	3		1	
Endometrial hyperplasia	1	1	1	1	1		1	
Endometrial cancer§					Initiation	Continuation	Initiation	Continuation
	1	1	1	1	4	2	4	2
Ovarian cancer§	1	1	1	1	1		1	
Uterine fibroids	1	1	1	1	2		2	
Anatomical abnormalities								
a. Distorted uterine cavity (any congenital or acquired uterine abnormality distorting the uterine cavity in a manner that is incompatible with IUD insertion)					4		4	
b. Other abnormalities (including cervical stenosis or cervical lacerations) not distorting the uterine cavity or interfering with IUD insertion					2		2	
Pelvic inflammatory disease (PID)								
a. Past PID (assuming no current risk factors of STIs)					Initiation	Continuation	Initiation	Continuation
i. With subsequent pregnancy	1	1	1	1	1	1	1	1
ii. Without subsequent pregnancy	1	1	1	1	2	2	2	2
b. Current PID	1	1	1	1	4	2†	4	2†

TABLE. (*Continued*) Summary of classifications for hormonal contraceptive methods and intrauterine devices*

Condition	COC/P/R	POP	DMPA	Implants	LNG-IUD		Cu-IUD	
STIs					Initiation	Continuation	Initiation	Continuation
a. Current purulent cervicitis or chlamydial infection or gonorrhea	1	1	1	1	4	2†	4	2†
b. Other STIs (excluding HIV and hepatitis)	1	1	1	1	2	2	2	2
c. Vaginitis (including *Trichomonas vaginalis* and bacterial vaginosis)	1	1	1	1	2	2	2	2
d. Increased risk for STIs	1	1	1	1	2/3†	2	2/3†	2
HIV/AIDS					Initiation	Continuation	Initiation	Continuation
High risk for HIV	1	1	1	1	2	2	2	2
HIV infection§	1	1	1	1	2	2	2	2
AIDS§	1†	1†	1†	1†	3	2†	3	2†
Clinically well on ARV therapy	If on treatment, see Drug Interactions section below				2	2	2	2
Other Infections								
Schistosomiasis								
a. Uncomplicated	1	1	1	1	1		1	
b. Fibrosis of the liver (if severe, see Cirrhosis)§	1	1	1	1	1		1	
Tuberculosis§					Initiation	Continuation	Initiation	Continuation
a. Nonpelvic	1†	1†	1†	1†	1	1	1	1
b. Pelvic	1†	1†	1†	1†	4	3	4	3
	If on treatment, see Drug Interactions section below							
Malaria	1	1	1	1	1		1	
Endocrine Conditions								
Diabetes								
a. History of gestational disease	1	1	1	1	1		1	
b. Nonvascular disease								
i. Noninsulin-dependent	2	2	2	2	2		1	
ii. Insulin-dependent§	2	2	2	2	2		1	
c. Nephropathy/retinopathy/neuropathy§	3/4†	2	3	2	2		1	
d. Other vascular disease or diabetes of >20 yrs' duration§	3/4†	2	3	2	2		1	
Thyroid disorders								
a. Simple goiter	1	1	1	1	1		1	
b. Hyperthyroid	1	1	1	1	1		1	
c. Hypothyroid	1	1	1	1	1		1	
Gastrointestinal Conditions								
Inflammatory bowel disease (IBD) (ulcerative colitis, Crohn disease)	2/3†	2	2	1	1		1	
Gallbladder disease								
a. Symptomatic								
i. Treated by cholecystectomy	2	2	2	2	2		1	
ii. Medically treated	3	2	2	2	2		1	
iii. Current	3	2	2	2	2		1	
b. Asymptomatic	2	2	2	2	2		1	
History of cholestasis								
a. Pregnancy-related	2	1	1	1	1		1	
b. Past COC-related	3	2	2	2	2		1	
Viral hepatitis	Initiation	Continuation						
a. Acute or flare	3/4†	2	1	1	1		1	
b. Carrier	1	1	1	1	1		1	
c. Chronic	1	1	1	1	1		1	
Cirrhosis								
a. Mild (compensated)	1		1	1	1		1	
b. Severe§ (decompensated)	4		3	3	3		1	

TABLE. (*Continued*) Summary of classifications for hormonal contraceptive methods and intrauterine devices*

Condition	COC/P/R	POP	DMPA	Implants	LNG-IUD		Cu-IUD	
Liver tumors								
a. Benign								
i. Focal nodular hyperplasia	2	2	2	2	2		1	
ii. Hepatocellular adenoma§	4	3	3	3	3		1	
b. Malignant§ (hepatoma)	4	3	3	3	3		1	
Anemias								
Thalassemia	1	1	1	1	1		2	
Sickle cell disease§	2	1	1	1	1		2	
Iron-deficiency anemia	1	1	1	1	1		2	
Solid Organ Transplantation								
Solid organ transplantation§					Initiation	Continuation	Initiation	Continuation
a. Complicated: graft failure (acute or chronic), rejection, cardiac allograft vasculopathy	4	2	2	2	3	2	3	2
b. Uncomplicated	2†	2	2	2	2		2	
Drug Interactions								
Antiretroviral therapy (see appendix M)					Initiation	Continuation	Initiation	Continuation
a. Nucleoside reverse transcriptase inhibitors (NRTIs)	1†	1	1	1	2/3†	2†	2/3†	2†
b. Non-nucleoside reverse transcriptase inhibitors (NNRTIs)	2†	2†	1	2†	2/3†	2†	2/3†	2†
c. Ritonavir-boosted protease inhibitors	3†	3†	1	2†	2/3†	2†	2/3†	2†
Anticonvulsant therapy								
a. Certain anticonvulsants (phenytoin, carbamazepine, barbiturates, primidone, topiramate, oxcarbazepine)	3†	3†	1	2†	1		1	
b. Lamotrigine	3†	1	1	1	1		1	
Antimicrobial therapy								
a. Broad-spectrum antibiotics	1	1	1	1	1		1	
b. Antifungals	1	1	1	1	1		1	
c. Antiparasitics	1	1	1	1	1		1	
d. Rifampicin or rifabutin therapy	3†	3†	1	2†	1		1	

* Abbreviations: COC = combined oral contraceptive; P = combined hormonal contraceptive patch; R = combined hormonal vaginal ring; POP = progestin-only pill; DMPA = depot medroxyprogesterone acetate; IUD = intrauterine device; LNG-IUD = levonorgestrel-releasing IUD; Cu-IUD = copper IUD; BMI = body mass index; DVT = deep venous thrombosis; PE = pulmonary embolism; hCG, = human chorionic gonadotropin; PID = pelvic inflammatory disease; STI = sexually transmitted infection; HIV = human immunodeficiency virus; AIDS = acquired immunodeficiency syndrome; NRTI = nucleoside reverse transcriptase inhibitor; NNRTI = non-nucleoside reverse transcriptase.
† Consult the appendix for this contraceptive method for a clarification to this classification.
§ Condition that exposes a woman to increased risk as a result of unintended pregnancy.

Reference

1. The Criteria Committee of the New York Heart Association. Nomenclature and criteria for diagnosis of diseases of the heart and great vessels. 9th ed. Boston, MA: Little, Brown & Co.; 1994.

Appendix M

Summary of Evidence Regarding Potential Drug Interactions between Hormonal Contraception and Antiretroviral Therapies

Limited data from small, mostly unpublished studies suggest that some antiretroviral (ARV) therapies might alter the pharmacokinetics of combined oral contraceptives (COCs). Few studies have measured clinical outcomes. However, contraceptive steroid levels in the blood decrease substantially with ritonavir-boosted protease inhibitors. Such decreases have the potential to compromise contraceptive effectiveness. Some of the interactions between contraceptives and ARVs also have led to increased ARV toxicity. For smaller effects that occur with non-nucleoside reverse transcriptase inhibitors, clinical significance is unknown, especially because studies have not examined steady-state levels of contraceptive hormones. No clinically significant interactions have been reported between contraceptive hormones and nucleoside reverse transcriptase inhibitors.

Tables 1 and 2 summarize the evidence available about drug interactions between ARV therapies and hormonal contraceptives. For up-to-date, detailed information about human immunodeficiency virus (HIV) drug interactions, the following resources might be helpful:

- *Guidelines for the Use of Antiretroviral Agents in HIV-1-Infected Adults and Adolescents* from the DHHS Panel on Antiretroviral Guidelines for Adults and Adolescents. Available at http://aidsinfo.nih.gov/contentfiles/AdultandAdolescentGL.pdf.
- HIV Drug Interactions website, University of Liverpool, UK. Available at www.hiv-druginteractions.org.

TABLE 1. Drug interactions between COCs and ARV drugs*

ARV	Contraceptive effects†	ARV effects†
Nucleoside reverse transcriptase inhibitors (NRTIs)		
Tenofovir disaproxil fumarate	EE ↔, NGM ↔ (*1*)	Tenofovir ↔ (*1*)
Zidovudine	No data	Zidovudine ↔ (*2*) No change in viral load or CD4+ (*2*)
Non-nucleoside reverse transcriptase inhibitors (NNRTIs)		
Efavirenz	EE ↑ (*3*), EE ↔ (*4*), NGM ↓ (*4*), LNG ↓ (*4*) Pregnancy rate 2.6/100 woman-years in 1 study in which up to 80% used hormonal contraceptives (35% used COC) (*5*)	Efavirenz ↔ (*3,4*)
Etravirine	EE ↔, NET ↔ (*6*)	Etravirine ↑ (*6*) Concurrent administration, generally safe and well tolerated (*6*)
Nevirapine	EE ↔, NET ↔ (*7*)	Nevirapine ↔ (*7*)
Protease inhibitors and ritonavir-boosted protease inhibitors		
Atazanavir/ritonavir	EE ↑, NET ↑ (*8*)	No data
Darunavir/ritonavir	EE ↓, NET ↔ (*9*)	Darunavir ↔ (*9*)
Fos-amprenavir/ritonavir	EE ↓ (*10,11*), NET ↓ (*11*)	Amprenavir ↔, ritonavir ↑, Elevated liver transaminases (*10*)
Indinavir§	EE ↔, NET ↔ (*12*)	No data
Lopinavir/ritonavir	EE ↓, NET ↔ (*13*)	No data
Nelfinavir	EE ↓, NET ↔ (*14*)	No data
Saquinavir§	No data	Saquinavir ↔ (*15,16*)
Tipranavir/ritonavir	EE↓ (*17*)	↑ Skin and musculoskeletal adverse events; possible drug hypersensitivity reaction (*17*)

* Abbreviations: COC = combined oral contraceptive; ARV = antiretroviral; EE = ethinyl estradiol; NGM = norgestimate; NNRTI = non-nucleoside reverse transcriptase inhibitor; LNG = levonorgestrel; NET = norethindrone.
† ↔, no change or change ≤30%; ↑, increase >30%; ↓, decrease >30%.
§ Saquinavir and indinavir are commonly given boosted by ritonavir, but there are no data on contraceptive interactions with the boosted regimens.

TABLE 2. Drug interactions between DMPA and ARV drugs*

ARV	Contraceptive effects†	ARV effects†
Nucleoside reverse transcriptase inhibitors (NRTIs)		
Zidovudine	No data	Zidovudine ↔ (*2*) No change in viral load
Non-nucleoside reverse transcriptase inhibitors (NNRTIs)		
Efavirenz	MPA ↔ (*18,19*) No ovulations during 3 cycles(*18,19*) Pregnancy rate 2.6/100 woman-years in 1 study where up to 80% used hormonal contraceptives (65% used POIs) (*5*)	Efavirenz ↔ (*18*) No change in viral load or CD4+, no grade 3- or 4-related adverse events§ (*20*)
Nevirapine	MPA ↔ (*18*) No ovulations during 3 cycles(*18*)	Nevirapine ↑ (*18*) No change in viral load or CD4+, no grade 3- or 4-related adverse events§ (*20*)
Protease inhibitors and ritonavir-boosted protease inhibitors		
Nelfinavir	MPA ↔ (*18*)	Nelfinavir ↔ (*18*) No change in viral load or CD4+, no grade 3- or 4-related adverse events§ (*20*)

* Abbreviations: DMPA = depot medroxyprogesterone acetate; ARV = antiretroviral; NRTI = nucleoside reverse transcriptase inhibitor; NNRTI = non-nucleoside reverse transcriptase; MPA = medroxyprogesterone acetate; POI = progestin-only injectables.
† ↔, no change or change ≤30%; ↑, increase > 30%.
§ The trial applied the standardized National Institutes of Health Division of AIDS Table for Grading Severity of Adult and Pediatric Adverse Events, 2004 (http://rcc.tech-res.com/Document/safetyandpharmacovigilance/DAIDS_AE_GradingTable_Clarification_August2009_Final.pdf). Grade 3 events are classified as severe. Severe events are defined as symptoms that limit activity or might require some assistance; require medical intervention or therapy; and might require hospitalization. Grade 4 events are classified as life threatening. Life-threatening events include symptoms that result in extreme limitation of activity and require substantial assistance; require substantial medical intervention and therapy; and probably require hospitalization or hospice.

References

1. Kearney BP, Isaacson E, Sayre J, Cheng AK. Tenofovir DF and oral contraceptives: lack of a pharmacokinetic drug interaction [Abstract A-1618]. In: Program and abstracts of the 43rd Interscience Conference on Antimicrobial Agents and Chemotherapy, Chicago, IL, September 14–17, 2003. Washington, DC: American Society for Microbiology; 2003.
2. Aweeka FT, Rosenkranz SL, Segal Y, et al. The impact of sex and contraceptive therapy on the plasma and intracellular pharmacokinetics of zidovudine. AIDS 2006;20:1833–41.
3. Joshi AS, Fiske WD, Benedek IH, et al. Lack of a pharmacokinetic interaction between efavirenz (DMP 266) and ethinyl estradiol in healthy female volunteers [Abstract 348]. 5th Conference on Retroviruses and Opportunistic Infections, Chicago, IL, February 1–5, 1998.
4. Sevinsky H, Eley T, He B, et al. Effect of efavirenz on the pharacokinetics of ethinyl estradiol and norgestimate in healthy female subjects [Abstract A958]. In: Program and abstracts of the 48th Interscience Conference on Antimicrobial Agents and Chemotherapy, Washington, DC, October 25–28, 2008. Washington, DC: American Society for Microbiology; 2008.
5. Danel C, Moh R, Anzian A, et al. Tolerance and acceptability of an efavirenz-based regimen in 740 adults (predominantly women) in West Africa. J Acquir Immune Defic Syndr 2006;42:29–35.
6. Scholler-Gyure M, Debroye C, Aharchi F, et al. No clinically relevant effect of TMC125 on the pharmacokinetics of oral contraceptives. 8th International Congress on Drug Therapy in HIV Infection, Glasgow, UK, November 12–16, 2006..
7. Mildvan D, Yarrish R, Marshak A, et al. Pharmacokinetic interaction between nevirapine and ethinyl estradiol/norethindrone when administered concurrently to HIV-infected women. J Acquir Immune Defic Syndr 2002;29:471–7.
8. Zhang J, Chung E, Eley T et al. Effect of atazanavir/ritonavir on the pharmacokinetics of ethinyl estradiol and 17-deactyl-norgestimate in healthy female subjects [Abstract A-1415]. In: Program and abstracts of the 47th Interscience Conference on Antimicrobial Agents and Chemotherapy, Chicago, IL, September 17–20, 2007. Washington, DC: American Society for Microbiology; 2009.
9. Sekar V, Lefebvre E S-GSeal. Pharacokinetic interaction between nevirapine and ethinyl estradiol, norethindrone, and TMC114, a new protease inhibitor [Abstract A-368]. In: Program and abstracts of the 46th Interscience Conference on Antimicrobial Agents and Chemotherapy, San Francisco, CA, September 27–30, 2006. Washington, DC: American Society for Microbiology; 2009.
10. Glaxo Smith Kline. Prescription medicines. Lexiva (fosamprenavir calcium). Glaxo Smith Kline 2009. Available from http://us.gsk.com/products/assets/us_lexiva.pdf. Accessed March 15, 2010.
11. Glaxo Smith Kline. Study APV10020. A phase I, open label, two period, single-sequence, drug-drug interaction study comparing steady-state plasma ethinyl estradiol and norethisterone pharmacokinetics following administration of brevinor for 21 days with and without fosamprenavir 700 mg twice daily (BID) and ritonavir 100 mg (BID) for 21 days in healthy adult female subjects. Glaxo Smith Kline 2009. Available from http://www.gsk-clinicalstudyregister.com/files/pdf/23138.pdf. Accessed March 15, 2010.
12. Merck & Company. Indinavir patient prescribing information. Merck & Company 2009. Available from http://www.merck.com/product/usa/pi_circulars/c/crixivan/crixivan_pi.pdf. Accessed March 15, 2010.
13. Abbott Laboratories. Lopinavir and ritonavir prescribing information, 2009. Abbott Laboratories 2009. Available from http://www.rxabbott.com/pdf/kaletratabpi.pdf. Accessed March 15, 2010.
14. Agouron Pharmaceuticals. Viracept (Nelfinavir mesylate) prescribing information, 2008. Agouron Pharmaceuticals 2009. Available from http://us.gsk.com/products/assets/us_viracept.pdf. Accessed March 15, 2010.

15. Mayer K, Poblete R, Hathaway B et al. Efficacy, effect of oral contraceptives, and adherence in HIV infected women receiving Fortovase (Saquinavir) soft gel capsule (SQV-SGC; FTV) thrice (TID) and twice (BID) daily regimens. XIII International AIDS Conference, 2000, Durban, South Africa 2009.
16. Frohlich M, Burhenne J, Martin-Facklam M, et al. Oral contraception does not alter single dose saquinavir pharmacokinetics in women. Br J Clin Pharmacol 2004;57:244–52.
17. Food and Drug Administration. Highlights of prescribing information. Aptivus (Tipranavir) Capsules. USFDA 2009. Available from http://www.accessdata.fda.gov/drugsatfda_docs/label/2008/021814s005,022292lbl.pdf.
18. Cohn SE, Park JG, Watts DH, et al. Depo-medroxyprogesterone in women on antiretroviral therapy: effective contraception and lack of clinically significant interactions. Clin Pharmacol Ther 2007;81:222–7.
19. Nanda K, Amaral E, Hays M, et al. Pharmacokinetic interactions between depot medroxyprogesterone acetate and combination antiretroviral therapy. Fertil Steril 2008;90:965–71.
20. Watts DH, Park JG, Cohn SE, et al. Safety and tolerability of depot medroxyprogesterone acetate among HIV-infected women on antiretroviral therapy: ACTG A5093. Contraception 2008;77:84–90.

Abbreviations and Acronyms

A	accept
AIDS	acquired immunodeficiency syndrome
ARV	antiretroviral
BMD	bone mineral density
BMI	body mass index
C	caution
CDC	Centers for Disease Control and Prevention
CHC	combined hormonal contraceptive
CI	coitus interruptus
COC	combined oral contraceptive
Cu-IUD	copper intrauterine device
D	delayed
DMPA	depot medroxyprogesterone acetate
DVT	deep venous thrombosis
ECP	emergency contraceptive pills
EE	ethinyl estradiol
E-IUD	emergency intrauterine device
ETG	etonogestrel
FAB	fertility awareness–based methods
hCG	human chorionic gonadotropin
HDL	high-density lipoprotein
HIV	human immunodeficiency virus
HPV	human papillomavirus
IBD	inflammatory bowel disease
IUS	intrauterine system
IUD	intrauterine device
LNG	levonorgestrel
LNG-IUD	levonorgestrel-releasing intrauterine device
MEC	Medical Eligibility Criteria
NET-EN	norethisterone enantate
NGM	norgestimate
NNRTI	non-nucleoside reverse transcriptase inhibitor
NRTI	nucleoside reverse transcriptase inhibitor
P	combined hormonal contraceptive patch
PE	pulmonary embolism
PID	pelvic inflammatory disease
POC	progestin-only contraceptive
POI	progestin-only injectable
POP	progestin-only pill
R	combined hormonal vaginal ring
SLE	systemic lupus erythematosus
STI	sexually transmitted infection
VTE	venous thromboembolism
WHO	World Health Organization

U.S. Medical Eligibility Criteria for Contraceptive Use, 2010

Atlanta, GA, February 17–19, 2009

Chairpersons: Herbert B. Peterson, MD, University of North Carolina, Chapel Hill, North Carolina; Kathryn M. Curtis, PhD, Centers for Disease Control and Prevention, Atlanta, Georgia.

CDC Steering Committee: Kathryn M. Curtis, PhD (Chair), Denise Jamieson, MD, John Lehnherr, Polly Marchbanks, PhD, Centers for Disease Control and Prevention, Atlanta, Georgia.

Systematic Review Authors and Presenters: Sherry Farr, PhD, Suzanne Gaventa Folger, PhD, Melissa Paulen, MPH, Naomi Tepper, MD, Maura Whiteman, PhD, Lauren Zapata, PhD, Centers for Disease Control and Prevention, Atlanta, Georgia; Kelly Culwell, MD, Nathalie Kapp, MD, World Health Organization, Geneva, Switzerland; Catherine Cansino, MD, Johns Hopkins Bayview Medical Center, Baltimore, Maryland.

Invited Participants: Abbey Berenson, MD, University of Texas Medical Branch, Nassau Bay, Texas; Paul Blumenthal, MD, Stanford University, Palo Alto, California (not able to attend); Willard Cates, Jr., MD, Family Health International, Research Triangle Park, North Carolina (not able to attend); Mitchell Creinin, MD, University of Pittsburgh, Pittsburgh, Pennsylvania; Vanessa Cullins, MD, Planned Parenthood Federation of America, New York, New York; Philip Darney, MD, University of California, San Francisco, California; Jennifer Dietrich, MD, Baylor College of Medicine, Houston, Texas; Linda Dominguez, Southwest Women's Health, Albuquerque, New Mexico; Melissa Gilliam, MD, The University of Chicago, Chicago, Illinois; Marji Gold, MD, Albert Einstein College of Medicine, Bronx, New York; Alisa Goldberg, MD, Brigham and Women's Hospital and Planned Parenthood of Massachusetts, Boston, Massachusetts; David Grimes, MD, Family Health International, Research Triangle Park, North Carolina (not able to attend); Robert Hatcher, MD, Emory University, Atlanta, Georgia; Stephen Heartwell, DrPH, Susan Thompson Buffett Foundation, Omaha, Nebraska; Andrew Kaunitz, MD, University of Florida, Jacksonville, Florida; Uta Landy, PhD, University of California, San Francisco, California (not able to attend); Hal Lawrence, MD, American College of Obstetricians and Gynecologists, Washington, DC; Ruth Lawrence, MD, American Academy of Pediatrics and University of Rochester, Rochester, New York; Laura MacIsaac, MD, Albert Einstein School of Medicine, New York, New York; Trent MacKay, MD, National Institute of Child Health and Human Development, National Institutes of Health, Bethesda, MD (not able to attend); Daniel Mishell, Jr, MD, University of Southern California, Los Angeles, California; Mary Mitchell, American College of Obstetricians and Gynecologists, Washington, DC; Susan Moskosky, MS, US Department of Health and Human Services, Rockville, Maryland; Patricia Murphy, DrPH, University of Utah, Salt Lake City, Utah; Kavita Nanda, MD, Family Health International, Research Triangle Park, North Carolina; Jeffrey Peipert, MD, Washington University, St. Louis, Missouri; Michael Policar, MD, University of California, San Francisco, California; Robert Rebar, MD, American Society of Reproductive Medicine, Birmingham, Alabama; Pablo Rodriquez, MD, Providence, Rhode Island (not able to attend); John Santelli, MD, Columbia University, New York, New York (not able to attend); Sharon Schnare, MSN, University of Washington, Seattle, Washington; David Soper, MD, University of South Carolina, Charleston, South Carolina; Lisa Soule, MD, Food and Drug Administration, Silver Spring, Maryland; James Trussell, PhD, Princeton University, Princeton, New Jersey; Carolyn Westhoff, MD, Columbia University, New York, New York (not able to attend); Susan Wysocki, National Association of Nurse Practitioners in Women's Health, Washington, DC; Mimi Zieman, MD, Emory University, Atlanta, Georgia.

Consultants: Wendy Book, MD, Emory University, Atlanta, Georgia; Shinya Ito, Hospital for Sick Children, Toronto, Canada; Beth Jonas, MD, University of North Carolina, Chapel Hill, North Carolina; Miriam Labbok, MD, University of North Carolina, Chapel Hill, North Carolina; Frederick Naftolin, MD, New York University, New York, New York; Lubna Pal, Yale University, New Haven, Connecticut; Robin Rutherford, MD, Emory University, Atlanta, Georgia; Roshan Shrestha, MD, Piedmont Hospital, Atlanta, Georgia; Kimberley Steele, MD, Johns Hopkins University, Baltimore, Maryland; Michael Streiff, MD, Johns Hopkins University, Baltimore, Maryland; Christine Wagner, PhD, University of Albany, Albany, New York; Joan Walker, MD, University of Oklahoma, Oklahoma City, Oklahoma.

CDC Attendees: Janet Collins, PhD, Susan Hillis, PhD, Dmitry Kissin MD, Sam Posner, PhD, Natalya Revzina, MD, Cheryl Robbins, PhD, Lee Warner, PhD.

This work was conducted within the Women's Health and Fertility Branch (Maurizio Macaluso, Branch Chief), in the Division of Reproductive Health (John Lehnherr, Acting Director), National Center for Chronic Disease Prevention and Health Promotion (Ursula Bauer, Director).

The *Morbidity and Mortality Weekly Report (MMWR)* Series is prepared by the Centers for Disease Control and Prevention (CDC) and is available free of charge in electronic format. To receive an electronic copy each week, visit *MMWR*'s free subscription page at *http://www.cdc.gov/mmwr/mmwrsubscribe.html*. Paper copy subscriptions are available through the Superintendent of Documents, U.S. Government Printing Office, Washington, DC 20402; telephone 202-512-1800.

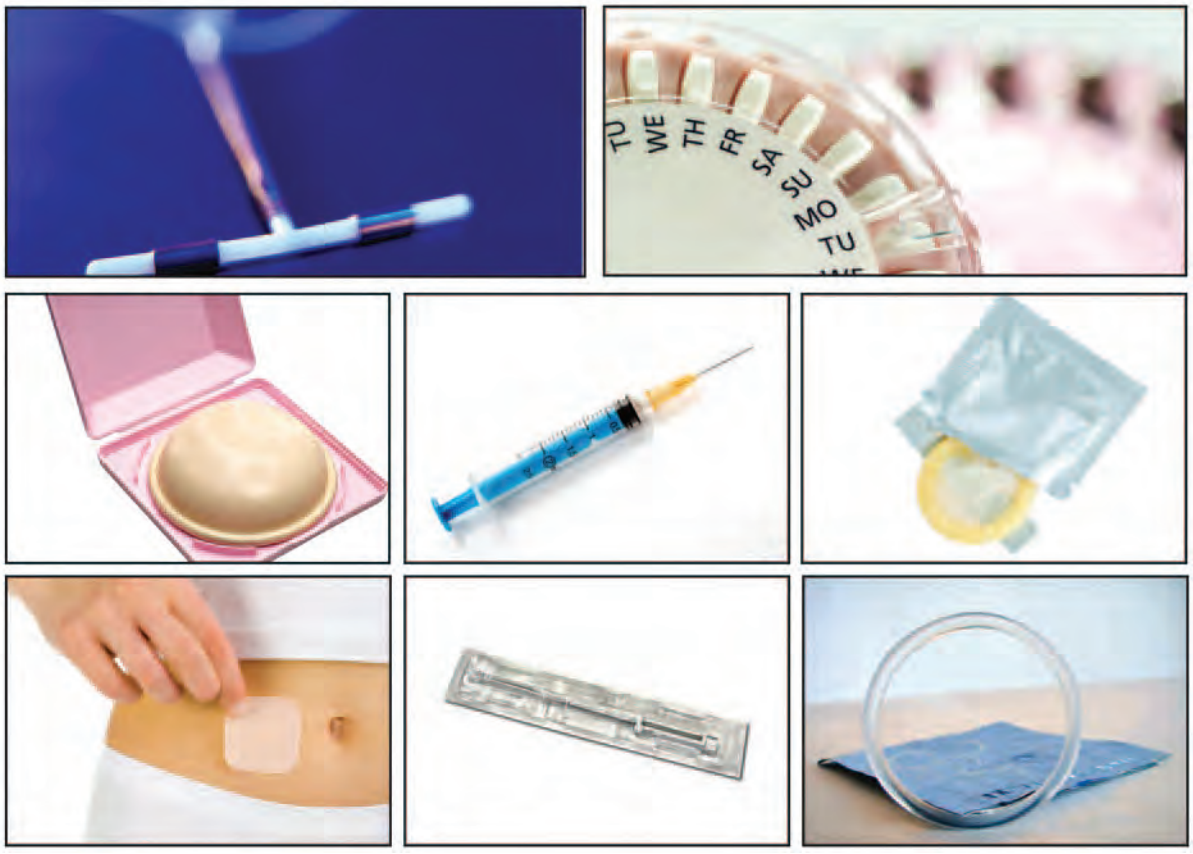
Data presented by the Notifiable Disease Data Team and 122 Cities Mortality Data Team in the weekly *MMWR* are provisional, based on weekly reports to CDC by state health departments. Address all inquiries about the *MMWR* Series, including material to be considered for publication, to Editor, *MMWR* Series, Mailstop E-90, CDC, 1600 Clifton Rd., N.E., Atlanta, GA 30333 or to *mmwrq@cdc.gov.*

☆U.S. Government Printing Office: 2010-623-026/41253 Region IV ISSN: 1057-5987

Centers for Disease Control and Prevention
MMWR
Morbidity and Mortality Weekly Report
Recommendations and Reports / Vol. 62 / No. 5
June 21, 2013

U.S. Selected Practice Recommendations for Contraceptive Use, 2013

Adapted from the World Health Organization Selected Practice Recommendations for Contraceptive Use, 2nd Edition



Continuing Education Examination available at http://www.cdc.gov/mmwr/cme/conted.html.

U.S. Department of Health and Human Services
Centers for Disease Control and Prevention

CONTENTS

CONTENTS (*Continued*)

Disclosure of Relationship

CDC, our planners, and our content experts wish to disclose they have no financial interests or other relationships with the manufacturers of commercial products, suppliers of commercial services, or commercial supporters. Planners have reviewed content to ensure there is no bias. This document will not include any discussion of the unlabeled use of a product or a product under investigational use, with the exception that some of the recommendations in this document might be inconsistent with package labeling. CDC does not accept commercial support.

Front cover photos, *left to right:* intrauterine device, oral contraceptive pills, diaphragm, syringe for injectable contraceptives, male condom, transdermal contraceptive patch, etonogestrel implant, vaginal ring.

The *MMWR* series of publications is published by the Office of Surveillance, Epidemiology, and Laboratory Services, Centers for Disease Control and Prevention (CDC), U.S. Department of Health and Human Services, Atlanta, GA 30333.

Suggested Citation: Centers for Disease Control and Prevention. [Title]. MMWR 2013;62(No. RR-#):[inclusive page numbers].

U.S. Selected Practice Recommendations for Contraceptive Use, 2013

Adapted from the World Health Organization Selected Practice Recommendations for Contraceptive Use, 2nd Edition

Prepared by
Division of Reproductive Health, National Center for Chronic Disease Prevention and Health Promotion

Summary

The U. S. Selected Practice Recommendations for Contraceptive Use 2013 *(U.S. SPR), comprises recommendations that address a select group of common, yet sometimes controversial or complex, issues regarding initiation and use of specific contraceptive methods. These recommendations are a companion document to the previously published CDC recommendations* U.S. Medical Eligibility Criteria for Contraceptive Use, 2010 *(U.S. MEC). U.S. MEC describes who can use various methods of contraception, whereas this report describes how contraceptive methods can be used. CDC based these U.S. SPR guidelines on the global family planning guidance provided by the World Health Organization (WHO). Although many of the recommendations are the same as those provided by WHO, they have been adapted to be more specific to U.S. practices or have been modified because of new evidence. In addition, four new topics are addressed, including the effectiveness of female sterilization, extended use of combined hormonal methods and bleeding problems, starting regular contraception after use of emergency contraception, and determining when contraception is no longer needed. The recommendations in this report are intended to serve as a source of clinical guidance for health-care providers; health-care providers should always consider the individual clinical circumstances of each person seeking family planning services. This report is not intended to be a substitute for professional medical advice for individual patients. Persons should seek advice from their health-care providers when considering family planning options.*

Introduction

Unintended pregnancy rates remain high in the United States; approximately 50% of all pregnancies are unintended, with higher proportions among adolescent and young women, women who are racial/ethnic minorities, and women with lower levels of education and income (*1*). Unintended pregnancies increase the risk for poor maternal and infant outcomes (*2*) and in 2002, resulted in $5 billion in direct medical costs in the United States (*3*). Approximately half of unintended pregnancies are among women who were not using contraception at the time they became pregnant; the other half are among women who became pregnant despite reported use of contraception (*4*). Therefore, strategies to prevent unintended pregnancy include assisting women at risk for unintended pregnancy and their partners with choosing appropriate contraceptive methods and helping women use methods correctly and consistently to prevent pregnancy. In 2010, CDC first adapted global guidance from the World Health Organization (WHO) to help health-care providers counsel women, men, and couples about contraceptive method choice. The *U.S. Medical Eligibility Criteria for Contraceptive Use, 2010* (U.S. MEC), focuses on who can safely use specific methods of contraception and provides recommendations for the safety of contraceptive methods for women with various medical conditions (e.g., hypertension and diabetes) and characteristics (e.g., age, parity, and smoking status) (Appendix A) (*5*). The recommendations in this new guide, *U.S. Selected Practice Recommendations for Contraceptive Use, 2013* (U.S. SPR), focuses on how contraceptive methods can be used and provides recommendations on optimal use of contraceptive methods for persons of all ages, including adolescents.

During the past 15 years, CDC has contributed to the development and updating of the WHO global family planning guidance. CDC has supported WHO by coordinating the identification, critical appraisal, and synthesis of the scientific evidence on which the WHO guidance is based. In 2002, WHO published the first edition of the *Selected Practice Recommendations for Contraceptive Use* (WHO SPR), which presented evidence-based global guidance on how to use contraceptive methods safely and effectively once they are deemed to be medically appropriate. Since then, WHO has regularly updated its guidance on the basis of new evidence, and the document is now in its second edition (*6*), with an additional update in 2008 (*7*). The WHO global guidance is not intended for use directly by health-care providers; rather, WHO intends for the guidance to be used by local or national policy makers, family planning program managers, and the

The material in this report originated in the National Center for Chronic Disease Prevention and Health Promotion, Ursula Bauer, PhD, Director; Division of Reproductive Health, Wanda Barfield, MD, Director.
Corresponding preparer: Kathryn M. Curtis, PhD, Division of Reproductive Health. Telephone: 770-488-5200; E-mail: kmc6@cdc.gov.

scientific community as a reference when they develop family planning guidance at the country or program level (*6*). For example, the United Kingdom adapted WHO SPR and in 2002 published the *U.K. Selected Practice Recommendations for Contraceptive Use* for use by U.K. health-care providers (*8*).

CDC initiated a formal adaptation process to create U.S. SPR, using both the second edition of WHO SPR (*6*) and the 2008 update (*7*) as the basis for the U.S. version. Although much of the guidance is the same as the WHO guidance, the recommendations are specific to U.S. family planning practice. In addition, guidance on contraceptive methods not available in the United States has been removed, and four new topics for guidance have been added (the effectiveness of female sterilization, extended use of combined hormonal methods and bleeding problems, starting regular contraception after use of emergency contraception, and determining when contraception is no longer needed). This document contains recommendations for health-care providers for the safe and effective use of contraceptive methods and addresses provision of contraceptive methods and management of side effects and other problems with contraceptive method use. Although the term woman is used throughout this report, these recommendations refer to all females of reproductive age, including adolescents. Adolescents are identified throughout this document as a special population that might benefit from more frequent follow-up. These recommendations are meant to serve as a source of clinical guidance for health-care providers; health-care providers should always consider the individual clinical circumstances of each person seeking family planning services. This report is not intended to be a substitute for professional medical advice for individual patients; persons should seek advice from their health-care providers when considering family planning options.

Methods

CDC initiated a process to adapt WHO SPR for the United States. This adaptation process included four steps: 1) determining the scope of and process for the adaptation, including an October 2010 meeting in which individual feedback was solicited from a small group of partners and experts; 2) preparing the systematic reviews of the evidence during October 2010–September 2011 to be used for the adaptation, including peer review; 3) convening a larger meeting of experts in October 2011 to examine the evidence and receive input on the recommendations; and 4) finalizing recommendations by CDC.

During October 21–22, 2010, CDC convened a meeting of 10 partners and U.S. family planning experts in Atlanta, Georgia, to discuss the scope of and process for a U.S. adaptation of WHO SPR. A list of participants is provided at the end of this report. CDC identified the specific WHO recommendations that might benefit from modification for the United States. Criteria used to modify the WHO recommendations included the availability of new scientific evidence or the context in which family planning services are provided in the United States. CDC also identified several WHO recommendations that needed additional specificity to be useful for U.S. health-care providers, as well as the need for additional recommendations not currently included in WHO SPR. In addition, the meeting members discussed removing recommendations that provide information about contraceptive methods that are not available in the United States.

Representatives from CDC and WHO conducted systematic reviews of the scientific evidence for each of the WHO recommendations being considered for adaptation and for each new topic being considered for addition to the guidance. The purpose of these systematic reviews was to identify evidence related to the common clinical challenges associated with the recommendations. When no direct evidence was available, indirect evidence and theoretical issues were considered. Standard guidelines were followed for reporting systematic reviews (*9,10*), and strength and quality of the evidence were graded using the system of the U.S. Preventive Services Task Force (*11*). Each complete systematic review was peer reviewed by two or three experts before its use in the adaptation process. Peer reviewers, who were identified from the list of persons scheduled to participate in the October 2011 meeting, were asked to comment on the search strategy, list of articles included in the reviews, and the summary of findings. The systematic reviews were finalized and provided to participants before the October 2011 meeting and were published in May 2013 (*12–30*).

During October 4–7, 2011, CDC convened a meeting in Atlanta, Georgia, of 36 experts who were invited to assist in guideline development and provide their perspective on the scientific evidence presented and the discussions on potential recommendations that followed. The group included obstetrician/gynecologists, pediatricians, family physicians, nurse-midwives, nurse practitioners, epidemiologists, and others with research and clinical practice expertise in contraceptive safety, effectiveness, and management. All participants received all of the systematic reviews before the meeting. During the meeting, the evidence from the systematic review for each topic was presented, and participants discussed the evidence and the translation of the scientific evidence into recommendations that would meet the needs of U.S. health-care providers. In particular, participants discussed whether and how the U.S. context might be different from the global context and whether these differences suggested any need for modifications to the global guidance. CDC gathered the input from the experts during the meeting and finalized the recommendations in this

report. The document was peer reviewed by meeting participants, who were asked to comment on specific issues that were raised during the meeting. Feedback also was received from an external review panel, composed of health-care providers who had not participated in the adaptation meetings. These providers were asked to provide comments on the accuracy, feasibility, and clarity of the recommendations, as well as to provide other comments. Areas of research that need additional investigation also were considered during the meeting (*31*).

How To Use This Document

The recommendations in this report are intended to help health-care providers address issues related to use of contraceptives, such as how to help a woman initiate use of a contraceptive method, which examinations and tests are needed before initiating use of a contraceptive method, what regular follow-up is needed, and how to address problems that often arise during use, including missed pills and side effects such as unscheduled bleeding. Each recommendation addresses what a woman or health-care provider can do in specific situations. For situations in which certain groups of women might be medically ineligible to follow the recommendations, comments and reference to U.S. MEC are provided (*5*). The full U.S. MEC recommendations and the evidence supporting those recommendations were published in 2010 (*5*).

The information in this document is organized by contraceptive method, and the methods generally are presented in order of effectiveness, from highest to lowest. However, the recommendations are not intended to provide guidance on every aspect of provision and management of contraceptive method use. Instead, they use the best available evidence to address specific issues regarding common, yet sometimes complex, clinical issues. Each contraceptive method section generally includes information about initiation of the method, regular follow-up, and management of problems with use (e.g., usage errors and side effects). Each section first provides the recommendation and then includes a comments and evidence section, which includes comments about the recommendations and a brief summary of the scientific evidence on which the recommendation is based.

Recommendations in this document are provided for permanent methods of contraception, such as vasectomy and female sterilization, as well as for reversible methods of contraception, including the copper-containing intrauterine device (Cu-IUD); levonorgestrel-releasing IUD (LNG-IUD); the etonogestrel implant; progestin-only injectables; progestin-only pills (POPs); combined hormonal contraceptive methods that contain both estrogen and a progestin, including combined oral contraceptives (COCs), a transdermal contraceptive patch, and a vaginal contraceptive ring; and the standard days method (SDM). Recommendations also are provided for emergency use of the Cu-IUD and emergency contraceptive pills (ECPs).

For each contraceptive method, recommendations are provided on the timing for initiation of the method and indications for when and for how long additional contraception, or a back-up method, is needed. Many of these recommendations include guidance that a woman can start a contraceptive method at any time during her menstrual cycle if it is reasonably certain that the woman is not pregnant. Guidance for health-care providers on how to be reasonably certain that a woman is not pregnant is provided.

For each contraceptive method, recommendations include the examinations and tests needed before initiation of the method. These recommendations apply to persons who are presumed to be healthy. Those with known medical problems or other special conditions might need additional examinations or tests before being determined to be appropriate candidates for a particular method of contraception. U.S. MEC might be useful in such circumstances (*5*). Most women need no or very few examinations or tests before initiating a contraceptive method. The following classification system was developed by WHO and adopted by CDC to categorize the applicability of the various examinations or tests before initiation of contraceptive methods (*6*):

Class A: These tests and examinations are essential and mandatory in all circumstances for safe and effective use of the contraceptive method.

Class B: These tests and examinations contribute substantially to safe and effective use, although implementation can be considered within the public health context, service context, or both. The risk for not performing an examination or test should be balanced against the benefits of making the contraceptive method available.

Class C: These tests and examinations do not contribute substantially to safe and effective use of the contraceptive method.

These classifications focus on the relation of the examinations or tests to safe initiation of a contraceptive method. They are not intended to address the appropriateness of these examinations or tests in other circumstances. For example, some of the examinations or tests that are not deemed necessary for safe and effective contraceptive use might be appropriate for good preventive health care or for diagnosing or assessing suspected medical conditions. Systematic reviews were conducted for several different types of examinations and tests to assess whether a screening test was associated with safe use of contraceptive methods. Because no single convention exists for screening panels for certain diseases, including diabetes,

lipid disorders, and liver diseases, the search strategies included broad terms for the tests and diseases of interest.

Summary charts and clinical algorithms that summarize the guidance for the various contraceptive methods have been developed for many of the recommendations, including when to start using specific contraceptive methods (Appendix B), examinations and tests needed before initiating the various contraceptive methods (Appendix C), routine follow-up after initiating contraception (Appendix D), management of bleeding irregularities (Appendix E), and management of IUDs when users are found to have pelvic inflammatory disease (PID) (Appendix F). These summaries might be helpful to health-care providers when managing family planning patients. Additional tools are available on the U.S. SPR website (http://www.cdc.gov/reproductivehealth/UnintendedPregnancy/USSPR.htm).

Summary of Changes from WHO SPR

Much of the guidance in U.S. SPR is the same or very similar to the WHO SPR guidance. U.S. SPR includes new guidance on the use of the combined contraceptive patch and vaginal ring, as well as recommendations for four new topics:

- how to start regular contraception after taking ECPs
- management of bleeding irregularities among women using extended or continuous combined hormonal contraceptives (including pills, the patch, and the ring)
- when a woman can rely on female sterilization for contraception
- when a woman can stop using contraceptives and not be at risk for unintended pregnancy

Adaptations to the WHO SPR recommendations include 1) changes to the length of the grace period for depot medroxyprogesterone acetate (DMPA) reinjection, 2) differences in some of the examinations and tests recommended before contraceptive method initiation, 3) differences in some of the recommendations for management of bleeding irregularities because of new data and drug availability in the United States, and 4) a modified missed pill algorithm to respond to concerns of the CDC expert group and other reviewers that simplified algorithms are preferable.

Contraceptive Method Choice

Many elements need to be considered individually by a woman, man, or couple when choosing the most appropriate contraceptive method. Some of these elements include safety, effectiveness, availability (including accessibility and affordability), and acceptability.

Contraceptive method effectiveness is critically important in minimizing the risk for unintended pregnancy, particularly among women for whom an unintended pregnancy would pose additional health risks. The effectiveness of contraceptive methods depends both on the inherent effectiveness of the method itself and on how consistently and correctly it is used (Table 1). Both consistent and correct use can vary greatly with characteristics such as age, income, desire to prevent or delay pregnancy, and culture. Methods that depend on consistent and correct use by clients have a wide range of effectiveness between typical and perfect users. IUDs and implants are considered long-acting, reversible contraception (LARC); these methods are highly effective because they do not depend on regular compliance from the user. LARC methods are appropriate for most women, including adolescents and nulliparous women. All women should be counseled about the full range and effectiveness of contraceptive options for which they are medically eligible so that they can identify the optimal method (Figure 1).

In choosing a method of contraception, the risk for human immunodeficiency virus (HIV) infection and other sexually transmitted diseases (STDs) also should be considered. Although hormonal contraceptives and IUDs are highly effective at preventing pregnancy, they do not protect against STDs and HIV. Consistent and correct use of the male latex condom reduces the risk for HIV infection and other STDs, including chlamydial infection, gonorrhea, and trichomoniasis (*32*). On the basis of a limited number of clinical studies, when a male condom cannot be used properly to prevent infection, a female condom should be considered (*32*). All patients, regardless of contraceptive choice, should be counseled about the use of condoms and the risk for STDs, including HIV infection (*32*). Additional information about prevention and treatment of STDs is available from the CDC *Sexually Transmitted Diseases Treatment Guidelines* (*32*).

Maintaining Updated Guidance

As with any evidence-based guidance document, a key challenge is keeping the recommendations up to date as new scientific evidence becomes available. Working with WHO, CDC uses the continuous identification of research evidence (CIRE) system to ensure that WHO and CDC guidance is based on the best available evidence and that a mechanism is in place to update guidance when new evidence becomes available (*33*). CDC will continue to work with WHO to identify and assess all new relevant evidence and determine whether changes in the recommendations are warranted. In

TABLE 1. Percentage of women experiencing an unintended pregnancy during the first year of typical use and the first year of perfect use of contraception and the percentage continuing use at the end of the first year — United States

Method	% of women experiencing an unintended pregnancy within the first year of use		% of women continuing use at 1 year[§]
	Typical use*	Perfect use[†]	
No method[¶]	85	85	—
Spermicides**	28	18	42
Fertility awareness–based methods[††]	24	—	47
Standard days method	—	5	—
Two day method	—	4	—
Ovulation method	—	3	—
Symptothermal method	—	0.4	—
Withdrawal	22	4	46
Sponge			
Parous women	24	20	36
Nulliparous women	12	9	—
Condom[§§]			
Female	21	5	41
Male	18	2	43
Diaphragm***	12	6	57
Combined pill and progestin-only pill	9	0.3	67
Evra patch	9	0.3	67
NuvaRing	9	0.3	67
Depo-Provera	6	0.2	56
Intrauterine devices			
Paragard (copper containing)	0.8	0.6	78
Mirena (levenorgestrel releasing)	0.2	0.2	80
Implanon	0.05	0.05	84
Female sterilization	0.5	0.5	100
Male sterilization	0.15	0.10	100
Lactational amenorrhea method[†††]	—	—	—

Source: Adapted from Trussell J. Contraceptive failure in the United States. Contraception 2011;83:397–404.

* Among typical couples who initiate use of a method (not necessarily for the first time), the percentage who experience an accidental pregnancy during the first year if they do not stop use for any other reason. Estimates of the probability of pregnancy during the first year of typical use for spermicides and the diaphragm are taken from the 1995 National Survey of Family Growth (NSFG) corrected for underreporting of abortion; estimates for fertility awareness-based methods, withdrawal, the male condom, the pill and Depo-Provera are taken from the 1995 and 2002 NSFG corrected for underreporting of abortion.

† Among couples who initiate use of a method (not necessarily for the first time) and who use it perfectly (both consistently and correctly), the percentage who experience an accidental pregnancy during the first year if they do not stop use for any other reason.

§ Among couples attempting to avoid pregnancy, the percentage who continues to use a method for 1 year.

¶ The percentage becoming pregnant in the second and third columns are based on data from populations where contraception is not used and from women who cease using contraception to become pregnant. Among such populations, approximately 89% become pregnant within 1 year. This estimate was lowered slightly (to 85%) to represent the percentage who would become pregnant within 1 year among women not relying on reversible methods of contraception if they abandoned contraception altogether.

** Foams, creams, gels, vaginal suppositories, and vaginal film.

†† The ovulation and two day methods are based on evaluation of cervical mucus. The standard days method avoids intercourse on cycle days 8–19. The symptothermal method is a double-check method based on evaluation of cervical mucus to determine the first fertile day and evaluation of cervical mucus and temperature to determine the last fertile day.

§§ Without spermicides.

*** With spermicidal cream or jelly.

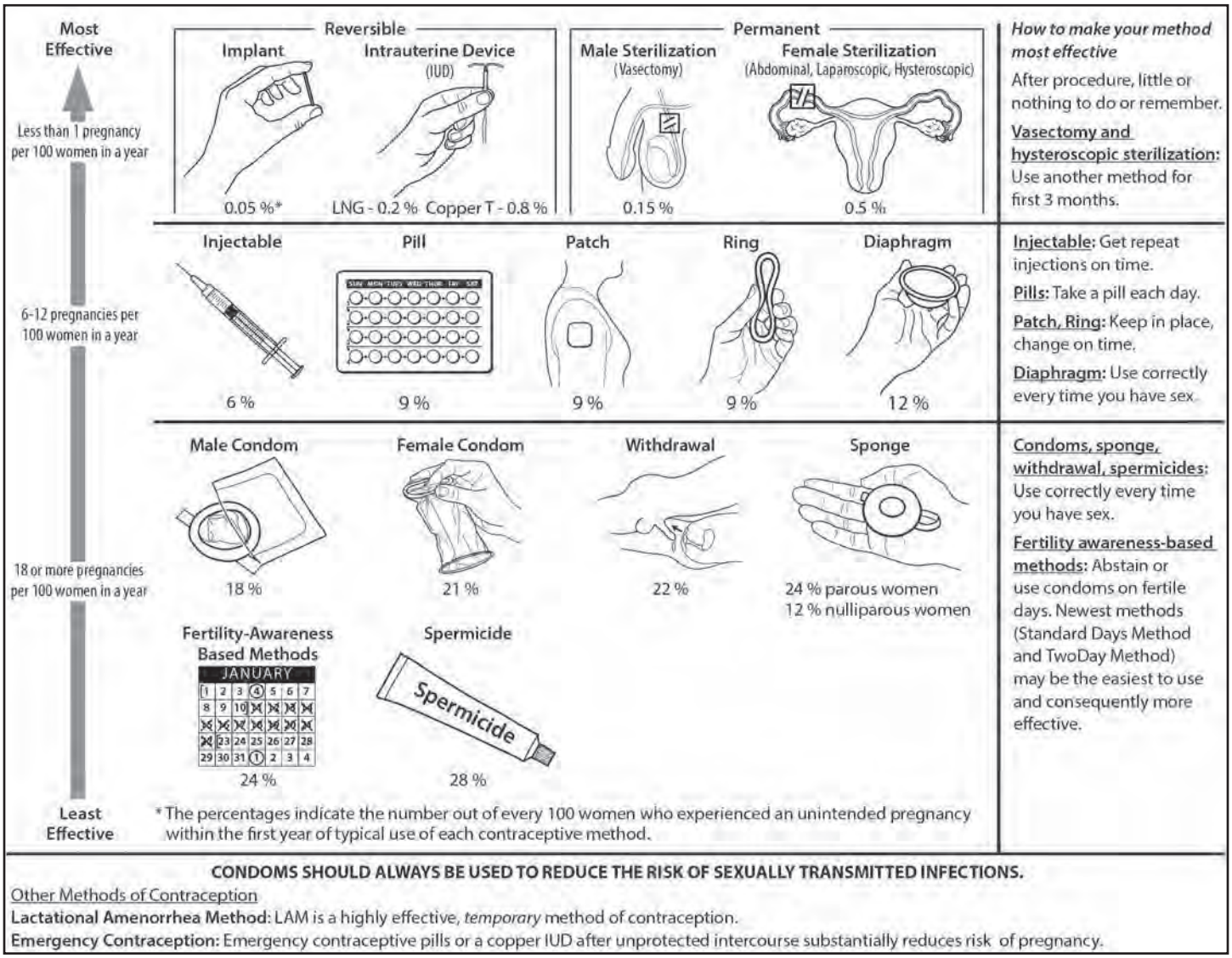
††† This is a highly effective, temporary method of contraception. However, to maintain in effective protection against pregnancy, another method of contraception must be used as soon as menstruation resumes, the frequency of duration of breastfeeds is reduced, bottle feeds are introduced, or the baby reaches age 6 months.

most cases, U.S. SPR will follow any updates in the WHO guidance, which typically occurs every 3–4 years (or sooner if warranted by new data). In addition, CDC will review any interim WHO updates for their application in the United States. CDC also will identify and assess any new literature for the recommendations that are not included in the WHO guidance and will completely review U.S. SPR every 3–4 years. Updates to the guidance can be found on the U.S. SPR website (http://www.cdc.gov/reproductivehealth/UnintendedPregnancy/USSPR.htm).

How To Be Reasonably Certain that a Woman Is Not Pregnant

In most cases, a detailed history provides the most accurate assessment of pregnancy risk in a woman who is about to start using a contraceptive method. Several criteria for assessing pregnancy risk are listed in the recommendation that follows. These criteria are highly accurate (i.e., a negative predictive value of 99%–100%) in ruling out pregnancy among women who are not pregnant (*34–37*). Therefore, CDC recommends that health-care providers use these criteria to assess pregnancy

FIGURE 1. Effectiveness of family planning methods



Sources: Adapted from World Health Organization (WHO) Department of Reproductive Health and Research, Johns Hopkins Bloomberg School of Public Health/ Center for Communication Programs (CCP). Knowledge for health project. Family planning: a global handbook for providers (2011 update). Baltimore, MD; Geneva, Switzerland: CCP and WHO; 2011; and Trussell J. Contraceptive failure in the United States. Contraception 2011;83:397–404.

status in a woman who is about to start using contraceptives (Box 1). If a woman meets one of these criteria (and therefore the health-care provider can be reasonably certain that she is not pregnant), a urine pregnancy test might be considered in addition to these criteria (based on clinical judgment), bearing in mind the limitations of the accuracy of pregnancy testing. If a woman does not meet any of these criteria, then the health-care provider cannot be reasonably certain that she is not pregnant, even with a negative pregnancy test. Routine pregnancy testing for every woman is not necessary.

On the basis of clinical judgment, health-care providers might consider the addition of a urine pregnancy test; however, they should be aware of the limitations, including accuracy of the test relative to the time of last sexual intercourse, recent delivery, or spontaneous or induced abortion. Routine pregnancy testing for every woman is not necessary. If a woman has had recent (i.e., within the last 5 days) unprotected sexual intercourse, consider offering emergency contraception (either a Cu-IUD or ECPs), if pregnancy is not desired.

Comments and Evidence Summary. The criteria for determining whether a woman is pregnant depend on the assurance that she has not ovulated within a certain amount of time after her last menses, spontaneous or induced abortion, or delivery. Among menstruating women, the timing of ovulation can vary widely. During an average 28-day cycle, ovulation generally occurs during days 9–20 (*38*). In addition, the

BOX 1. How To Be Reasonably Certain that a Woman Is Not Pregnant

A health-care provider can be reasonably certain that a woman is not pregnant if she has no symptoms or signs of pregnancy and meets any one of the following criteria:

- is ≤7 days after the start of normal menses
- has not had sexual intercourse since the start of last normal menses
- has been correctly and consistently using a reliable method of contraception
- is ≤7 days after spontaneous or induced abortion
- is within 4 weeks postpartum
- is fully or nearly fully breastfeeding (exclusively breastfeeding or the vast majority [≥85%] of feeds are breastfeeds),* amenorrheic, and <6 months postpartum

*Source: Labbok M, Perez A, Valdez V, et al. The Lactational Amenorrhea Method (LAM): a postpartum introductory family planning method with policy and program implications. Adv Contracept 1994;10:93–109.

likelihood of ovulation is low from days 1–7 of the menstrual cycle (*39*). After a spontaneous or an induced abortion, ovulation can occur within 2–3 weeks and has been found to occur as early as 8–13 days after the end of the pregnancy. Therefore, the likelihood of ovulation is low ≤7 days after an abortion (*40–42*). A recent systematic review reported that the mean day of first ovulation among postpartum nonlactating women occurred 45–94 days after delivery (*43*). In one study, the earliest ovulation was reported at 25 days after delivery. Among women who are within 6 months postpartum, are fully or nearly fully breastfeeding, and are amenorrheic, the risk for pregnancy is <2% (*44*).

Although pregnancy tests often are performed before initiating contraception, the accuracy of qualitative urine pregnancy tests varies depending on the timing of the test relative to missed menses, recent sexual intercourse, or recent pregnancy. The sensitivity of a pregnancy test is defined as the concentration of human chorionic gonadotropin (hCG) at which 95% of tests are positive. Most qualitative pregnancy tests approved by the U.S. Food and Drug Administration (FDA) report a sensitivity of 20–25 mIU/mL in urine (*45–48*) However, pregnancy detection rates can vary widely because of differences in test sensitivity and the timing of testing relative to missed menses (*47*,*49*). Some studies have shown that an additional 11 days past the day of expected menses are needed to detect 100% of pregnancies using qualitative tests (*46*). In addition, pregnancy tests cannot detect a pregnancy resulting from recent sexual intercourse. Qualitative tests also might have positive results for several weeks after termination of pregnancy because hCG can be present for several weeks after delivery or abortion (spontaneous or induced) (*50–52*).

For contraceptive methods other than IUDs, the benefits of starting to use a contraceptive method likely exceed any risk, even in situations in which the health-care provider is uncertain whether the woman is pregnant. Therefore, the health-care provider can consider having patients start using contraceptive methods other than IUDs at any time, with a follow-up pregnancy test in 2–4 weeks. The risks of not starting to use contraception should be weighed against the risks of initiating contraception use in a woman who might be already pregnant. Most studies have shown no increased risk for adverse outcomes, including congenital anomalies or neonatal or infant death, among infants exposed in utero to COCs (*53–55*). Studies also have shown no increased risk for neonatal or infant death or developmental abnormalities among infants exposed in utero to DMPA (*54*,*56*,*57*).

In contrast, for women who want to begin using an IUD (Cu-IUD or LNG-IUD), in situations in which the health-care provider is uncertain whether the woman is pregnant, the woman should be provided with another contraceptive method to use until the health-care provider is reasonably certain that she is not pregnant and can insert the IUD. Pregnancies among women with IUDs are at higher risk for complications such as spontaneous abortion, septic abortion, preterm delivery, and chorioamnionitis (*58*).

A systematic review identified four analyses of data from three diagnostic accuracy studies that evaluated the performance of the criteria listed above through use of a pregnancy checklist compared with a urine pregnancy test conducted concurrently (*12*). The performance of the checklist to diagnose or exclude pregnancy varied, with sensitivity of 55%–100% and specificity of 39%–89%. The negative predictive value was consistent across studies at 99%–100%; the pregnancy checklist correctly ruled out women who were not pregnant. One of the studies assessed the added usefulness of signs and symptoms of pregnancy and found that these criteria did not substantially improve the performance of the pregnancy checklist, although the number of women with signs and symptoms was small (*34*) (Level of evidence: Diagnostic accuracy studies, fair, direct).

Intrauterine Contraception

Three IUDs are available in the United States, the Cu-IUD and two LNG-IUDs (containing a total of either 13.5 mg or 52 mg levonorgestrel). Fewer than 1 woman out of 100 becomes pregnant in the first year of using IUDs (with typical use) (*59*). IUDs are long acting, are reversible, and can be

used by women of all ages, including adolescents, and both by parous and nulliparous women. IUDs do not protect against STDs; consistent and correct use of male latex condoms reduces the risk for STDs, including HIV.

Initiation of Cu-IUDs

Timing

- The Cu-IUD can be inserted at any time if it is reasonably certain that the woman is not pregnant (Box 1).
- The Cu-IUD also can be inserted within 5 days of the first act of unprotected sexual intercourse as an emergency contraceptive. If the day of ovulation can be estimated, the Cu-IUD also can be inserted >5 days after sexual intercourse as long as insertion does not occur >5 days after ovulation.

Need for Back-Up Contraception

- No additional contraceptive protection is needed after Cu-IUD insertion.

Special Considerations

Amenorrhea (Not Postpartum)

- **Timing:** The Cu-IUD can be inserted at any time if it is reasonably certain that the woman is not pregnant (Box 1).
- **Need for back-up contraception:** No additional contraceptive protection is needed.

Postpartum (Including After Cesarean Section)

- **Timing:** The Cu-IUD can be inserted at any time postpartum, including immediately postpartum (U.S. MEC 1 or 2) (Box 2), if it is reasonably certain that the woman is not pregnant (Box 1). The Cu-IUD should not be inserted in a woman with puerperal sepsis (U.S. MEC 4).
- **Need for back-up contraception:** No additional contraceptive protection is needed.

Postabortion (Spontaneous or Induced)

- **Timing:** The Cu-IUD can be inserted within the first 7 days, including immediately postabortion (U.S. MEC 1 for first trimester abortion and U.S. MEC 2 for second trimester abortion). The Cu-IUD should not be inserted immediately after septic abortion (U.S. MEC 4).
- **Need for back-up contraception:** No additional contraceptive protection is needed.

Switching from Another Contraceptive Method

- **Timing:** The Cu-IUD can be inserted immediately if it is reasonably certain that the woman is not pregnant (Box 1). Waiting for her next menstrual period is unnecessary.
- **Need for back-up contraception:** No additional contraceptive protection is needed.

Comments and Evidence Summary. In situations in which the health-care provider is not reasonably certain that the woman is not pregnant, the woman should be provided with another contraceptive method to use until the health-care provider can be reasonably certain that she is not pregnant and can insert the Cu-IUD.

A systematic review identified eight studies that suggested that timing of Cu-IUD insertion in relation to the menstrual cycle in nonpostpartum women had little effect on long-term outcomes (rates of continuation, removal, expulsion, or pregnancy) or on short-term outcomes (pain at insertion, bleeding at insertion, or immediate expulsion) (*13*) (Level of evidence: II-2, fair, direct).

BOX 2. Categories of medical eligibility criteria for contraceptive use

U.S. MEC 1 = A condition for which there is no restriction for the use of the contraceptive method.

U.S. MEC 2 = A condition for which the advantages of using the method generally outweigh the theoretical or proven risks.

U.S. MEC 3 = A condition for which the theoretical or proven risks usually outweigh the advantages of using the method.

U.S. MEC 4 = A condition that represents an unacceptable health risk if the contraceptive method is used.

Abbreviations: U.S. MEC = *U.S. Medical Eligibility Criteria for Contraceptive Use, 2010.*
Source: CDC. U.S. medical eligibility criteria for contraceptive use. MMWR 2010;59(No. RR-4).

Initiation of LNG-IUDs

Timing of LNG-IUD Insertion

- The LNG-IUD can be inserted at any time if it is reasonably certain that the woman is not pregnant (Box 1).

Need for Back-Up Contraception

- If the LNG-IUD is inserted within the first 7 days since menstrual bleeding started, no additional contraceptive protection is needed.
- If the LNG-IUD is inserted >7 days since menstrual bleeding started, the woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.

Special Considerations

Amenorrhea (Not Postpartum)

- **Timing:** The LNG-IUD can be inserted at any time if it is reasonably certain that the woman is not pregnant (Box 1).
- **Need for back-up contraception:** The woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.

Postpartum (Including After Cesarean Section)

- **Timing:** The LNG-IUD can be inserted at any time, including immediately postpartum (U.S. MEC 1 or 2) if it is reasonably certain that the woman is not pregnant (Box 1). The LNG-IUD should not be inserted in a woman with puerperal sepsis (U.S. MEC 4).
- **Need for back-up contraception:** If the woman is <6 months postpartum, amenorrheic, and fully or nearly fully breastfeeding (exclusively breastfeeding or the vast majority [≥85%] of feeds are breastfeeds) (*60*), no additional contraceptive protection is needed. Otherwise, a woman who is ≥21 days postpartum and has not experienced return of her menstrual cycle needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days. If her menstrual cycles have returned and it has been >7 days since menstrual bleeding began, she needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.

Postabortion (Spontaneous or Induced)

- **Timing:** The LNG-IUD can be inserted within the first 7 days, including immediately postabortion (U.S. MEC 1 for first-trimester abortion and U.S. MEC 2 for second-trimester abortion). The LNG-IUD should not be inserted immediately after a septic abortion (U.S. MEC 4).
- **Need for back-up contraception:** The woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days unless the IUD is placed at the time of a surgical abortion.

Switching from Another Contraceptive Method

- **Timing:** The LNG-IUD can be inserted immediately if it is reasonably certain that the woman is not pregnant (Box 1). Waiting for her next menstrual period is unnecessary.
- **Need for back-up contraception:** If it has been >7 days since menstrual bleeding began, the woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.
- **Switching from a Cu-IUD:** If the woman has had sexual intercourse since the start of her current menstrual cycle and it has been >5 days since menstrual bleeding started, theoretically, residual sperm might be in the genital tract, which could lead to fertilization if ovulation occurs. A health-care provider can consider providing ECPs at the time of LNG-IUD insertion.

Comments and Evidence Summary. In situations in which the health-care provider is uncertain whether the woman might be pregnant, the woman should be provided with another contraceptive method to use until the health-care provider can be reasonably certain that she is not pregnant and can insert the LNG-IUD. If a woman needs to use additional contraceptive protection when switching to an LNG-IUD from another contraceptive method, consider continuing her previous method for 7 days after LNG-IUD insertion. No direct evidence was found regarding the effects of inserting LNG-IUDs on different days of the cycle on short- or long-term outcomes (*13*).

Examinations and Tests Needed Before Initiation of a Cu-IUD or an LNG-IUD

Among healthy women, few examinations or tests are needed before initiation of an IUD (Table 2). Bimanual examination and cervical inspection are necessary before IUD insertion. A baseline weight and BMI measurement might be useful for monitoring IUD users over time. If a woman has not been screened for STDs according to STD screening guidelines, screening can be performed at the time of insertion. Women with known medical problems or other special conditions might need additional examinations or tests before being determined to be appropriate candidates for a particular method of contraception. U.S. MEC might be useful in such circumstances (*5*).

Comments and Evidence Summary. Weight (BMI): Obese women can use IUDs (U.S. MEC 1) (*5*); therefore, screening for obesity is not necessary for the safe initiation of IUDs. However, measuring weight and calculating BMI (weight [kg] / height [m]^2) at baseline might be helpful for monitoring any changes and counseling women who might be concerned about weight change perceived to be associated with their contraceptive method.

Bimanual examination and cervical inspection: Bimanual examination and cervical inspection are necessary before IUD insertion to assess uterine size and position and to detect any cervical or uterine abnormalities that might indicate infection or otherwise prevent IUD insertion (*61*,*62*).

STDs: Women should be routinely screened for chlamydial infection and gonorrhea according to national screening guidelines. The CDC *Sexually Transmitted Diseases Treatment Guidelines* provide information on screening eligibility, timing, and frequency of screening and on screening for persons

TABLE 2. Classification of examinations and tests needed before IUD insertion

Examination or test	Class*	
	Copper-containing IUD	Levonorgestrel-releasing IUD
Examinations		
Blood pressure	C	C
Weight (BMI) (weight [kg]/height [m]^2)	—†	—†
Clinical breast examination	C	C
Bimanual examination and cervical inspection	A	A
Laboratory tests		
Glucose	C	C
Lipids	C	C
Liver enzymes	C	C
Hemoglobin	C	C
Thrombogenic mutations	C	C
Cervical cytology (Papanicolaou smear)	C	C
STD screening with laboratory tests	—§	—§
HIV screening with laboratory tests	C	C

Abbreviations: BMI = body mass index; HIV = human immunodeficiency virus; IUD = intrauterine device; STD = sexually transmitted disease; U.S. MEC = *U.S. Medical Eligibility Criteria for Contraceptive Use, 2010*.

* **Class A:** essential and mandatory in all circumstances for safe and effective use of the contraceptive method. **Class B:** contributes substantially to safe and effective use, but implementation may be considered within the public health and/or service context; the risk of not performing an examination or test should be balanced against the benefits of making the contraceptive method available. **Class C:** does not contribute substantially to safe and effective use of the contraceptive method.

† Weight (BMI) measurement is not needed to determine medical eligibility for any methods of contraception because all methods can be used (U.S. MEC 1) or generally can be used (U.S. MEC 2) among obese women (Box 2). However, measuring weight and calculating BMI at baseline might be helpful for monitoring any changes and counseling women who might be concerned about weight change perceived to be associated with their contraceptive method.

§ Most women do not require additional STD screening at the time of IUD insertion if they have already been screened according to CDC's *STD Treatment Guidelines* (available at http://www.cdc.gov/std/treatment). If a woman has not been screened according to guidelines, screening can be performed at the time of IUD insertion, and insertion should not be delayed. Women with purulent cervicitis or current chlamydial infection or gonorrhea should not undergo IUD insertion (U.S. MEC 4). Women who have a very high individual likelihood of STD exposure (e.g., those with a currently infected partner) generally should not undergo IUD insertion (U.S. MEC 3). For these women, IUD insertion should be delayed until appropriate testing and treatment occur.

with risk factors (*32*). If STD screening guidelines have been followed, most women do not need additional STD screening at the time of IUD insertion. If a woman has not been screened according to guidelines, screening can be performed at the time of IUD insertion and insertion should not be delayed. Women with purulent cervicitis or current chlamydial infection or gonorrhea should not undergo IUD insertion (U.S. MEC 4). Women who have a very high individual likelihood of STD exposure (e.g., those with a currently infected partner) generally should not undergo IUD insertion (U.S. MEC 3) (*5*). For these women, IUD insertion should be delayed until appropriate testing and treatment occur. A systematic review did not identify any evidence regarding women who were screened versus not screened for STDs before IUD insertion (*14*). Although women with STDs at the time of IUD insertion have a higher risk for PID, the overall rate of PID among all IUD users is low (*63*,*64*).

Hemoglobin: Women with iron-deficiency anemia can use the LNG-IUD (U.S. MEC 1) (*5*); therefore, screening for anemia is not necessary for safe initiation of the LNG-IUD. Women with iron-deficiency anemia generally can use the Cu-IUD (U.S. MEC 2). Measurement of hemoglobin before initiation of Cu-IUDs is not necessary because of the minimal change in hemoglobin among women with and without anemia using Cu-IUDs. A systematic review identified four studies that provided direct evidence for changes in hemoglobin among women with anemia who received Cu-IUDs (*30*). Evidence from one randomized trial (*65*) and one prospective cohort study (*66*) showed no significant changes in hemoglobin among Cu-IUD users with anemia, whereas two prospective cohort studies (*67*,*68*) showed a statistically significant decrease in hemoglobin levels during 12 months of follow-up; however, the magnitude of the decrease was small and most likely not clinically significant. The systematic review also identified 21 studies that provided indirect evidence by examining changes in hemoglobin among healthy women receiving Cu-IUDs (*69*–*89*), which generally showed no clinically significant changes in hemoglobin levels with up to 5 years of follow-up (Level of evidence: I to II-2, fair, direct).

Liver enzymes: Women with liver disease can use the Cu-IUD (U.S. MEC 1) (*5*); therefore, screening for liver disease is not necessary for the safe initiation of the Cu-IUD. Although women with certain liver diseases generally should not use the LNG-IUD (U.S. MEC 3) (*5*), screening for liver disease before initiation of the LNG-IUD is not necessary because of the low prevalence of these conditions and the high likelihood that women with liver disease already would have had the condition diagnosed. A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened with liver enzyme tests before initiation of hormonal contraceptive use (*14*). The prevalence of liver disorders among women of reproductive age is low. In 2008, among adults aged 18–44 years, the percentage with liver disease (not further specified) was 1.0% (*90*). In 2009, the incidence of acute hepatitis A, B, or C among women was <1 per 100,000 population (*91*). During 1998–2007, the incidence of liver carcinoma among women was approximately 3 per 100,000 population (*92*). Because estrogen and progestins are metabolized in the liver, the use of hormonal contraceptives among women with liver disease

might, theoretically, be a concern. The use of hormonal contraceptives, specifically COCs and POPs, does not affect disease progression or severity in women with hepatitis, cirrhosis, or benign focal nodular hyperplasia (*93*,*94*), although evidence is limited, and no evidence exists for the LNG-IUD.

Clinical breast examination: Women with breast disease can use the Cu-IUD (U.S. MEC 1) (*5*); therefore, screening for breast disease is not necessary for the safe initiation of the Cu-IUD. Although women with current breast cancer should not use the LNG-IUD (U.S. MEC 4) (*5*), screening asymptomatic women with a clinical breast examination before inserting an IUD is not necessary because of the low prevalence of breast cancer among women of reproductive age. A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened with a breast examination before initiation of hormonal contraceptives (*15*). The incidence of breast cancer among women of reproductive age in the United States is low. In 2009, the incidence of breast cancer among women aged 20–49 years was approximately 72 per 100,000 women (*95*).

Cervical cytology: Although women with cervical cancer should not undergo IUD insertion (U.S. MEC 4) (*5*), screening asymptomatic women with cervical cytology before IUD insertion is not necessary because of the high rates of cervical screening, low incidence of cervical cancer in the United States, and high likelihood that a woman with cervical cancer already would have had the condition diagnosed. A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened with cervical cytology before initiation of IUDs (*14*). Cervical cancer is rare in the United States, with an incidence rate of 8.1 per 100,000 women per year during 2004–2008 (*95*). The incidence and mortality rates from cervical cancer have declined dramatically in the United States, largely because of cervical cytology screening (*96*). Overall screening rates for cervical cancer in the United States are high; among women aged 22–30 years, approximately 87% reported having cervical cytology screening within the last 3 years (*97*).

HIV screening: Although women with acquired immunodeficiency syndrome (AIDS) who are not clinically well should generally not undergo IUD insertion (U.S. MEC 3) (*5*), HIV screening is not necessary before IUD insertion because of the high likelihood that a woman in the United States with such an advanced stage of disease already would have had the condition diagnosed. A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened for HIV infection before IUD insertion (*14*). Limited evidence suggests that IUDs are not associated with disease progression, increased infection, or other adverse health effects among women with HIV infection (*98*).

Other screening: Women with hypertension, diabetes, hyperlipidemia, or thrombogenic mutations can use (U.S. MEC 1) or generally can use (U.S. MEC 2) IUDs (*5*). Therefore, screening for these conditions is not necessary for the safe initiation of IUDs.

Provision of Prophylactic Antibiotics at the Time of IUD Insertion

- Prophylactic antibiotics are generally not recommended for Cu-IUD or LNG-IUD insertion.

Comments and Evidence Summary. Theoretically, IUD insertion could induce bacterial spread and lead to complications such as PID or infective endocarditis. A metaanalysis was conducted of randomized controlled trials examining antibiotic prophylaxis versus placebo or no treatment for IUD insertion (*99*). Use of prophylaxis reduced the frequency of unscheduled return visits but did not significantly reduce the incidence of PID or premature IUD discontinuation. Although the risk for PID was higher within the first 20 days after insertion, the incidence of PID was low among all women who had IUDs inserted (*63*). In addition, the American Heart Association recommends that the use of prophylactic antibiotics solely to prevent infective endocarditis is not needed for genitourinary procedures (*100*). Studies have not demonstrated a conclusive link between genitourinary procedures and infective endocarditis or a preventive benefit of prophylactic antibiotics during such procedures (*100*).

Routine Follow-Up After IUD Insertion

These recommendations address when routine follow-up is needed for safe and effective continued use of contraception for healthy women. The recommendations refer to general situations and might vary for different users and different situations. Specific populations that might benefit from more frequent follow-up visits include adolescents, persons with certain medical conditions or characteristics, and persons with multiple medical conditions.

- Advise a woman to return at any time to discuss side effects or other problems, if she wants to change the method being used, and when it is time to remove or replace the contraceptive method. No routine follow-up visit is required.
- At other routine visits, health-care providers who see IUD users should do the following:
  - Assess the woman's satisfaction with her contraceptive method and whether she has any concerns about method use.

  - Assess any changes in health status, including medications, that would change the appropriateness of the IUD for safe and effective continued use on the basis of U.S. MEC (e.g., category 3 and 4 conditions and characteristics).
  - Consider performing an examination to check for the presence of the IUD strings.
  - Consider assessing weight changes and counseling women who are concerned about weight changes perceived to be associated with their contraceptive method.

Comments and Evidence Summary. Evidence from a systematic review about the effect of a specific follow-up visit schedule on IUD continuation is very limited and of poor quality. The evidence did not suggest that greater frequency of visits or earlier timing of the first follow-up visit after insertion improves continuation of use (*16*) (Level of evidence: II-2, poor, direct). Evidence from four studies from a systematic review on the incidence of PID among IUD initiators, or IUD removal as a result of PID, suggested that the incidence of PID did not differ between women using Cu-IUDs and those using DMPA, COCs, or LNG-IUDs (*17*) (Level of evidence: I to II-2, good, indirect). Evidence on the timing of PID after IUD insertion is mixed. Although the rate of PID was generally low, the largest study suggested that the rate of PID was significantly higher in the first 20 days after insertion (*63*) (Level of evidence: I to II-3, good to poor, indirect).

Bleeding Irregularities with Cu-IUD Use

- Before Cu-IUD insertion, provide counseling about potential changes in bleeding patterns during Cu-IUD use. Unscheduled spotting or light bleeding, as well as heavy or prolonged bleeding, is common during the first 3–6 months of Cu-IUD use, is generally not harmful, and decreases with continued Cu-IUD use.
- If clinically indicated, consider an underlying gynecological problem, such as Cu-IUD displacement, an STD, pregnancy, or new pathologic uterine conditions (e.g., polyps or fibroids), especially in women who have already been using the Cu-IUD for a few months or longer and who have developed a new onset of heavy or prolonged bleeding. If an underlying gynecological problem is found, treat the condition or refer for care.
- If an underlying gynecological problem is not found and the woman requests treatment, the following treatment option can be considered during days of bleeding:
  - Nonsteroidal antiinflammatory drugs (NSAIDs) for short-term treatment (5–7 days)
- If bleeding persists and the woman finds it unacceptable, counsel her on alternative contraceptive methods, and offer another method if it is desired.

Comments and Evidence Summary. During contraceptive counseling and before insertion of the Cu-IUD, information about common side effects such as unscheduled spotting or light bleeding or heavy or prolonged menstrual bleeding, especially during the first 3–6 months of use, should be discussed (*70*). These bleeding irregularities are generally not harmful. Enhanced counseling about expected bleeding patterns and reassurance that bleeding irregularities are generally not harmful has been shown to reduce method discontinuation in clinical trials with other contraceptives (i.e., DMPA) (*101*,*102*).

Evidence is limited on specific drugs, doses, and durations of use for effective treatments for bleeding irregularities with Cu-IUD use; therefore, although this document includes general recommendations for treatments to consider, evidence for specific regimens is lacking.

A systematic review identified 11 articles that examined various therapeutic treatments for heavy menstrual bleeding, prolonged menstrual bleeding, or both among women using Cu-IUDs (*18*). Nine studies examined the use of various oral NSAIDs for the treatment of heavy or prolonged menstrual bleeding among Cu-IUD users and compared them to either a placebo or a baseline cycle. Three of these trials examined the use of indomethacin (*103–105*), another three examined mefenamic acid (*106–108*), and another three examined flufenamic acid (*103*,*104*,*109*). Other NSAIDs used in the reported trials included alclofenac (*103*,*104*), suprofen (*110*), and diclofenac sodium (*111*). All but one NSAID study (*107*) demonstrated statistically significant or notable reductions in mean total menstrual blood loss with NSAID use. One study among 19 Cu-IUD users with heavy bleeding suggested that treatment with oral tranexamic acid can significantly reduce mean blood loss during treatment compared with placebo (*111*). Data regarding the overall safety of tranexamic acid are limited; an FDA warning states that tranexamic acid is contraindicated in women with active thromboembolic disease or with a history or intrinsic risk for thrombosis or thromboembolism (*112*,*113*). Treatment with aspirin demonstrated no statistically significant change in mean blood loss among women whose pretreatment menstrual blood loss was >80 mL or 60–80 mL; treatment resulted in a significant increase among women whose pretreatment menstrual blood loss was <60 mL (*114*). One study examined the use of a synthetic form of vasopressin, intranasal desmopressin (300 μg/day), for the first 5 days of menses for three treatment cycles and found a significant reduction in mean blood loss

compared with baseline (*106*) (Level of evidence: I to II-3, poor to fair, direct). Only one small study examined treatment of spotting with three separate NSAIDs and did not observe improvements in spotting in any of the groups (*103*) (Level of evidence: I, poor, direct).

Bleeding Irregularities (Including Amenorrhea) with LNG-IUD Use

- Before LNG-IUD insertion, provide counseling about potential changes in bleeding patterns during LNG-IUD use. Unscheduled spotting or light bleeding is expected during the first 3–6 months of LNG-IUD use, is generally not harmful, and decreases with continued LNG-IUD use. Over time, bleeding generally decreases with LNG-IUD use, and many women experience only light menstrual bleeding or amenorrhea. Heavy or prolonged bleeding, either unscheduled or menstrual, is uncommon during LNG-IUD use.

Irregular Bleeding (Spotting, Light Bleeding, or Heavy or Prolonged Bleeding)

- If clinically indicated, consider an underlying gynecological problem, such as LNG-IUD displacement, an STD, pregnancy, or new pathologic uterine conditions (e.g., polyps or fibroids). If an underlying gynecological problem is found, treat the condition or refer for care.
- If bleeding persists and the woman finds it unacceptable, counsel her on alternative contraceptive methods, and offer another method if it is desired.

Amenorrhea

- Amenorrhea does not require any medical treatment. Provide reassurance.
  - If a woman's regular bleeding pattern changes abruptly to amenorrhea, consider ruling out pregnancy if clinically indicated.
- If amenorrhea persists and the woman finds it unacceptable, counsel her on alternative contraceptive methods, and offer another method if it is desired

Comments and Evidence Summary. During contraceptive counseling and before insertion of the LNG-IUD, information about common side effects such as unscheduled spotting or light bleeding, especially during the first 3–6 months of use, should be discussed. Approximately half of LNG-IUD users are likely to experience amenorrhea or oligomenorrhea by 2 years of use (*115*). These bleeding irregularities are generally not harmful. Enhanced counseling about expected bleeding patterns and reassurance that bleeding irregularities are generally not harmful has been shown to reduce method discontinuation in clinical trials with other hormonal contraceptives (i.e., DMPA) (*101*,*102*). No direct evidence was found regarding therapeutic treatments for bleeding irregularities during LNG-IUD use.

Management of the IUD when a Cu-IUD or an LNG-IUD User Is Found To Have PID

- Treat the PID according to the CDC *Sexually Transmitted Diseases Treatment Guidelines* (*32*).
- Provide comprehensive management for STDs, including counseling about condom use.
- The IUD does not need to be removed immediately if the woman needs ongoing contraception.
- Reassess the woman in 48–72 hours. If no clinical improvement occurs, continue antibiotics and consider removal of the IUD.
- If the woman wants to discontinue use, remove the IUD sometime after antibiotics have been started to avoid the potential risk for bacterial spread resulting from the removal procedure.
- If the IUD is removed, consider ECPs if appropriate. Counsel the woman on alternative contraceptive methods, and offer another method if it is desired.
- A summary of IUD management in women with PID is provided (Appendix F).

Comments and Evidence Summary. Treatment outcomes do not generally differ between women with PID who retain the IUD and those who have the IUD removed; however, appropriate antibiotic treatment and close clinical follow-up are necessary.

A systematic review identified four studies that included women using copper or nonhormonal IUDs who developed PID and compared outcomes between women who had the IUD removed or did not (*19*). One randomized trial showed that women with IUDs removed had longer hospitalizations than those who did not, although no differences in PID recurrences or subsequent pregnancies were observed (*116*). Another randomized trial showed no differences in laboratory findings among women who removed the IUD compared with those who did not (*117*). One prospective cohort study showed no differences in clinical or laboratory findings during hospitalization; however, the IUD removal group had longer hospitalizations (*118*). One randomized trial showed that the rate of recovery for most clinical signs and symptoms was higher among women who had the IUD removed than among women who did not (*119*). No evidence was found regarding women using LNG-IUDs (Level of evidence: I to II-2, fair, direct).

Management of the IUD when a Cu-IUD or an LNG-IUD User Is Found To Be Pregnant

- Evaluate for possible ectopic pregnancy.
- Advise the woman that she has an increased risk for spontaneous abortion (including septic abortion that might be life threatening) and of preterm delivery if the IUD is left in place. The removal of the IUD reduces these risks but might not decrease the risk to the baseline level of a pregnancy without an IUD.
  - If she does not want to continue the pregnancy, counsel her about options.
  - If she wants continue the pregnancy, advise her to seek care promptly if she has heavy bleeding, cramping, pain, abnormal vaginal discharge, or fever.

IUD Strings Are Visible or Can Be Retrieved Safely from the Cervical Canal

- Advise the woman that the IUD should be removed as soon as possible.
  - If the IUD is to be removed, remove it by pulling on the strings gently.
  - Advise the woman that she should return promptly if she has heavy bleeding, cramping, pain, abnormal vaginal discharge, or fever.
- If she chooses to keep the IUD, advise her to seek care promptly if she has heavy bleeding, cramping, pain, abnormal vaginal discharge, or fever.

IUD Strings Are Not Visible and Cannot Be Retrieved Safely

- If ultrasonography is available, consider performing or referring for ultrasound examination to determine the location of the IUD. If the IUD cannot be located, it might have been expelled or have perforated the uterine wall.
- If ultrasonography is not possible or the IUD is determined by ultrasound to be inside the uterus, advise the woman to seek care promptly if she has heavy bleeding, cramping, pain, abnormal vaginal discharge, or fever.

Comments and Evidence Summary. Removing the IUD improves the pregnancy outcome if the IUD strings are visible or the device can be retrieved safely from the cervical canal. Risks for spontaneous abortion, preterm delivery, and infection are substantial if the IUD is left in place.

Theoretically, the fetus might be affected by hormonal exposure from an LNG-IUD; however, whether this exposure increases the risk for fetal abnormalities is unknown.

A systematic review identified nine studies suggesting that women who did not remove their IUDs during pregnancy were at greater risk for adverse pregnancy outcomes (including spontaneous abortion, septic abortion, preterm delivery, and chorioamnionitis) compared with women who had their IUDs removed or who did not have an IUD (*58*). Cu-IUD removal decreased risks but not to the baseline risk for pregnancies without an IUD. One case series examined LNG-IUDs. When they were not removed, eight in 10 pregnancies ended in spontaneous abortions (Level of evidence: II-2, fair, direct).

Implants

The etonogestrel implant, a single rod with 68 mg of etonogestrel, is available in the United States. Fewer than 1 woman out of 100 become pregnant in the first year of use of the etonogestrel implant with typical use (*59*). The implant is long acting, is reversible, and can be used by women of all ages, including adolescents. The implant does not protect against STDs; consistent and correct use of male latex condoms reduces the risk for STDs, including HIV.

Initiation of Implants

Timing

- The implant can be inserted at any time if it is reasonably certain that the woman is not pregnant (Box 1).

Need for Back-Up Contraception

- If the implant is inserted within the first 5 days since menstrual bleeding started, no additional contraceptive protection is needed.
- If the implant is inserted >5 days since menstrual bleeding started, the woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.

Special Considerations

Amenorrhea (Not Postpartum)

- **Timing:** The implant can be inserted at any time if it is reasonably certain that the woman is not pregnant (Box 1).
- **Need for back-up contraception:** The woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.

Postpartum (Breastfeeding)

- **Timing:** The implant can be inserted at any time (U.S. MEC 2 if <1 month postpartum and U.S. MEC 1 if ≥1 month postpartum) if it is reasonably certain that the woman is not pregnant (Box 1).
- **Need for back-up contraception:** If the woman is <6 months postpartum, amenorrheic, and fully or nearly fully breastfeeding (exclusively breastfeeding or the vast majority [≥85%] of feeds

are breastfeeds) (*60*), no additional contraceptive protection is needed. Otherwise, a woman who is ≥21 days postpartum and has not experienced return of her menstrual cycle needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days. If her menstrual cycles have returned and it has been >5 days since menstrual bleeding started, she needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.

Postpartum (Not Breastfeeding)

- **Timing:** The implant can be inserted at any time, including immediately postpartum (U.S. MEC 1) if it is reasonably certain that the woman is not pregnant (Box 1).
- **Need for back-up contraception:** A woman who is ≥21 days postpartum and has not experienced return of her menstrual cycle needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days. If her menstrual cycles have returned and it has been >5 days since menstrual bleeding started, she needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.

Postabortion (Spontaneous or Induced)

- **Timing:** The implant can be inserted within the first 7 days, including immediately after the abortion (U.S. MEC 1).
- **Need for back-up contraception:** The woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days unless the implant is placed at the time of a surgical abortion.

Switching from Another Contraceptive Method

- **Timing:** The implant can be inserted immediately if it is reasonably certain that the woman is not pregnant (Box 1). Waiting for her next menstrual period is unnecessary.
- **Need for back-up contraception:** If it has been >5 days since menstrual bleeding started, the woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days after insertion.
- **Switching from an IUD:** If the woman has had sexual intercourse since the start of her current menstrual cycle and it has been >5 days since menstrual bleeding started, theoretically, residual sperm might be in the genital tract, which could lead to fertilization if ovulation occurs. A health-care provider may consider any of the following options:
  - Advise the woman to retain the IUD for at least 7 days after the implant is inserted and return for IUD removal.
  - Advise the woman to abstain from sexual intercourse or use barrier contraception for 7 days before removing the IUD and switching to the new method.
  - Advise the woman to use ECPs at the time of IUD removal.

Comments and Evidence Summary. In situations in which the health-care provider is uncertain whether the woman might be pregnant, the benefits of starting the implant likely exceed any risk; therefore, starting the implant should be considered at any time, with a follow-up pregnancy test in 2–4 weeks.

If a woman needs to use additional contraceptive protection when switching to an implant from another contraceptive method, consider continuing her previous method for 7 days after implant insertion. No direct evidence was found regarding the effects of starting the etonogestrel implant at different times of the cycle.

Examinations and Tests Needed Before Implant Insertion

Among healthy women, no examinations or tests are needed before initiation of an implant, although a baseline weight and BMI measurement might be useful for monitoring implant users over time (Table 3). Women with known medical problems or other special conditions might need additional examinations or tests before being determined to be appropriate candidates for a particular method of contraception. U.S. MEC might be useful in such circumstances (*5*).

TABLE 3. Classification of examinations and tests needed before implant insertion

Examination or test	Class*
Examination	
Blood pressure	C
Weight (BMI) (weight [kg]/height [m]^2)	—†
Clinical breast examination	C
Bimanual examination and cervical inspection	C
Laboratory test	
Glucose	C
Lipids	C
Liver enzymes	C
Hemoglobin	C
Thrombogenic mutations	C
Cervical cytology (Papanicolaou smear)	C
STD screening with laboratory tests	C
HIV screening with laboratory tests	C

Abbreviations: BMI = body mass index; HIV = human immunodeficiency virus; STD = sexually transmitted disease; U.S. MEC = *U.S. Medical Eligibility Criteria for Contraceptive Use, 2010*.

* **Class A:** essential and mandatory in all circumstances for safe and effective use of the contraceptive method. **Class B:** contributes substantially to safe and effective use, but implementation may be considered within the public health and/or service context; the risk of not performing an examination or test should be balanced against the benefits of making the contraceptive method available. **Class C:** does not contribute substantially to safe and effective use of the contraceptive method.

† Weight (BMI) measurement is not needed to determine medical eligibility for any methods of contraception because all methods can be used (U.S. MEC 1) or generally can be used (U.S. MEC 2) among obese women (Box 2). However, measuring weight and calculating BMI at baseline might be helpful for monitoring any changes and counseling women who might be concerned about weight change perceived to be associated with their contraceptive method.

Comments and Evidence Summary. Weight (BMI): Obese women can use implants (U.S. MEC 1) (*5*); therefore, screening for obesity is not necessary for the safe initiation of implants. However, measuring weight and calculating BMI at baseline might be helpful for monitoring any changes and counseling women who might be concerned about weight change perceived to be associated with their contraceptive method.

Bimanual examination and cervical inspection: A pelvic examination is not necessary before initiation of implants because it would not facilitate detection of conditions for which implant use would be unsafe. Women with current breast cancer should not use implants (U.S. MEC 4); women with certain liver diseases generally should not use implants (U.S. MEC 3) (*5*). However, none of these conditions are likely to be detected by pelvic examination (*120*). A systematic review identified two case-control studies that compared delayed and immediate pelvic examination before initiation of hormonal contraceptives, specifically oral contraceptives or DMPA (*15*). No differences in risk factors for cervical neoplasia, incidence of STDs, incidence of abnormal Papanicolaou smears, or incidence of abnormal wet mounts were observed. No evidence was found regarding implants (Level of evidence: II-2 fair, direct).

Liver enzymes: Although women with certain liver diseases generally should not use implants (U.S. MEC 3) (*5*), screening for liver disease before initiation of implants is not necessary because of the low prevalence of these conditions and the high likelihood that women with liver disease already would have had the condition diagnosed. A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened with liver enzyme tests before initiation of hormonal contraceptives (*14*). The prevalence of liver disorders among women of reproductive age is low. In 2008, the percentage of adults aged 18–44 years with liver disease (not further specified) was 1.0% (*90*). In 2009, the incidence of acute hepatitis A, B, or C among women was <1 per 100,000 population (*91*). During 1998–2007, the incidence of liver carcinoma among women was approximately 3 per 100,000 population (*92*). Because estrogen and progestins are metabolized in the liver, the use of hormonal contraceptives among women with liver disease might, theoretically, be a concern. The use of hormonal contraceptives, specifically COCs and POPs, does not affect disease progression or severity in women with hepatitis, cirrhosis, or benign focal nodular hyperplasia (*93*,*94*), although evidence is limited and no evidence exists for implants.

Clinical breast examination: Although women with current breast cancer should not use implants (U.S. MEC 4) (*5*), screening asymptomatic women with a clinical breast examination before initiating an implant is not necessary because of the low prevalence of breast cancer among women of reproductive age (15–49 years). A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened with a breast examination before initiation of hormonal contraceptives (*15*). The incidence of breast cancer among women of reproductive age in the United States is low. In 2009, the incidence of breast cancer among women aged 20–49 years was approximately 72 per 100,000 women (*95*).

Other screening: Women with hypertension, diabetes, hyperlipidemia, anemia, thrombogenic mutations, cervical intraepithelial neoplasia, cervical cancer, STDs, or HIV infection can use (U.S. MEC 1) or generally can use (U.S. MEC 2) implants (*5*); therefore, screening for these conditions is not necessary for the safe initiation of implants.

Routine Follow-Up After Implant Insertion

These recommendations address when routine follow-up is needed for safe and effective continued use of contraception for healthy women. The recommendations refer to general situations and might vary for different users and different situations. Specific populations that might benefit from more frequent follow-up visits include adolescents, those with certain medical conditions or characteristics, and those with multiple medical conditions.

- Advise a woman to return at any time to discuss side effects or other problems, if she wants to change the method being used, and when it is time to remove or replace the contraceptive method. No routine follow-up visit is required.
- At other routine visits, health-care providers seeing implant users should do the following:
  - Assess the woman's satisfaction with her contraceptive method and whether she has any concerns about method use.
  - Assess any changes in health status, including medications, that would change the appropriateness of the implant for safe and effective continued use based on U.S. MEC (e.g., category 3 and 4 conditions and characteristics).
  - Consider assessing weight changes and counseling women who are concerned about weight changes perceived to be associated with their contraceptive method.

Comments and Evidence Summary. A systematic review did not identify any evidence regarding whether a routine follow-up visit after initiating an implant improves correct or continued use (*16*).

Bleeding Irregularities (Including Amenorrhea) During Implant Use

- Before implant insertion, provide counseling about potential changes in bleeding patterns during implant use. Unscheduled spotting or light bleeding is common with implant use, and some women experience amenorrhea. These bleeding changes are generally not harmful and might or might not decrease with continued implant use. Heavy or prolonged bleeding, unscheduled or menstrual, is uncommon during implant use.

Irregular Bleeding (Spotting, Light Bleeding, or Heavy or Prolonged Bleeding)

- If clinically indicated, consider an underlying gynecological problem, such as interactions with other medications, an STD, pregnancy, or new pathologic uterine conditions (e.g., polyps or fibroids). If an underlying gynecological problem is found, treat the condition or refer for care.
- If an underlying gynecologic problem is not found and the woman wants treatment, the following treatment options during days of bleeding can be considered:
  - NSAIDS for short-term treatment (5–7 days)
  - Hormonal treatment (if medically eligible) with low-dose COCs or estrogen for short-term treatment (10–20 days)
- If irregular bleeding persists and the woman finds it unacceptable, counsel her on alternative methods, and offer another method if it is desired.

Amenorrhea

- Amenorrhea does not require any medical treatment. Provide reassurance.
  - If a woman's regular bleeding pattern changes abruptly to amenorrhea, consider ruling out pregnancy if clinically indicated.
- If amenorrhea persists and the woman finds it unacceptable, counsel her on alternative contraceptive methods, and offer another method if it is desired.

Comments and Evidence Summary. During contraceptive counseling and before insertion of the implant, information about common side effects, such as unscheduled spotting or light bleeding and amenorrhea, especially during the first year of use should be discussed. A pooled analysis of data from 11 clinical trials indicate that a significant proportion of etonogestrel implant users had relatively little bleeding: 22% of women experienced amenorrhea and 34% experienced infrequent spotting, although 7% reported frequent bleeding and 18% reported prolonged bleeding (*121*). Unscheduled bleeding or amenorrhea is generally not harmful. Enhanced counseling about expected bleeding patterns and reassurance that bleeding irregularities are generally not harmful has been shown to reduce discontinuation in clinical trials with other hormonal contraceptives (i.e., DMPA) (*101*,*102*).

A systematic review and four newly published studies examined several medications for the treatment of bleeding irregularities with primarily LNG contraceptive implants (*122*–*126*). Two small studies found significant cessation of bleeding within 7 days of start of treatment among women taking oral celecoxib (200 mg) daily for 5 days or oral mefenamic acid (500 mg) 3 times daily for 5 days compared with placebo (*124*,*125*). Differences in bleeding cessation were not found among women with etonogestrel implants taking mifepristone but were found when women with the implants combined mifepristone with either ethinyl estradiol or doxycycline (*126*,*127*). Doxycycline alone or in combination with ethinyl estradiol did not improve bleeding cessation among etonogestrel implant users (*126*). Among LNG implant users, mifepristone reduced the number of bleeding or spotting days but only after 6 months of treatment (*128*). Evidence also suggests that estrogen (*129*–*131*), daily COCs (*129*), levonorgestrel pills (*130*), tamoxifen (*132*), or tranexamic acid (*133*) can reduce the number of bleeding or spotting days during treatment among levonorgestrel implant users. In one small study, vitamin E was found to significantly reduce the mean number of bleeding days after the first treatment cycle; however, another larger study reported no significant differences in length of bleeding and spotting episodes with vitamin E treatment (*134*,*135*). Use of aspirin did not result in a significant difference in median length of bleeding or bleeding and spotting episodes after treatment (*134*). One study among implant users reported a reduction in number of bleeding days after initiating ibuprofen; however, another trial did not demonstrate any significant differences in the number of spotting and bleeding episodes with ibuprofen compared with placebo (*123*,*130*).

Injectables

Progestin-only injectable contraceptives (DMPA, 150 mg intramuscularly or 104 mg subcutaneously) are available in the United States; the only difference between these two formulations is the route of administration. Approximately 6 out of 100 women will become pregnant in the first year of use of DMPA with typical use (*59*). DMPA is reversible and can be used by women of all ages, including adolescents. DMPA does not protect against STDs; consistent and correct use of male latex condoms reduces the risk for STDs, including HIV.

Initiation of Injectables

Timing

- The first DMPA injection can be given at any time if it is reasonably certain that the woman is not pregnant (Box 1).

Need for Back-Up Contraception

- If DMPA is started within the first 7 days since menstrual bleeding started, no additional contraceptive protection is needed.
- If DMPA is started >7 days since menstrual bleeding started, the woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.

Special Considerations

Amenorrhea (Not Postpartum)

- **Timing:** The first DMPA injection can be given at any time if it is reasonably certain that the woman is not pregnant (Box 1).
- **Need for back-up contraception:** The woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.

Postpartum (Breastfeeding)

- **Timing:** The first DMPA injection can be given at any time, including immediately postpartum (U.S. MEC 2 if <1 month postpartum and U.S. MEC 1 if ≥1 month postpartum) if it is reasonably certain that the woman is not pregnant (Box 1).
- **Need for back-up contraception:** If the woman is <6 months postpartum, amenorrheic, and fully or nearly fully breastfeeding (exclusively breastfeeding or the vast majority [≥85%] of feeds are breastfeeds) (*60*), no additional contraceptive protection is needed. Otherwise, a woman who is ≥21 days postpartum and has not experienced return of her menstrual cycle needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days. If her menstrual cycles have returned and it has been >7 days since menstrual bleeding started, she needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.

Postpartum (Not Breastfeeding)

- **Timing:** The first DMPA injection can be given at any time, including immediately postpartum (U.S. MEC 1) if it is reasonably certain that the woman is not pregnant (Box 1).
- **Need for back-up contraception:** A woman who is ≥21 days postpartum and has not experienced return of her menstrual cycle needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days. If her menstrual cycles have returned and it has been >7 days since menstrual bleeding started, she needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.

Postabortion (Spontaneous or Induced)

- **Timing:** The first DMPA injection can be given within the first 7 days, including immediately postabortion (U.S. MEC 1).
- **Need for back-up contraception:** The woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days unless the injection is given at the time of a surgical abortion.

Switching from Another Contraceptive Method

- **Timing:** The first DMPA injection can be given immediately if it is reasonably certain that the woman is not pregnant (Box 1). Waiting for her next menstrual period is unnecessary.
- **Need for back-up contraception:** If it has been >7 days since menstrual bleeding started, the woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.
- **Switching from an IUD:** If the woman has had sexual intercourse since the start of her current menstrual cycle and it has been >5 days since menstrual bleeding started, theoretically, residual sperm might be in the genital tract, which could lead to fertilization if ovulation occurs. A health-care provider may consider any of the following options:
  - Advise the women to retain the IUD for at least 7 days after the injection and return for IUD removal.
  - Advise the woman to abstain from sexual intercourse or use barrier contraception for 7 days before removing the IUD and switching to the new method.
  - Advise the woman to use ECPs at the time of IUD removal.

Comments and Evidence Summary. In situations in which the health-care provider is uncertain whether the woman might be pregnant, the benefits of starting DMPA likely exceed any risk; therefore, starting DMPA should be considered at any time, with a follow-up pregnancy test in 2–4 weeks. If a woman needs to use additional contraceptive protection when switching to DMPA from another contraceptive method, consider continuing her previous method for 7 days after DMPA injection.

A systematic review identified eight articles examining DMPA initiation on different days of the menstrual cycle (*20*). Evidence from two studies with small samples indicated that DMPA injections given up to day 7 of the menstrual cycle inhibited ovulation; when DMPA was administered after

day 7, ovulation occurred in some women. Cervical mucus was of poor quality (i.e., not favorable for sperm penetration) in 90% of women within 24 hours of the injection (Level of evidence: II-2, fair) (*136–138*). Studies found that use of another contraceptive method until DMPA could be initiated (bridging option) did not help women initiate DMPA and was associated with more unintended pregnancies than immediate receipt of DMPA (*139–143*) (Level of evidence: I to II-3, fair to poor, indirect).

Examinations and Tests Needed Before Initiation of an Injectable

Among healthy women, no examinations or tests are needed before initiation of DMPA, although a baseline weight and BMI measurement might be useful for monitoring DMPA users over time (Table 4). Women with known medical problems or other special conditions might need additional examinations or tests before being determined to be appropriate candidates for a particular method of contraception. U.S. MEC might be useful in such circumstances (*5*).

Comments and Evidence Summary. Weight (BMI): Obese women can use (U.S. MEC 1) or generally can use (U.S. MEC 2) DMPA (*5*); therefore, screening for obesity is not necessary for the safe initiation of DMPA. However, measuring weight and calculating BMI at baseline might be helpful for monitoring any changes and counseling women who might be concerned about weight change perceived to be associated with their contraceptive method. (See guidance on follow-up for DMPA users for evidence on weight gain with DMPA use.)

TABLE 4. Classification of examinations and tests needed before DMPA initiation

Examination or test	Class*
Examination	
Blood pressure	C
Weight (BMI) (weight [kg]/height [m]^2)	—†
Clinical breast examination	C
Bimanual examination and cervical inspection	C
Laboratory test	
Glucose	C
Lipids	C
Liver enzymes	C
Hemoglobin	C
Thrombogenic mutations	C
Cervical cytology (Papanicolaou smear)	C
STD screening with laboratory tests	C
HIV screening with laboratory tests	C

Abbreviations: BMI = body mass index; DMPA = depot medroxyprogesterone acetate; HIV = human immunodeficiency virus; STD = sexually transmitted disease; U.S. MEC = *U.S. Medical Eligibility Criteria for Contraceptive Use, 2010*.

* **Class A:** essential and mandatory in all circumstances for safe and effective use of the contraceptive method. **Class B:** contributes substantially to safe and effective use, but implementation may be considered within the public health and/or service context; the risk of not performing an examination or test should be balanced against the benefits of making the contraceptive method available. **Class C:** does not contribute substantially to safe and effective use of the contraceptive method.

† Weight (BMI) measurement is not needed to determine medical eligibility for any methods of contraception because all methods can be used (U.S. MEC 1) or generally can be used (U.S. MEC 2) among obese women (Box 2). However, measuring weight and calculating BMI at baseline might be helpful for monitoring any changes and counseling women who might be concerned about weight change perceived to be associated with their contraceptive method.

Bimanual examination and cervical inspection: Pelvic examination is not necessary before initiation of DMPA because it does not facilitate detection of conditions for which DMPA would be unsafe. Although women with current breast cancer should not use DMPA (U.S. MEC 4), and women with severe hypertension, heart disease, vascular disease, migraine headaches with aura, or certain liver diseases generally should not use DMPA (U.S. MEC 3) (*5*), none of these conditions are likely to be detected by pelvic examination (*120*). A systematic review identified two case-control studies that compared delayed versus immediate pelvic examination before initiation of hormonal contraceptives, specifically oral contraceptives or DMPA (*15*). No differences in risk factors for cervical neoplasia, incidence of STDs, incidence of abnormal Papanicolaou smears, or incidence of abnormal wet mounts were observed (Level of evidence: II-2, fair, direct).

Blood pressure: Women with hypertension generally can use DMPA (U.S. MEC 2), with the exception of women with severe hypertension or vascular disease, who generally should not use DMPA (U.S. MEC 3) (*5*). Screening for hypertension before initiation of DMPA is not necessary because of the low prevalence of undiagnosed severe hypertension and the high likelihood that women with these conditions already would have had them diagnosed. A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened with a blood pressure measurement before initiation of progestin-only contraceptives (*21*). The prevalence of undiagnosed hypertension among women of reproductive age is low. During 1999–2008 among women aged 20–44 years in the United States, the percentage with diagnosed hypertension was 7.8%, and the percentage with undiagnosed hypertension was 1.9% (*144*).

Glucose: Although women with complicated diabetes generally should not use DMPA (U.S. MEC 3) (*5*), screening for diabetes before initiation of DMPA is not necessary because of the low prevalence of undiagnosed diabetes and the high likelihood that women with complicated diabetes would already have had the condition diagnosed. A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened with glucose measurement before initiation of hormonal contraceptives (*14*). The prevalence of diabetes among women of reproductive age is low. During 1999–2008 among women aged 20–44 years

in the United States, the percentage with diagnosed diabetes was 3% and the percentage with undiagnosed diabetes was 0.5% (*144*). Although hormonal contraceptives can have some adverse effects on glucose metabolism in healthy and diabetic women, the overall clinical effect is minimal (*145–151*).

Liver enzymes: Although women with certain liver diseases generally should not use DMPA (U.S. MEC 3) (*5*), screening for liver disease before initiation of DMPA is not necessary because of the low prevalence of these conditions and the high likelihood that women with liver disease already would have had the condition diagnosed. A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened with liver enzyme tests before initiation of hormonal contraceptives (*14*). The prevalence of liver disorders among women of reproductive age is low. In 2008 among adults aged 18–44 years, the percentage with liver disease (not further specified) was 1.0% (*90*). In 2009, the incidence of acute hepatitis A, B, or C among women was <1 per 100,000 population (*91*). During 1998–2007, the incidence of liver carcinoma among women was approximately 3 per 100,000 population (*92*). Because estrogen and progestins are metabolized in the liver, the use of hormonal contraceptives among women with liver disease might, theoretically, be a concern. The use of hormonal contraceptives, specifically COCs and POPs, does not affect disease progression or severity in women with hepatitis, cirrhosis, or benign focal nodular hyperplasia (*93*,*94*), although evidence is limited and no evidence exists for DMPA.

Clinical breast examination: Although women with current breast cancer should not use DMPA (U.S. MEC 4) (*5*), screening asymptomatic women with a clinical breast examination before initiating DMPA is not necessary because of the low prevalence of breast cancer among women of reproductive age. A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened with a clinical breast examination before initiation of hormonal contraceptives (*15*). The incidence of breast cancer among women of reproductive age in the United States is low. In 2009, the incidence of breast cancer among women aged 20–49 years was approximately 72 per 100,000 women (*95*).

Other screening: Women with hyperlipidemia, anemia, thrombogenic mutations, cervical intraepithelial neoplasia, cervical cancer, HIV infection, or other STDs can use (U.S. MEC 1) or generally can use (U.S. MEC 2) DMPA (*5*); therefore, screening for these conditions is not necessary for the safe initiation of DMPA.

Routine Follow-Up After Injectable Initiation

These recommendations address when routine follow-up is recommended for safe and effective continued use of contraception for healthy women. The recommendations refer to general situations and might vary for different users and different situations. Specific populations that might benefit from more frequent follow-up visits include adolescents, those with certain medical conditions or characteristics, and those with multiple medical conditions.

- Advise a woman to return at any time to discuss side effects or other problems, if she wants to change the method being used, and when it is time for reinjection. No routine follow-up visit is required.
- At other routine visits, health-care providers seeing injectable users should do the following:
  - Assess the woman's satisfaction with her contraceptive method and whether she has any concerns about method use.
  - Assess any changes in health status, including medications, that would change the appropriateness of the injectable for safe and effective continued use based on U.S. MEC (e.g., category 3 and 4 conditions and characteristics).
  - Consider assessing weight changes and counseling women who are concerned about weight changes perceived to be associated with their contraceptive method.

Comments and Evidence Summary. Although no evidence exists regarding whether a routine follow-up visit after initiating DMPA improves correct or continued use, monitoring weight or BMI change over time is important for DMPA users.

A systematic review identified a limited body of evidence that examined whether weight gain in the few months after DMPA initiation predicted future weight gain (*17*). Two studies found significant differences in weight gain or BMI at follow-up periods ranging from 12 to 36 months between early weight gainers (i.e., those who gained >5% of their baseline body weight within 6 months after initiation) and those who were not early weight gainers (*152*,*153*). The differences between groups were more pronounced at 18, 24, and 36 months than at 12 months. One study found that most adolescent DMPA users who had gained >5% of their baseline weight by 3 months gained even more weight by 12 months (*154*) (Level of evidence: II-2, fair, to II-3, fair, direct).

Timing of Repeat Injections

Reinjection Interval

- Provide repeat DMPA injections every 3 months (13 weeks).

Special Considerations

Early Injection

- The repeat DMPA injection can be given early when necessary.

Late Injection

- The repeat DMPA injection can be given up to 2 weeks late (15 weeks from the last injection) without requiring additional contraceptive protection.
- If the woman is >2 weeks late (>15 weeks from the last injection) for a repeat DMPA injection, she can have the injection if it is reasonably certain that she is not pregnant (Box 1). She needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days. She might consider the use of emergency contraception if appropriate.

Comments and Evidence Summary. There are no time limits on early injections; injections can be given when necessary (e.g., when a woman cannot return at the routine interval). WHO has extended the time that a woman can have a late reinjection (i.e., grace period) for DMPA use from 2 weeks to 4 weeks on the basis of data from one study showing low pregnancy rates through 4 weeks; however, the CDC expert group did not consider the data to be generalizable to the United States because a large proportion of women in the study were breastfeeding. Therefore, U.S. SPR recommends a grace period of 2 weeks.

A systematic review identified 12 studies evaluating time to pregnancy or ovulation after the last injection of DMPA (*155*). Although pregnancy rates were low during the 2-week interval following the reinjection date and for 4 weeks following the reinjection date, data were sparse and one study included a large proportion of breastfeeding women (*156–158*). Studies also indicated a wide variation in time to ovulation after the last DMPA injection, with the majority ranging from 15 to 49 weeks from the last injection (*159–167*) (Level of evidence: II-2, fair, direct).

Bleeding Irregularities (Including Amenorrhea) During Injectable Use

- Before DMPA initiation, provide counseling about potential changes in bleeding patterns during DMPA use. Amenorrhea and unscheduled spotting or light bleeding is common with DMPA use, and heavy or prolonged bleeding can occur with DMPA use. These bleeding irregularities are generally not harmful and might decrease with continued DMPA use.

Unscheduled Spotting or Light Bleeding

- If clinically indicated, consider an underlying gynecological problem, such as interactions with other medications, an STD, pregnancy, or new pathologic uterine conditions (e.g., polyps or fibroids). If an underlying gynecological problem is found, treat the condition or refer for care.
- If an underlying gynecologic problem is not found and the woman wants treatment, the following treatment option during days of bleeding can be considered:
  - NSAIDs for short-term treatment (5–7 days)
- If unscheduled spotting or light bleeding persists and the woman finds it unacceptable, counsel her on alternative contraceptive methods, and offer another method if it is desired.

Heavy or Prolonged Bleeding

- If clinically indicated, consider an underlying gynecological problem, such as interactions with other medications, an STD, pregnancy, or new pathologic uterine conditions (such as fibroids or polyps). If an underlying gynecologic problem is identified, treat the condition or refer for care.
- If an underlying gynecologic problem is not found and the woman wants treatment, the following treatment options during days of bleeding can be considered:
  - NSAIDS for short-term treatment (5–7 days)
  - Hormonal treatment (if medically eligible) with low-dose COCs or estrogen for short-term treatment (10–20 days)
- If heavy or prolonged bleeding persists and the woman finds it unacceptable, counsel her on alternative contraceptive methods, and offer another method if it is desired.

Amenorrhea

- Amenorrhea does not require any medical treatment. Provide reassurance.
  - If a woman's regular bleeding pattern changes abruptly to amenorrhea, consider ruling out pregnancy if clinically indicated.
- If amenorrhea persists and the woman finds it unacceptable, counsel her on alternative contraceptive methods, and offer another method if it is desired.

Comments and Evidence Summary. During contraceptive counseling and before initiation of DMPA, information about common side effects such as irregular bleeding should be discussed. Unscheduled bleeding or spotting is common with DMPA use (*168*). Additionally, amenorrhea is common after ≥1 years of continuous use (*168,169*). These bleeding irregularities are generally not harmful. Enhanced counseling among DMPA users detailing expected bleeding patterns and

reassurance that these irregularities generally are not harmful has been shown to reduce DMPA discontinuation in clinical trials (*101*,*102*).

A systematic review, as well as two additional studies, examined the treatment of bleeding irregularities during DMPA use (*122*,*170*,*171*). Two small studies found significant cessation of bleeding within 7 days of starting treatment among women taking valdecoxib for 5 days or mefenamic acid for 5 days compared with placebo (*172*,*173*). Treatment with ethinyl estradiol was found to stop bleeding better than placebo during the treatment period, although rates of discontinuation were high, and safety outcomes were not examined (*174*). In one small study among DMPA users who had been experiencing amenorrhea for 2 months, treatment with COCs was found to alleviate amenorrhea better than placebo (*175*). No studies examined the effects of aspirin on bleeding irregularities among DMPA users.

Combined Hormonal Contraceptives

Combined hormonal contraceptives contain both estrogen and a progestin and include 1) COCs (various formulations), 2) a transdermal contraceptive patch (which releases 150 μg of norelgestromin and 20 μg ethinyl estradiol daily), and 3) a vaginal contraceptive ring (which releases 120 μg etonogestrel and 15 μg ethinyl estradiol daily). Approximately 9 out of 100 women become pregnant in the first year of use with combined hormonal contraceptives with typical use (*59*). These methods are reversible and can be used by women of all ages. Combined hormonal contraceptives are generally used for 21–24 consecutive days, followed by 4–7 hormone-free days (either no use or placebo pills). These methods are sometimes used for an extended period with infrequent or no hormone-free days. Combined hormonal contraceptives do not protect against STDs; consistent and correct use of male latex condoms reduces the risk for STDs, including HIV.

Initiation of Combined Hormonal Contraceptives

Timing

- Combined hormonal contraceptives can be initiated at any time if it is reasonably certain that the woman is not pregnant (Box 1).

Need for Back-Up Contraception

- If combined hormonal contraceptives are started within the first 5 days since menstrual bleeding started, no additional contraceptive protection is needed.
- If combined hormonal contraceptives are started >5 days since menstrual bleeding started, the woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.

Special Considerations

Amenorrhea (Not Postpartum)

- **Timing:** Combined hormonal contraceptives can be started at any time if it is reasonably certain that the woman is not pregnant (Box 1).
- **Need for back-up contraception:** The woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.

Postpartum (Breastfeeding)

- **Timing:** Combined hormonal contraceptives can be started when the woman is medically eligible to use the method (*176*) and if it is reasonably certain that she is not pregnant. (Box 1).
  - Postpartum women who are breastfeeding should not use combined hormonal contraceptives during the first 3 weeks after delivery (U.S. MEC 4) because of concerns about increased risk for venous thromboembolism and generally should not use combined hormonal contraceptives during the fourth week postpartum (U.S. MEC 3) because of concerns about potential effects on breastfeeding performance. Postpartum, breastfeeding women with other risk factors for venous thromboembolism generally should not use combined hormonal contraceptives 4–6 weeks after delivery (U.S. MEC 3).
- **Need for back-up contraception:** If the woman is <6 months postpartum, amenorrheic, and fully or nearly fully breastfeeding (exclusively breastfeeding or the vast majority [≥85%] of feeds are breastfeeds) (*60*), no additional contraceptive protection is needed. Otherwise, a woman who is ≥21 days postpartum and has not experienced return of her menstrual cycle needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days. If her menstrual cycles have returned and it has been >5 days since menstrual bleeding started, she needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.

Postpartum (Not Breastfeeding)

- **Timing:** Combined hormonal contraceptives can be started when the woman is medically eligible (*176*) and if it is reasonably certain that she is not pregnant (Box 1).
  - Postpartum women should not use combined hormonal contraceptives during the first 3 weeks after delivery (U.S. MEC 4) because of concerns about increased risk

for venous thromboembolism. Postpartum women with other risk factors for venous thromboembolism generally should not use combined hormonal contraceptives 3–6 weeks after delivery (U.S. MEC 3).

- **Need for back-up contraception:** A woman who is ≥21 days postpartum and whose menstrual cycles have not returned needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days. If her menstrual cycles have returned and it has been >5 days since menstrual bleeding started, she needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.

Postabortion (Spontaneous or Induced)

- **Timing:** Combined hormonal contraceptives can be started within the first 7 days after first or second trimester abortion, including immediately postabortion (U.S. MEC 1).
- **Need for back-up contraception:** She needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days unless combined hormonal contraceptives are started at the time of a surgical abortion.

Switching from Another Contraceptive Method

- **Timing:** Combined hormonal contraceptives can be started immediately if it is reasonably certain that the woman is not pregnant (Box 1). Waiting for her next menstrual period is unnecessary.
- **Need for back-up contraception:** If it has been >5 days since menstrual bleeding started, she needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.
- **Switching from an IUD:** If the woman has had sexual intercourse since the start of her current menstrual cycle and it has been >5 days since menstrual bleeding started, theoretically, residual sperm might be in the genital tract, which could lead to fertilization if ovulation occurs. A health-care provider may consider any of the following options:
  - Advise the women to retain the IUD for at least 7 days after combined hormonal contraceptives are initiated and return for IUD removal.
  - Advise the woman to abstain from sexual intercourse or use barrier contraception for 7 days before removing the IUD and switching to the new method.
  - Advise the woman to use ECPs at the time of IUD removal.

Comments and Evidence Summary. In situations in which the health-care provider is uncertain whether the woman might be pregnant, the benefits of starting combined hormonal contraceptives likely exceed any risk; therefore, starting combined hormonal contraceptives should be considered at any time, with a follow-up pregnancy test in 2–4 weeks. If a woman needs to use additional contraceptive protection when switching to combined hormonal contraceptives from another contraceptive method, consider continuing her previous method for 7 days after starting combined hormonal contraceptives.

A systematic review of 18 studies examined the effects of starting combined hormonal contraceptives on different days of the menstrual cycle (*22*). Overall, the evidence suggested that pregnancy rates did not differ by the timing of combined hormonal contraceptive initiation (*143*,*177–179*) (Level of evidence: I to II-3, fair, indirect). The more follicular activity that occurred before starting COCs, the more likely ovulation was to occur; however, no ovulations occurred when COCs were started at a follicle diameter of 10 mm (mean cycle day 7.6) or when the ring was started at 13 mm (median cycle day 11) (*180–189*) (Level of evidence: I to II-3, fair, indirect). Bleeding patterns and other side effects did not vary with the timing of combined hormonal contraceptive initiation (*177*,*178*,*190–194*) (Level of evidence: I to II-2, good to poor, direct). Although continuation rates of combined hormonal contraceptives were initially improved by the "quick start" approach (i.e., starting on the day of the visit), the advantage disappeared over time (*178*,*179*,*190–195*) (Level of evidence: I to II-2, good to poor, direct).

Examinations and Tests Needed Before Initiation of Combined Hormonal Contraceptives

Among healthy women, few examinations or tests are needed before initiation of combined hormonal contraceptives (Table 5). Blood pressure should be measured before initiation of combined hormonal contraceptives. Baseline weight and BMI measurements might be useful for monitoring combined hormonal contraceptive users over time. Women with known medical problems or other special conditions might need additional examinations or tests before being determined to be appropriate candidates for a particular method of contraception. U.S. MEC might be useful in such circumstances (*5*).

Comments and Evidence Summary. Blood pressure: Women who have more severe hypertension (systolic pressure of ≥160 mm Hg or diastolic pressure of ≥100 mm Hg) or vascular disease should not use combined hormonal contraceptives (U.S. MEC 4), and women who have less severe hypertension (systolic pressure of 140–159 mm Hg or diastolic pressure of 90–99 mm Hg) or adequately controlled hypertension generally should not use combined hormonal contraceptives (U.S. MEC 3) (*5*). Therefore, blood pressure should be measured before initiating combined hormonal contraceptives. If access to health care is limited, blood pressure measurements may be obtained in nonclinical settings, such as

TABLE 5. Classification of examinations and tests needed before combined hormonal contraceptive initiation

Examination or laboratory test	Class*
Examination	
Blood pressure	A†
Weight (BMI) (weight [kg]/height [m]^2)	—§
Clinical breast examination	C
Bimanual examination and cervical inspection	C
Laboratory test	
Glucose	C
Lipids	C
Liver enzymes	C
Hemoglobin	C
Thrombogenic mutations	C
Cervical cytology (Papanicolaou smear)	C
STD screening with laboratory tests	C
HIV screening with laboratory tests	C

Abbreviations: BMI = body mass index; HIV = human immunodeficiency virus; STD = sexually transmitted disease; U.S. MEC = *U.S. Medical Eligibility Criteria for Contraceptive Use, 2010*.

* **Class A:** essential and mandatory in all circumstances for safe and effective use of the contraceptive method. **Class B:** contributes substantially to safe and effective use, but implementation may be considered within the public health and/or service context; the risk of not performing an examination or test should be balanced against the benefits of making the contraceptive method available. **Class C:** does not contribute substantially to safe and effective use of the contraceptive method.

† In cases in which access to health care might be limited, the blood pressure measurement can be obtained by the woman in a nonclinical setting (e.g., pharmacy or fire station) and self-reported to the provider.

§ Weight (BMI) measurement is not needed to determine medical eligibility for any methods of contraception because all methods can be used (U.S. MEC 1) or generally can be used (U.S. MEC 2) among obese women (Box 2). However, measuring weight and calculating BMI at baseline might be helpful for monitoring any changes and counseling women who might be concerned about weight change perceived to be associated with their contraceptive method.

pharmacies or fire stations, and reported by the woman to her provider. Evidence suggests that cardiovascular outcomes are worse among women who did not have their blood pressure measured before initiating COCs.

A systematic review identified six articles from three studies that reported cardiovascular outcomes among women who had blood pressure measurements and women who did not have blood pressure measurements before initiating COCs (*21*). Three case-control studies showed that women who did not have blood pressure measurements before initiating COCs had a higher risk for acute myocardial infarction than women who did have blood pressure measurements (*196*–*198*). Two case-control studies showed that women who did not have blood pressure measurements before initiating COCs had a higher risk for ischemic stroke than women who did have blood pressure measurements (*199*,*200*). One case-control study showed no difference in the risk for hemorrhagic stroke among women who initiated COCs regardless of whether their blood pressure was measured (*201*). Studies that examined hormonal contraceptive methods other than COCs were not identified (Level of evidence: II-2, fair, direct).

Weight (BMI): Obese women generally can use combined hormonal contraceptives (U.S. MEC 2) (*5*); therefore, screening for obesity is not necessary for the safe initiation of combined hormonal contraceptives. However, measuring weight and calculating BMI at baseline might be helpful for monitoring any changes and counseling women who might be concerned about weight change perceived to be associated with their contraceptive method.

Bimanual examination and cervical inspection: Pelvic examination is not necessary before initiation of combined hormonal contraceptives because it does not facilitate detection of conditions for which hormonal contraceptives would be unsafe. Women with certain conditions such as current breast cancer, severe hypertension or vascular disease, heart disease, migraine headaches with aura, and certain liver diseases, as well as women aged ≥35 years who smoke ≥15 cigarettes per day, should not use (U.S. MEC 4) or generally should not use (U.S. MEC 3) combined hormonal contraceptives (*5*); however, none of these conditions are likely to be detected by pelvic examination (*120*). A systematic review identified two case-control studies that compared delayed and immediate pelvic examination before initiation of hormonal contraceptives, specifically oral contraceptives or DMPA (*15*). No differences in risk factors for cervical neoplasia, incidence of STDs, incidence of abnormal Papanicolaou smears, or incidence of abnormal wet mounts were found (Level of evidence: II-2 fair, direct).

Glucose: Although women with complicated diabetes should not use (U.S. MEC 4) or generally should not use (U.S. MEC 3) combined hormonal contraceptives, depending on the severity of the condition (*5*), screening for diabetes before initiation of hormonal contraceptives is not necessary because of the low prevalence of undiagnosed diabetes and the high likelihood that women with complicated diabetes already would have had the condition diagnosed. A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened with glucose measurement before initiation of hormonal contraceptives (*14*). The prevalence of diabetes among women of reproductive age is low. During 1999–2008 among women aged 20–44 years in the United States, the percentage with diagnosed diabetes was 3% and the percentage with undiagnosed diabetes was 0.5% (*144*). Although hormonal contraceptives can have some adverse effects on glucose metabolism in healthy and diabetic women, the overall clinical effect is minimal (*145*–*151*).

Lipids: Although some women with hyperlipidemias generally should not use combined hormonal contraceptives (U.S. MEC 2/3, depending on the type and severity of the hyperlipidemia and presence of other cardiovascular risk factors) (*5*), screening for hyperlipidemia before initiation of

hormonal contraceptives is not necessary because of the low prevalence of undiagnosed disease in women of reproductive age and the low likelihood of clinically significant changes with use of hormonal contraceptives. A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened with lipid measurement before initiation of hormonal contraceptives (*14*). The prevalence of hyperlipidemia among women of reproductive age is low. During 1999–2008 among women aged 20–44 years in the United States, approximately 10% had hypercholesterolemia, defined as total cholesterol ≥ 240 mg/dL or currently taking lipid-lowering medications, and the prevalence of undiagnosed hypercholesterolemia was approximately 2% (*144*). Studies have shown mixed results about the effects of hormonal methods on lipid levels, and the clinical significance of these changes is unclear (*202–204*). In addition, women with abnormal lipid levels at baseline were not found to have increased risk for adverse changes to their lipid profile when using hormonal methods (*202*).

Liver enzymes: Although women with certain liver diseases should not use (U.S. MEC 4) or generally should not use (U.S. MEC 3) combined hormonal contraceptives (*5*), screening for liver disease before initiation of combined hormonal contraceptives is not necessary because of the low prevalence of these conditions and the high likelihood that women with liver disease already would have had the condition diagnosed. A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened with liver enzyme tests before initiation of hormonal contraceptives (*14*). The prevalence of liver disorders among women of reproductive age is low. In 2008 among adults aged 18–44 years, the percentage with liver disease (not further specified) was 1.0% (*90*). In 2009, the incidence of acute hepatitis A, B, or C among women was <1 per 100,000 population (*91*). During 1998–2007, the incidence of liver carcinoma among women was approximately 3 per 100,000 population (*92*). Because estrogen and progestins are metabolized in the liver, the use of hormonal contraceptives among women with liver disease might, theoretically, be a concern. The use of hormonal contraceptives, specifically COCs and POPs, does not affect disease progression or severity in women with hepatitis, cirrhosis, or benign focal nodular hyperplasia (*93*,*94*), although evidence is limited; no evidence exists for other types of combined hormonal contraceptives.

Thrombogenic mutations: Women with thrombogenic mutations should not use combined hormonal contraceptives (U.S. MEC 4) (*5*) because of the increased risk for venous thromboembolism (*205*). However, studies have shown that universal screening for thrombogenic mutations before initiating COCs is not cost-effective because of the rarity of the conditions and the high cost of screening (*206–208*).

Clinical breast examination: Although women with current breast cancer should not use combined hormonal contraceptives (U.S. MEC 4) (*5*), screening asymptomatic women with a clinical breast examination before initiating combined hormonal contraceptives is not necessary because of the low prevalence of breast cancer among women of reproductive age. A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened with a breast examination before initiation of hormonal contraceptives (*15*). The incidence of breast cancer among women of reproductive age in the United States is low. In 2009, the incidence of breast cancer among women aged 20–49 years was approximately 72 per 100,000 women (*95*).

Other screening: Women with anemia, cervical intraepithelial neoplasia, cervical cancer, HIV infection, or other STDs can use (U.S. MEC 1) or generally can use (U.S. MEC 2) combined hormonal contraceptives (*5*); therefore, screening for these conditions is not necessary for the safe initiation of combined hormonal contraceptives.

Number of Pill Packs that Should Be Provided at Initial and Return Visits

- At the initial and return visits, provide or prescribe up to a 1-year supply of COCs (e.g., 13 28-day pill packs), depending on the woman's preferences and anticipated use.
- A woman should be able to obtain COCs easily in the amount and at the time she needs them.

Comments and Evidence Summary. The more pill packs given up to 13 cycles, the higher the continuation rates. Restricting the number of pill packs distributed or prescribed can result in unwanted discontinuation of the method and increased risk for pregnancy.

A systematic review of the evidence suggested that providing a greater number of pill packs was associated with increased continuation (*23*). Studies that compared provision of one versus 12 packs, one versus 12 or 13 packs, or three versus seven packs found increased continuation of pill use among women provided with more pill packs (*209–211*). However, one study found that there was no difference in continuation when patients were provided one and then three packs versus four packs all at once (*212*). In addition to continuation, a greater number of pills packs provided was associated with fewer pregnancy tests, fewer pregnancies, and lower cost per client. However, a greater number of pill packs (i.e., 13 packs versus three packs) also was associated with increased pill wastage in one study (*210*) (Level of evidence: I to II-2, fair, direct).

Routine Follow-Up After Combined Hormonal Contraceptive Initiation

These recommendations address when routine follow-up is recommended for safe and effective continued use of contraception for healthy women. The recommendations refer to general situations and might vary for different users and different situations. Specific populations that might benefit from more frequent follow-up visits include adolescents, those with certain medical conditions or characteristics, and those with multiple medical conditions.

- Advise a woman to return at any time to discuss side effects or other problems or if she wants to change the method being used. No routine follow-up visit is required.
- At other routine visits, health-care providers seeing combined hormonal contraceptive users should do the following:
  - Assess the woman's satisfaction with her contraceptive method and whether she has any concerns about method use.
  - Assess any changes in health status, including medications, that would change the appropriateness of combined hormonal contraceptives for safe and effective continued use based on U.S. MEC (e.g., category 3 and 4 conditions and characteristics).
  - Assess blood pressure.
  - Consider assessing weight changes and counseling women who are concerned about weight changes perceived to be associated with their contraceptive method.

Comments and Evidence Summary. No evidence exists regarding whether a routine follow-up visit after initiating combined hormonal contraceptives improves correct or continued use. Monitoring blood pressure is important for combined hormonal contraceptive users. Health-care providers might consider recommending women obtain blood pressure measurements in nonclinical settings (e.g., pharmacy or fire station).

A systematic review identified five studies that examined the incidence of hypertension among women who began using a COC versus those who started a nonhormonal method of contraception or a placebo (*17*). Few women developed hypertension after initiating COCs, and studies examining increases in blood pressure after COC initiation found mixed results. No studies were identified that examined changes in blood pressure among patch or vaginal ring users (Level of evidence: I, fair, to II-2, fair, indirect).

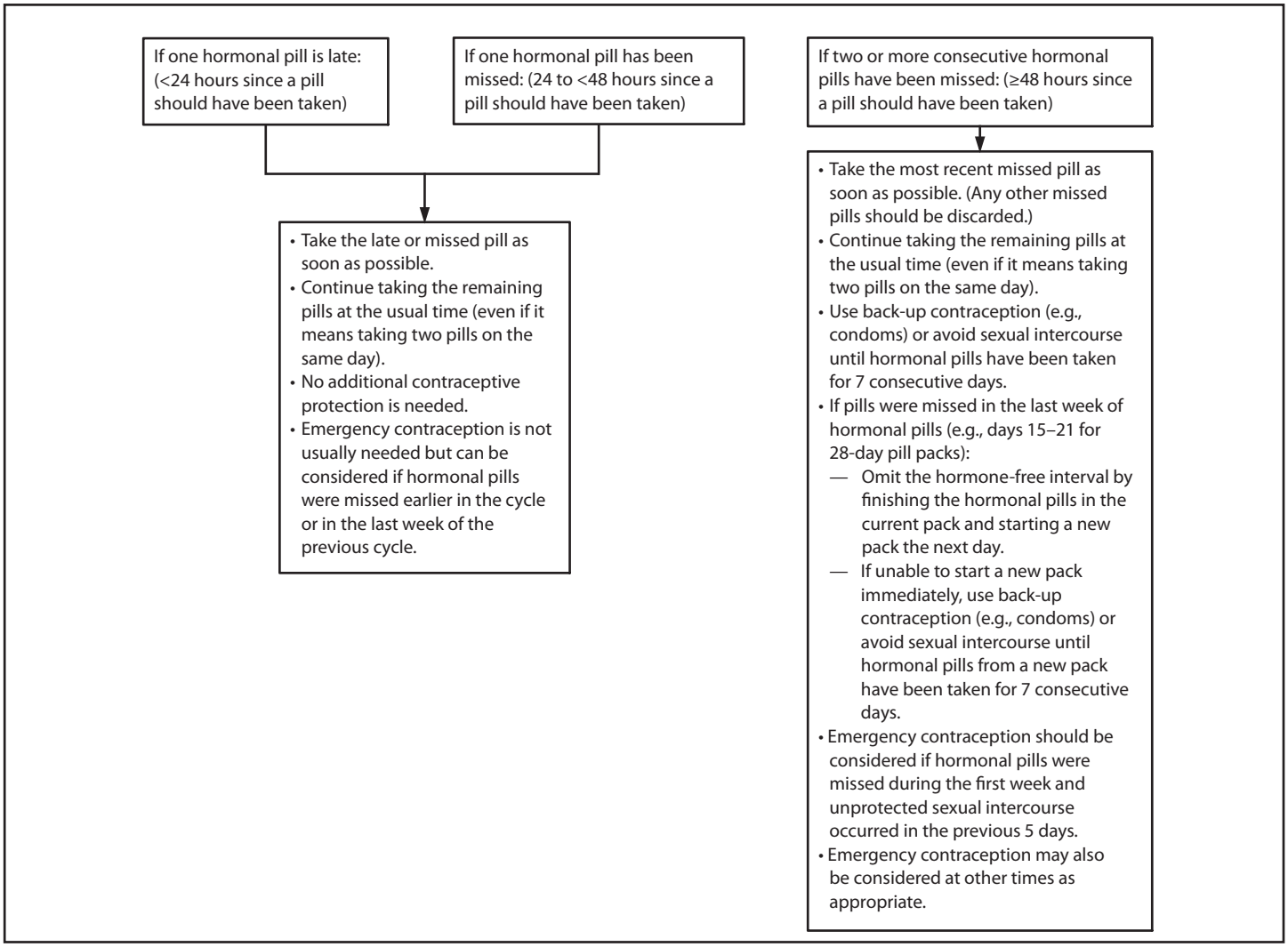
Late or Missed Doses and Side Effects from Combined Hormonal Contraceptive Use

For the following recommendations, a dose is considered late when <24 hours have elapsed since the dose should have been taken. A dose is considered missed if ≥24 hours have elapsed since the dose should have been taken. For example, if a COC pill was supposed to have been taken on Monday at 9:00 a.m. and is taken at 11:00 a.m., the pill is late; however, by Tuesday morning at 11:00 a.m., Monday's 9:00 a.m. pill has been missed and Tuesday's 9:00 a.m. pill is late. For COCs, the recommendations only apply to late or missed hormonally active pills and not to placebo pills. Recommendations are provided for late or missed pills (Figure 2), the patch (Figure 3), and the ring (Figure 4).

Comments and Evidence Summary. Inconsistent or incorrect use of combined hormonal contraceptives is a major cause of combined hormonal contraceptive failure. Extending the hormone-free interval is considered to be a particularly risky time to miss combined hormonal contraceptives. Seven days of continuous combined hormonal contraceptive use is deemed necessary to reliably prevent ovulation. The recommendations reflect a balance between simplicity and precision of science. Women who frequently miss COCs or experience other usage errors with combined hormonal patch or combined vaginal ring should consider an alternative contraceptive method that is less dependent on the user to be effective (e.g., IUD, implant, or injectable).

A systematic review identified 36 studies that examined measures of contraceptive effectiveness of combined hormonal contraceptives during cycles with extended hormone-free intervals, shortened hormone-free intervals, or deliberate nonadherence on days not adjacent to the hormone-free interval (*24*). Most of the studies examined COCs (*188*,*213*–*240*), two examined the combined hormonal patch (*234*,*241*), and six examined the combined vaginal ring (*185*,*242*–*246*). No direct evidence on the effect of missed pills on the risk for pregnancy was found. Studies of women deliberately extending the hormone-free interval up to 14 days found wide variability in the amount of follicular development and occurrence of ovulation (*216*,*219*,*221*,*222*,*224*,*225*,*227*–*230*); in general, the risk for ovulation was low, and among women who did ovulate, cycles were usually abnormal. In studies of women who deliberately missed pills on various days during the cycle not adjacent to the hormone-free interval, ovulation occurred infrequently (*214*,*220*–*222*,*230*,*231*,*233*,*234*). Studies comparing 7-day hormone-free intervals with shorter hormone-free intervals found lower rates of pregnancy (*213*,*217*,*226*,*232*) and significantly greater suppression of ovulation (*215*,*225*,*236*–*238*,*240*) among women with shorter intervals in all but one study (*235*), which found no difference. Two studies that compared 30-μg ethinyl estradiol pills with 20-μg ethinyl estradiol pills showed more follicular activity when 20-μg ethinyl estradiol pills were missed (*216*,*219*). In

FIGURE 2. Recommended actions after late or missed combined oral contraceptives



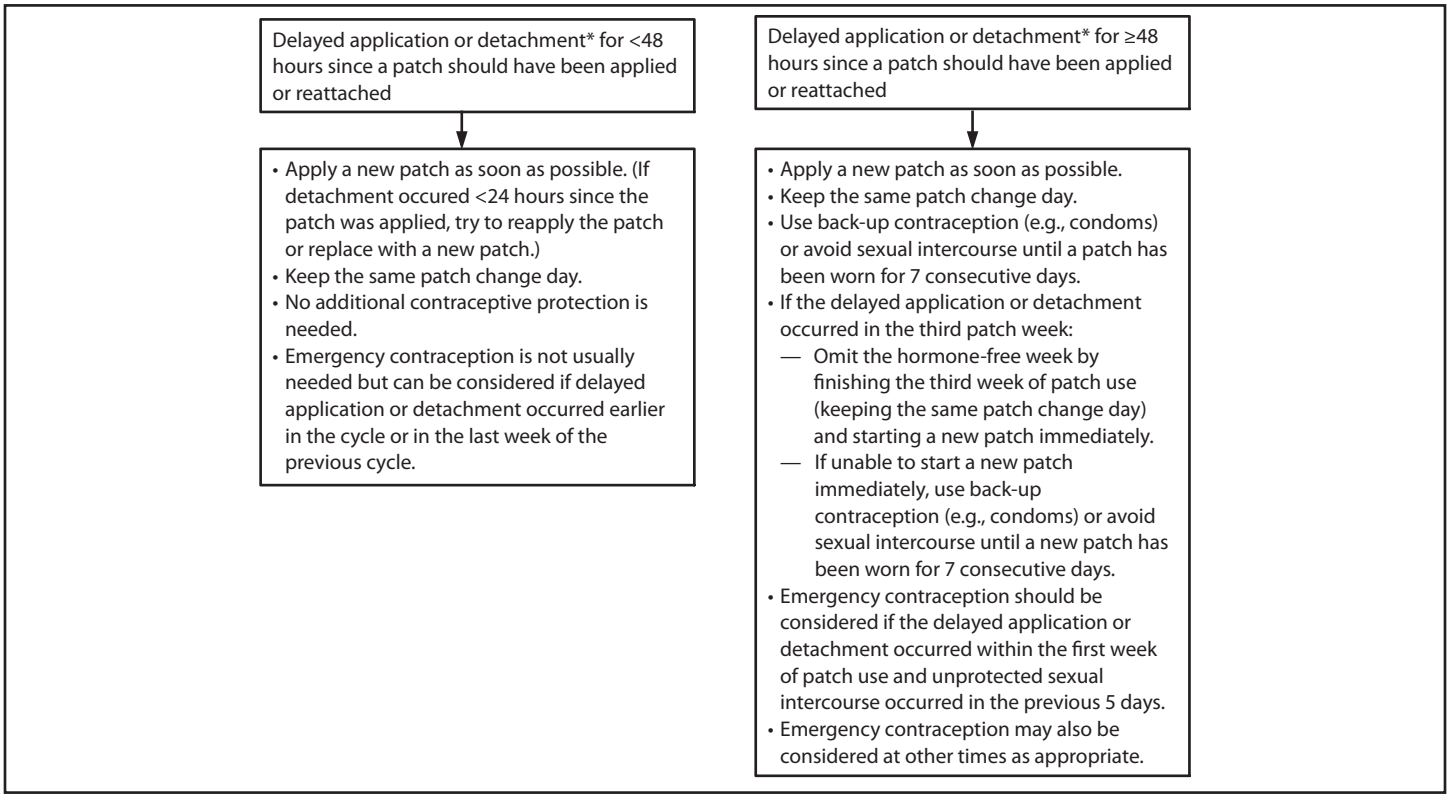
studies examining the combined vaginal ring, three studies found that nondeliberate extension of the hormone-free interval for 24 to <48 hours from the scheduled period did not increase the risk for pregnancy (*242*,*243*,*245*); one study found that ring insertion after a deliberately extended hormone-free interval that allowed a 13-mm follicle to develop interrupted ovarian function and further follicular growth (*185*); and one study found that inhibition of ovulation was maintained after deliberately forgetting to remove the ring for up to 2 weeks after normal ring use (*246*). In studies examining the combined hormonal patch, one study found that missing 1–3 consecutive days before patch replacement (either wearing one patch 3 days longer before replacement or going 3 days without a patch before replacing the next patch) on days not adjacent to the patch-free interval resulted in little follicular activity and low risk for ovulation (*234*), and one pharmacokinetic study found that serum levels of ethinyl estradiol and progestin norelgestromin remained within reference ranges after extending patch wear for 3 days (*241*). No studies were found on extending the patch-free interval. In studies that provide indirect evidence on the effects of missed combined hormonal contraception on surrogate measures of pregnancy, how differences in surrogate measures correspond to pregnancy risk is unclear (Level of evidence: I, good, indirect to II-3, poor, direct).

Vomiting or Severe Diarrhea While Using COCs

Certain steps should be taken by women who experience vomiting or severe diarrhea while using COCs (Figure 5).

Comments and Evidence Summary. Theoretically, the contraceptive effectiveness of COCs might be decreased because of vomiting or severe diarrhea. Because of the lack of evidence that addresses vomiting or severe diarrhea while using COCs, these recommendations are based on the recommendations

FIGURE 3. Recommended actions after delayed application or detachment with combined hormonal patch



* If detachment takes place but the woman is unsure when the detachment occurred, consider the patch to have been detached for ≥48 hours since a patch should have been applied or reattached.

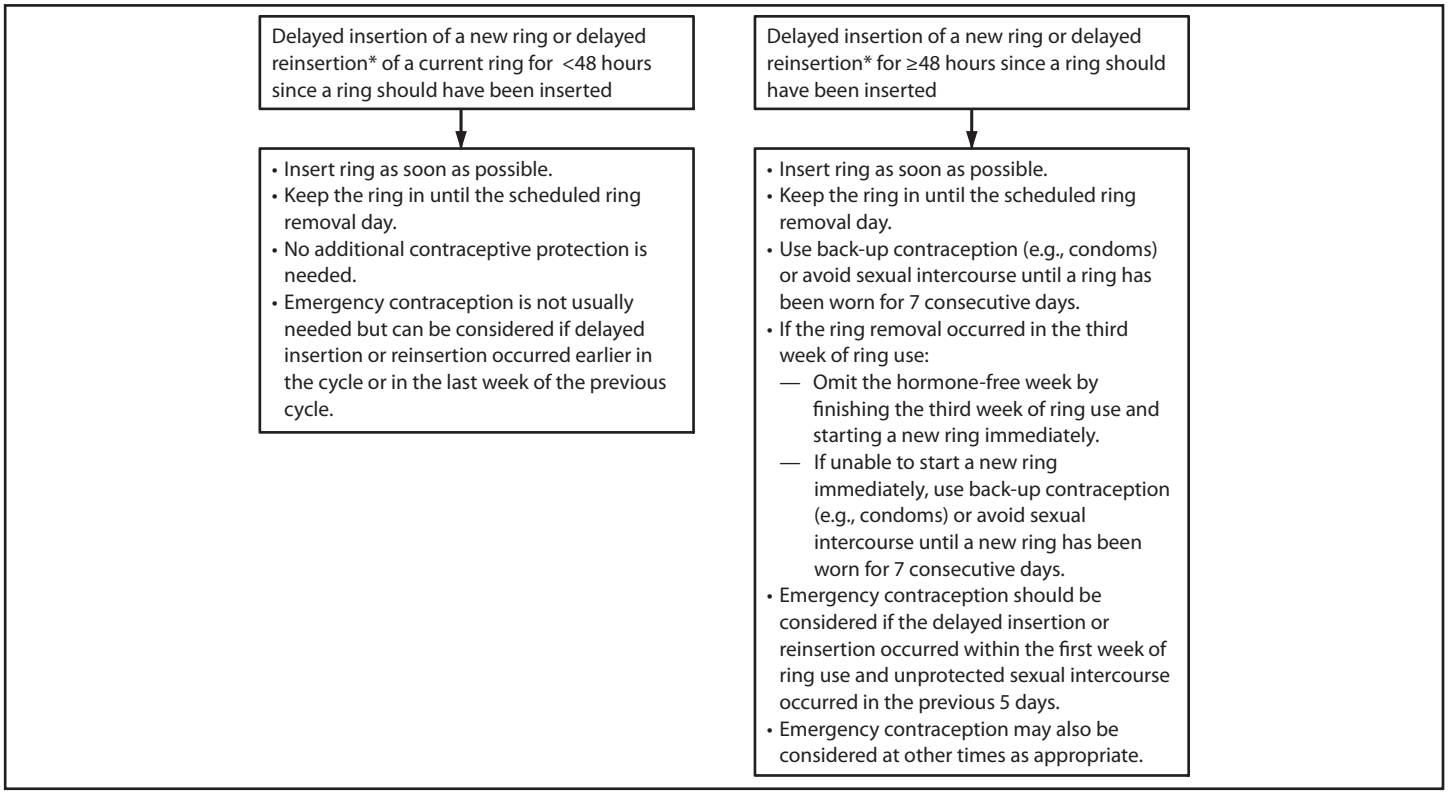
for missed COCs. No evidence was found on the effects of vomiting or diarrhea on measures of contraceptive effectiveness including pregnancy, follicular development, hormone levels, or cervical mucus quality.

Unscheduled Bleeding with Extended or Continuous Use of Combined Hormonal Contraceptives

- Before initiation of combined hormonal contraceptives, provide counseling about potential changes in bleeding patterns during extended or continuous combined hormonal contraceptive use. (Extended contraceptive use is defined as a planned hormone-free interval after at least two contiguous cycles. Continuous contraceptive use is defined as uninterrupted use of hormonal contraception without a hormone-free interval [*247*].)
- Unscheduled spotting or bleeding is common during the first 3–6 months of extended or continuous combined hormonal contraceptive use. It is generally not harmful and decreases with continued combined hormonal contraceptive use.
- If clinically indicated, consider an underlying gynecological problem, such as inconsistent use, interactions with other medications, cigarette smoking, an STD, pregnancy, or new pathologic uterine conditions (e.g., polyps or fibroids). If an underlying gynecological problem is found, treat the condition or refer for care.
- If an underlying gynecological problem is not found and the woman wants treatment, the following treatment option can be considered:
  - Advise the woman to discontinue combined hormonal contraceptive use (i.e., a hormone-free interval) for 3–4 consecutive days; a hormone-free interval is not recommended during the first 21 days of using the continuous or extended combined hormonal contraceptive method. A hormone-free interval also is not recommended more than once per month because contraceptive effectiveness might be reduced.
- If unscheduled spotting or bleeding persists and the woman finds it unacceptable, counsel her on alternative contraceptive methods, and offer another method if it is desired.

Comments and Evidence Summary. During contraceptive counseling and before initiating extended or continuous

FIGURE 4. Recommended actions after delayed insertion or reinsertion with combined vaginal ring



* If removal takes place but the woman is unsure of how long the ring has been removed, consider the ring to have been removed for ≥48 hours since a ring should have been inserted or reinserted.

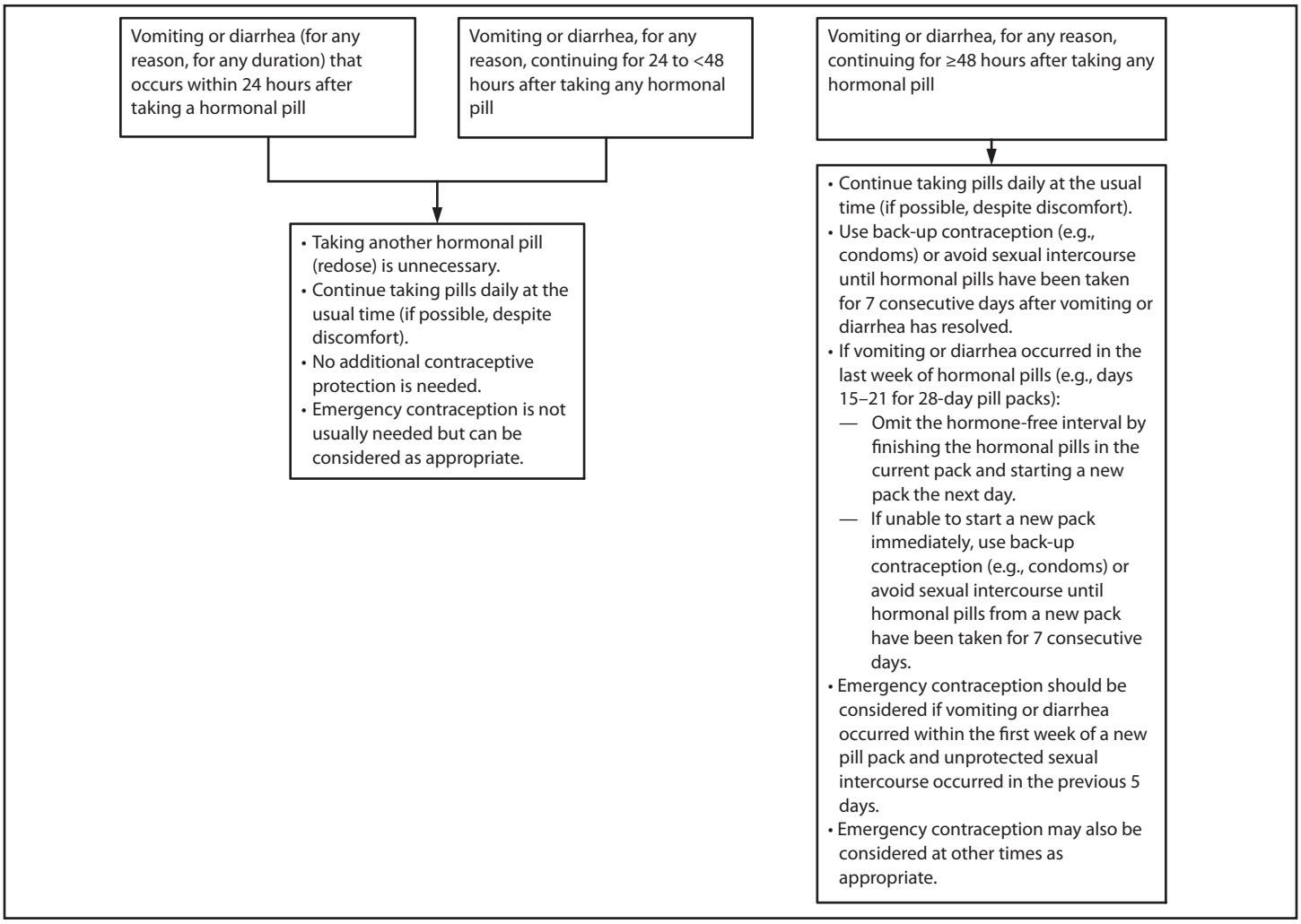
combined hormonal contraceptives, information about common side effects such as unscheduled spotting or bleeding, especially during the first 3–6 months of use, should be discussed (*248*). These bleeding irregularities are generally not harmful and usually improve with persistent use of the hormonal method. To avoid unscheduled spotting or bleeding, counseling should emphasize the importance of correct use and timing; for users of contraceptive pills, emphasize consistent pill use. Enhanced counseling about expected bleeding patterns and reassurance that bleeding irregularities are generally not harmful has been shown to reduce method discontinuation in clinical trials with DMPA (*101*,*102*).

A systematic review identified three studies with small study populations that addressed treatments for unscheduled bleeding among women using extended or continuous combined hormonal contraceptives (*25*). In two separate randomized clinical trials in which women were taking either contraceptive pills or using the contraceptive ring continuously for 168 days, women assigned to a hormone-free interval of 3 or 4 days reported improved bleeding. Although they noted an initial increase in flow, this was followed by an abrupt decrease 7–8 days later with eventual cessation of flow 11–12 days later. These findings were compared with women who continued to use their method without a hormone-free interval, in which a greater proportion reported either treatment failure or fewer days of amenorrhea (*249*,*250*). In another randomized trial of 66 women with unscheduled bleeding among women using 84 days of hormonally active contraceptive pills, oral doxycycline (100 mg twice daily) initiated the first day of bleeding and taken for 5 days did not result in any improvement in bleeding compared with placebo (*251*) (Level of evidence: I, fair, direct).

Progestin-Only Pills

POPs contain only a progestin and no estrogen and are available in the United States. Approximately 9 out of 100 women become pregnant in the first year of use with POPs with typical use (*59*). POPs are reversible and can be used by women of all ages. POPs do not protect against STDs; consistent and correct use of male latex condoms reduces the risk for STDs, including HIV.

FIGURE 5. Recommended steps after vomiting or diarrhea while using combined oral contraceptives



Initiation of POPs

Timing

- POPs can be started at any time if it is reasonably certain that the woman is not pregnant (Box 1).

Need for Back-Up Contraception

- If POPs are started within the first 5 days since menstrual bleeding started, no additional contraceptive protection is needed.
- If POPs are started >5 days since menstrual bleeding started, the woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 2 days.

Special Considerations

Amenorrhea (Not Postpartum)

- **Timing:** POPs can be started at any time if it is reasonably certain that the woman is not pregnant (Box 1).
- **Need for back-up contraception:** The woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 2 days.

Postpartum (Breastfeeding)

- **Timing:** POPs can be started at any time, including immediately postpartum (U.S. MEC 2 if <1 month postpartum and U.S. MEC 1 if ≥1 month postpartum) if it is reasonably certain that the woman is not pregnant (Box 1).
- **Need for back-up contraception:** If the woman is <6 months postpartum, amenorrheic, and fully or nearly fully breastfeeding (exclusively breastfeeding or the vast majority [≥85%] of feeds are breastfeeds) (*60*), no additional contraceptive protection is needed. Otherwise, a woman who is ≥21 days postpartum and has not experienced return of her menstrual cycles needs to abstain from sexual intercourse or use additional contraceptive protection for the next 2 days. If her menstrual cycles have

returned and it has been >5 days since menstrual bleeding started, she needs to abstain from sexual intercourse or use additional contraceptive protection for the next 2 days.

Postpartum (Not Breastfeeding)

- **Timing:** POPs can be started at any time, including immediately postpartum (U.S. MEC 1), if it is reasonably certain that the woman is not pregnant (Box 1).
- **Need for back-up contraception:** Women who are ≥21 days postpartum and whose menstrual cycles have not returned need to abstain from sexual intercourse or use additional contraceptive protection for the next 2 days. If her menstrual cycles have returned and it has been >5 days since menstrual bleeding started, she needs to abstain from sexual intercourse or use additional contraceptive protection for the next 2 days.

Postabortion (Spontaneous or Induced)

- **Timing:** POPs can be started within the first 7 days, including immediately postabortion (U.S. MEC 1).
- **Need for back-up contraception:** The woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 2 days unless POPs are started at the time of a surgical abortion.

Switching from Another Contraceptive Method

- **Timing:** POPs can be started immediately if it is reasonably certain that the woman is not pregnant (Box 1). Waiting for her next menstrual period is unnecessary.
- **Need for back-up contraception:** If it has been >5 days since menstrual bleeding started, she needs to abstain from sexual intercourse or use additional contraceptive protection for the next 2 days.
- **Switching from an IUD:** If the woman has had sexual intercourse since the start of her current menstrual cycle and it has been >5 days since menstrual bleeding started, theoretically, residual sperm might be in the genital tract, which could lead to fertilization if ovulation occurs. A health-care provider may consider any of the following options:
  - Advise the women to retain the IUD for at least 2 days after POPs are initiated and return for IUD removal.
  - Advise the woman to abstain from sexual intercourse or use barrier contraception for 2 days before removing the IUD and switching to the new method.
  - Advise the woman to use ECPs at the time of IUD removal.

Comments and Evidence Summary. In situations in which the health-care provider is uncertain whether the woman might be pregnant, the benefits of starting POPs likely exceed any risk; therefore, starting POPs should be considered at any time, with a follow-up pregnancy test in 2–4 weeks.

Unlike COCs, POPs inhibit ovulation in about half of cycles, although the rates vary widely by individual (*252*). Peak serum steroid levels are reached about 2 hours after administration, followed by rapid distribution and elimination, such that by 24 hours after administration, serum steroid levels are near baseline (*252*). Therefore, taking POPs at approximately the same time each day is important. An estimated 48 hours of POP use has been deemed necessary to achieve the contraceptive effects on cervical mucus (*252*). If a woman needs to use additional contraceptive protection when switching to POPs from another contraceptive method, consider continuing her previous method for 2 days after starting POPs. No direct evidence was found regarding the effects of starting POPs at different times of the cycle.

Examinations and Tests Needed Before Initiation of POPs

Among healthy women, no examinations or tests are needed before initiation of POPs, although a baseline weight and BMI measurement might be useful for monitoring POP users over time (Table 6). Women with known medical problems or other special conditions might need additional examinations or tests before being determined to be appropriate candidates

TABLE 6. Classification of examinations and tests needed before POP initiation

Examination or laboratory test	Class*
Examination	
Blood pressure	C
Weight (BMI) (weight [kg]/height [m]^2)	—†
Clinical breast examination	C
Bimanual examination and cervical inspection	C
Laboratory test	
Glucose	C
Lipids	C
Liver enzymes	C
Hemoglobin	C
Thrombogenic mutations	C
Cervical cytology (Papanicolaou smear)	C
STD screening with laboratory tests	C
HIV screening with laboratory tests	C

Abbreviations: BMI = body mass index; HIV = human immunodeficiency virus; POP = progestin-only pill; STD = sexually transmitted disease; U.S. MEC = *U.S. Medical Eligibility Criteria for Contraceptive Use, 2010.*

* **Class A:** essential and mandatory in all circumstances for safe and effective use of the contraceptive method. **Class B:** contributes substantially to safe and effective use, but implementation may be considered within the public health and/or service context; the risk of not performing an examination or test should be balanced against the benefits of making the contraceptive method available. **Class C:** does not contribute substantially to safe and effective use of the contraceptive method.

† Weight (BMI) measurement is not needed to determine medical eligibility for any methods of contraception because all methods can be used (U.S. MEC 1) or generally can be used (U.S. MEC 2) among obese women (Box 2). However, measuring weight and calculating BMI at baseline might be helpful for monitoring any changes and counseling women who might be concerned about weight change perceived to be associated with their contraceptive method.

for a particular method of contraception. U.S. MEC might be useful in such circumstances (*5*).

Comments and Evidence Summary. Weight (BMI): Obese women can use POPs (U.S. MEC 1) (*5*); therefore, screening for obesity is not necessary for the safe initiation of POPs. However, measuring weight and calculating BMI at baseline might be helpful for monitoring any changes and counseling women who might be concerned about weight change perceived to be associated with their contraceptive method.

Bimanual examination and cervical inspection: Pelvic examination is not necessary before initiation of POPs because it does not facilitate detection of conditions for which POPs would be unsafe. Women with current breast cancer should not use POPs (U.S. MEC 4), and women with certain liver diseases generally should not use POPs (U.S. MEC 3) (*5*); however, neither of these conditions are likely to be detected by pelvic examination (*120*). A systematic review identified two case-control studies that compared delayed versus immediate pelvic examination before initiation of hormonal contraceptives, specifically oral contraceptives or DMPA (*15*). No differences in risk factors for cervical neoplasia, incidence of STDs, incidence of abnormal Papanicolaou smears, or incidence of abnormal wet mounts were observed (Level of evidence: II-2 fair, direct).

Liver enzymes: Although women with certain liver diseases generally should not use POPs (U.S. MEC 3) (*5*), screening for liver disease before initiation of POPs is not necessary because of the low prevalence of these conditions and the high likelihood that women with liver disease already would have had the condition diagnosed. A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened with liver enzyme tests before initiation of hormonal contraceptives (*14*). The prevalence of liver disorders among women of reproductive age is low. In 2008 among U.S. adults aged 18–44 years, the percentage with liver disease (not further specified) was 1.0% (*90*). In 2009, the incidence of acute hepatitis A, B, or C among women was <1 per 100,000 population (*91*). During 1998–2007, the incidence of liver carcinoma among women was approximately 3 per 100,000 population (*92*). Because estrogen and progestins are metabolized in the liver, the use of hormonal contraceptives among women with liver disease might, theoretically, be a concern. The use of hormonal contraceptives, specifically COCs and POPs, does not affect disease progression or severity in women with hepatitis, cirrhosis, or benign focal nodular hyperplasia (*93*,*94*).

Clinical breast examination: Although women with current breast cancer should not use POPs (U.S. MEC 4) (*5*), screening asymptomatic women with a clinical breast examination before initiating POPs is not necessary because of the low prevalence of breast cancer among women of reproductive age. A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened with a clinical breast examination before initiation of hormonal contraceptives (*15*). The incidence of breast cancer among women of reproductive age in the United States is low. In 2009, the incidence of breast cancer among women ages 20–49 was approximately 72 per 100,000 women (*95*).

Other screening: Women with hypertension, diabetes, hyperlipidemia, anemia, thrombogenic mutations, cervical intraepithelial neoplasia, cervical cancer, STDs, or HIV infection can use (U.S. MEC 1) or generally can use (U.S. MEC 2) POPs (*5*); therefore, screening for these conditions is not necessary for the safe initiation of POPs.

Number of Pill Packs that Should Be Provided at Initial and Return Visits

- At the initial and return visits, provide or prescribe up to a 1-year supply of POPs (e.g., 13 28-day pill packs), depending on the woman's preferences and anticipated use.
- A woman should be able to obtain POPs easily in the amount and at the time she needs them.

Comments and Evidence Summary. The more pill packs given up to 13 cycles, the higher the continuation rates. Restricting the number of pill packs distributed or prescribed can result in unwanted discontinuation of the method and increased risk for pregnancy.

A systematic review of the evidence suggested that providing a greater number of pill packs was associated with increased continuation (*23*). Studies that compared provision of one versus 12 packs, one versus 12 or 13 packs, or three versus seven packs found increased continuation of pill use among women provided with more pill packs (*209–211*). However, one study found that there was no difference in continuation when patients were provided one and then three packs versus four packs all at once (*212*). In addition to continuation, a greater number of pill packs provided was associated with fewer pregnancy tests, fewer pregnancies, and lower cost per client. However, a greater number of pill packs (13 packs versus three packs) also was associated with increased pill wastage in one study (*210*) (Level of evidence: I to II-2, fair, direct).

Routine Follow-Up After POP Initiation

These recommendations address when routine follow-up is recommended for safe and effective continued use of contraception for healthy women. The recommendations refer to general situations and might vary for different users and

different situations. Specific populations that might benefit from more frequent follow-up visits include adolescents, those with certain medical conditions or characteristics, and those with multiple medical conditions.

- Advise a woman to return at any time to discuss side effects or other problems or if she wants to change the method being used. No routine follow-up visit is required.
- At other routine visits, health-care providers seeing POP users should do the following:
  - Assess the woman's satisfaction with her contraceptive method and whether she has any concerns about method use.
  - Assess any changes in health status, including medications, that would change the appropriateness of POPs for safe and effective continued use based on U.S. MEC (e.g., category 3 and 4 conditions and characteristics).
  - Consider assessing weight changes and counseling women who are concerned about weight changes perceived to be associated with their contraceptive method.

Comments and Evidence Summary. No evidence was found regarding whether a routine follow-up visit after initiating POPs improves correct or continued use.

Missed POPs

For the following recommendations, a dose is considered missed if it has been >3 hours since it should have been taken.

- Take one pill as soon as possible.
- Continue taking pills daily, one each day, at the same time each day, even if it means taking two pills on the same day.
- Use back-up contraception (e.g., condoms) or avoid sexual intercourse until pills have been taken correctly, on time, for 2 consecutive days.
- Emergency contraception should be considered if the woman has had unprotected sexual intercourse.

Comments and Evidence Summary. Inconsistent or incorrect use of oral contraceptive pills is a major reason for oral contraceptive failure. Unlike COCs, POPs inhibit ovulation in about half of cycles, although this rate varies widely by individual (*252*). Peak serum steroid levels are reached about 2 hours after administration, followed by rapid distribution and elimination, such that by 24 hours after administration, serum steroid levels are near baseline (*252*). Therefore, taking POPs at approximately the same time each day is important. An estimated 48 hours of POP use was deemed necessary to achieve the contraceptive effects on cervical mucus (*252*). Women who frequently miss POPs should consider an alternative contraceptive method that is less dependent on the user to be effective (e.g., IUD, implant, or injectable). No evidence was found regarding the effects of missed POPs available in the United States on measures of contraceptive effectiveness including pregnancy, follicular development, hormone levels, or cervical mucus quality.

Vomiting or Severe Diarrhea (for any Reason or Duration) that Occurs Within 3 Hours After Taking a Pill

- Take another pill as soon as possible (if possible, despite discomfort).
- Continue taking pills daily, one each day, at the same time each day.
- Use back-up contraception (e.g., condoms) or avoid sexual intercourse until 2 days after vomiting or diarrhea has resolved.
- Emergency contraception should be considered if the woman has had unprotected sexual intercourse.

Comments and Evidence Summary. Theoretically, the contraceptive effectiveness of POPs might be decreased because of vomiting or severe diarrhea. Because of the lack of evidence to address this question, these recommendations are based on the recommendations for missed POPs. No evidence was found regarding the effects of vomiting or diarrhea on measures of contraceptive effectiveness, including pregnancy, follicular development, hormone levels, or cervical mucus quality.

Standard Days Method

SDM is a method based on fertility awareness; users must avoid unprotected sexual intercourse on days 8–19 of the menstrual cycle (*253*). Approximately 5 out of 100 women become pregnant in the first year of use with perfect (i.e., correct and consistent) use of SDM (*253*); effectiveness based on typical use is not available for this method but is expected to be lower than that for perfect use. SDM is reversible and can be used by women of all ages. SDM does not protect against STDs; consistent and correct use of male latex condoms reduces the risk for STDs, including HIV.

Use of SDM Among Women with Various Menstrual Cycle Durations

Menstrual Cycles of 26–32 Days

- These women may use the method.
- Provide a barrier method of contraception for protection on days 8–19 if she wants one.
- If she has unprotected sexual intercourse during days 8–19, consider the use of emergency contraception if appropriate.

Two or More Menstrual Cycles of <26 or >32 Days Within Any 1 Year of SDM Use

- Advise the woman that the method might not be appropriate for her because of a higher risk for pregnancy. Help her consider another method.

Comments and Evidence Summary. The probability of pregnancy is increased when the menstrual cycle is outside the range of 26–32 days, even if unprotected sexual intercourse is avoided on days 8–19. A study of 7,600 menstrual cycles, including information on cycle length and signs of ovulation, concluded that the theoretical effectiveness of SDM is greatest for women with cycles of 26–32 days, that the method is still effective for women who occasionally have a cycle outside this range, and that it is less effective for women who consistently have cycles outside this range. Information from daily hormonal measurements shows that the timing of the 6-day fertile window varies greatly, even among women with regular cycles (*39*,*254*,*255*).

Emergency Contraception

Emergency contraception consists of methods that can be used by women after sexual intercourse to prevent pregnancy. Emergency contraception methods have varying ranges of effectiveness depending on the method and timing of administration. Four options are available in the United States: the Cu-IUD and three types of ECPs.

Types of Emergency Contraception

Intrauterine Device

- Cu-IUD

ECPs

- Ulipristal acetate (UPA) in a single dose (30 mg)
- Levonorgestrel in a single dose (1.5 mg) or as a split dose (1 dose of 0.75 mg of levonorgestrel followed by a second dose of 0.75 mg of levonorgestrel 12 hours later)
- Combined estrogen and progestin in 2 doses (Yuzpe regimen: 1 dose of 100 μg of ethinyl estradiol plus 0.50 mg of levonorgestrel followed by a second dose of 100 μg of ethinyl estradiol plus 0.50 mg of levonorgestrel 12 hours later)

Initiation of Emergency Contraception

Timing

Cu-IUD

- The Cu-IUD can be inserted within 5 days of the first act of unprotected sexual intercourse as an emergency contraceptive.
- In addition, when the day of ovulation can be estimated, the Cu-IUD can be inserted beyond 5 days after sexual intercourse, as long as insertion does not occur >5 days after ovulation.

ECPs

- ECPs should be taken as soon as possible within 5 days of unprotected sexual intercourse.

Comments and Evidence Summary. Cu-IUDs are highly effective as emergency contraception (*256*) and can be continued as regular contraception. UPA and levonorgestrel ECPs have similar effectiveness when taken within 3 days after unprotected sexual intercourse; however, UPA has been shown to be more effective than the levonorgestrel formulation 3–5 days after unprotected sexual intercourse (*257*). The combined estrogen and progestin regimen is less effective than UPA or levonorgestrel and also is associated with more frequent occurrence of side effects (nausea and vomiting) (*258*). The levonorgestrel formulation might be less effective than UPA among obese women (*257*).

Two studies of UPA use found consistent decreases in pregnancy rates when administered within 120 hours of unprotected sexual intercourse (*257*,*259*). Five studies found that the levonorgestrel and combined regimens decreased risk for pregnancy through the fifth day after unprotected sexual intercourse; however, rates of pregnancy were slightly higher when ECPs were taken after 3 days (*260–264*). A meta-analysis of levonorgestrel ECPs found that pregnancy rates were low when administered within 4 days after unprotected sexual intercourse but increased at 4–5 days (*265*) (Level of evidence: I to II-2, good to poor, direct).

Advance Provision of ECPs

- An advance supply of ECPs may be provided so that ECPs will be available when needed and can be taken as soon as possible after unprotected sexual intercourse.

Comments and Evidence Summary. A systematic review identified 17 studies that reported on safety or effectiveness of advance ECPs in adult or adolescent women (*26*). Any use of ECPs was two to seven times greater among women who received an advance supply of ECPs. However, a summary estimate (relative risk = 0.97; 95% confidence interval = 0.77–1.22) of five randomized controlled trials did not indicate a significant reduction in unintended pregnancies at 12 months with advance provision of ECPs. In the majority of studies among adults or adolescents, patterns of regular contraceptive use, pregnancy rates, and incidence of STDs did not vary between those who received advance ECPs and those who did not. Although available evidence supports the safety of advance

provision of ECPs, effectiveness of advance provision of ECPs in reducing pregnancy rates at the population level has not been demonstrated (Level of evidence: I to II-3, good to poor, direct).

Initiation of Regular Contraception After ECPs

UPA

- Any regular contraceptive method can be started immediately after the use of UPA.
- The woman needs to abstain from sexual intercourse or use barrier contraception for 14 days or until her next menses, whichever comes first.
- Advise the woman to have a pregnancy test if she does not have a withdrawal bleed within 3 weeks.

Levonorgestrel and Combined Estrogen and Progestin ECPs

- Any regular contraceptive method can be started immediately after the use of levonorgestrel or combined estrogen and progestin ECPs.
- The woman needs to abstain from sexual intercourse or use barrier contraception for 7 days.
- Advise the woman to have a pregnancy test if she does not have a withdrawal bleed within 3 weeks.

Comments and Evidence Summary. Data on when a woman can start regular contraception after ECPs are limited to expert opinion and product labeling (*27*). Theoretically, the effectiveness of systemic hormonal contraception might be decreased when administered concurrently or in close succession because of the antiprogestin properties of UPA (*266*,*267*); these theoretical concerns do not exist for combined estrogen and progestin or levonorgestrel formulations of ECPs. The resumption or initiation of regular hormonal contraception after ECP use involves consideration of the risk for pregnancy if ECPs fail and the risks for unintended pregnancy if contraception initiation is delayed until the subsequent menstrual cycle. If a woman is planning to initiate contraception after the next menstrual period after ECP use, the cycle in which ECPs are used might be shortened, prolonged, or involve unscheduled bleeding.

Prevention and Management of Nausea and Vomiting with ECP Use

Nausea and Vomiting

- Levonorgestrel and UPA ECPs cause less nausea and vomiting than combined estrogen and progestin ECPs.
- Routine use of antiemetics before taking ECPs is not recommended. Pretreatment with antiemetics may be considered depending on availability and clinical judgment.

Vomiting Within 3 Hours of Taking ECPs

- Another dose of ECP should be taken as soon as possible. Use of an antiemetic should be considered.

Comments and Evidence Summary. Many women do not experience nausea or vomiting when taking ECPs, and predicting which women will experience nausea or vomiting is difficult. Although routine use of antiemetics before taking ECPs is not recommended, antiemetics are effective in some women and can be offered when appropriate. Health-care providers who are deciding whether to offer antiemetics to women taking ECPs should consider the following: 1) women taking combined estrogen and progestin ECPs are more likely to experience nausea and vomiting than those who take levonorgestrel or UPA ECPs; 2) evidence indicates that antiemetics reduce the occurrence of nausea and vomiting in women taking combined estrogen and progestin ECPs; and 3) women who take antiemetics might experience other side effects from the antiemetics.

A systematic review examined incidence of nausea and vomiting with different ECP regimens and effectiveness of antinausea drugs in reducing nausea and vomiting with ECP use (*28*). The levonorgestrel regimen was associated with significantly less nausea than a nonstandard dose of UPA (50 mg) and the standard combined estrogen and progestin regimen (*268–270*). Use of the split-dose levonorgestrel showed no differences in nausea and vomiting compared with the single-dose levonorgestrel (*260*,*261*,*263*,*271*) (Level of evidence: I, good-fair, indirect). Two trials of antinausea drugs, meclizine and metoclopramide, taken before combined estrogen and progestin ECPs, reduced the severity of nausea (*272*,*273*). Significantly less vomiting occurred with meclizine but not metoclopramide (Level of evidence: I, good-fair, direct). No direct evidence was found regarding the effects of vomiting after taking ECPs.

Female Sterilization

Laparoscopic, abdominal, and hysteroscopic methods of female sterilization are available in the United States, and some of these procedures can be performed in an outpatient procedure or office setting. Fewer than 1 out of 100 women become pregnant in the first year after female sterilization (*59*). Because these methods are intended to be irreversible, all women should be appropriately counseled about the permanency of sterilization and the availability of highly

effective, long-acting, reversible methods of contraception. Female sterilization does not protect against STDs; consistent and correct use of male latex condoms reduces the risk for STDs, including HIV.

When Hysteroscopic Sterilization Is Reliable for Contraception

- Before a woman can rely on hysteroscopic sterilization for contraception, a hysterosalpingogram (HSG) must be performed 3 months after the sterilization procedure to confirm bilateral tubal occlusion.
- The woman should be advised that she needs to abstain from sexual intercourse or use additional contraceptive protection until she has confirmed bilateral tubal occlusion.

When Laparoscopic and Abdominal Approches Are Reliable for Contraception

- A woman can rely on sterilization for contraception immediately after laparoscopic and abdominal approaches. No additional contraceptive protection is needed.

Comments and Evidence Summary. HSG confirmation is necessary to confirm bilateral tubal occlusion after hysteroscopic sterilization. The inserts for the hysteroscopic sterilization system available in the United States are placed bilaterally into the fallopian tubes and require 3 months for adequate fibrosis and scarring leading to bilateral tubal occlusion. After hysteroscopic sterilization, advise the woman to correctly and consistently use an effective method of contraception while awaiting confirmation. If compliance with another method might be a problem, a woman and her health-care provider may consider DMPA injection at the time of sterilization to ensure adequate contraception for 3 months. Unlike laparoscopic and abdominal sterilizations, pregnancy risk beyond 7 years of follow-up has not been studied among women who received hysteroscopic sterilization.

Pregnancy risk with at least 10 years of follow-up has been studied among women who received laparoscopic and abdominal sterilizations (*274*,*275*). Although these methods are highly effective, pregnancies can occur many years after the procedure, and the risk for pregnancy is higher among younger women (*274*,*276*).

A systematic review was conducted to identify studies that reported whether pregnancies occurred after hysteroscopic sterilization (*29*). Twenty-four studies were identified that reported whether pregnancies occurred after hysteroscopic sterilization and found that very few pregnancies occurred among women with confirmed bilateral tubal occlusion; however, few studies include long-term follow-up, and none with follow-up for >7 years. Among women who had successful bilateral placement, most pregnancies that occurred after hysteroscopic sterilization were in women who did not have confirmed bilateral tubal occlusion at 3 months, either because of lack of follow up or misinterpretation of HSG results (*277–279*). Some pregnancies occurred within 3 months of placement, including among women who were already pregnant at the time of the procedure, women who did not use alternative contraception, or women who had failures of alternative contraception (*277*,*278*,*280–283*). Although these studies generally demonstrated high rates of bilateral placement, some pregnancies occurred as a result of lack of bilateral placement identified on later imaging (*277*,*278*,*280*,*281*,*283*,*284*). Most pregnancies occurred after deviations from FDA directions, which include placement in the early follicular phase of the menstrual cycle, imaging at 3 months to document proper placement, and use of effective alternative contraception until documented occlusion (Level of evidence: II-3, fair, direct).

Male Sterilization

Male sterilization, or vasectomy, is one of the few contraceptive methods available to men and can be performed in an outpatient procedure or office setting. Fewer than 1 woman out of 100 becomes pregnant in the first year after her male partner undergoes sterilization (*59*). Because male sterilization is intended to be irreversible, all men should be appropriately counseled about the permanency of sterilization and the availability of highly effective, long-acting, reversible methods of contraception for women. Male sterilization does not protect against STDs; consistent and correct use of male latex condoms reduces the risk for STDs, including HIV.

When Vasectomy Is Reliable for Contraception

- A semen analysis should be performed 8–16 weeks after a vasectomy to ensure the procedure was successful.
- The man should be advised that he should use additional contraceptive protection or abstain from sexual intercourse until he has confirmation of vasectomy success by postvasectomy semen analysis.

Other Postprocedure Recommendations

- The man should refrain from ejaculation for approximately 1 week after the vasectomy to allow for healing of surgical sites and, after certain methods of vasectomy, occlusion of the vas.

Comments and Evidence Summary. The Vasectomy Guideline Panel of the American Urological Association performed a systematic review of key issues concerning the practice of vasectomy (*285*). All English-language publications on vasectomy published during 1949–2011 were reviewed. For more information, see the American Urological Association *Vasectomy Guidelines* (available at http://www.auanet.org/education/vasectomy.cfm).

Motile sperm disappear within a few weeks after vasectomy (*286–289*). The time to azoospermia varies widely in different studies; however, by 12 weeks after the vasectomy, 80% of men have azoospermia, and almost all others have rare nonmotile sperm (defined as ≤100,000 nonmotile sperm per milliliter) (*285*). The number of ejaculations after vasectomy is not a reliable indicator of when azoospermia or rare nonmotile sperm will be achieved (*285*). Once azoospermia or rare nonmotile sperm has been achieved, patients can rely on the vasectomy for contraception, although not with 100% certainty. The risk for pregnancy after a man has achieved postvasectomy azoospermia is approximately one in 2,000 (*290–294*).

A median of 78% (range 33%–100%) of men return for a single postvasectomy semen analysis (*285*). In the largest cohorts that appear typical of North American vasectomy practice, approximately two thirds of men (55%–71%) return for at least one postvasectomy semen analysis (*291*,*295–299*). Assigning men an appointment after their vasectomy might improve compliance with follow-up (*300*).

When Women Can Stop Using Contraceptives

- Contraceptive protection is still needed for women aged >44 years if the woman wants to avoid pregnancy.

Comments and Evidence Summary. The age at which a woman is no longer at risk for pregnancy is not known. Although uncommon, spontaneous pregnancies occur among women aged >44 years. Both the American College of Obstetricians and Gynecologists and the North American Menopause Society recommend that women continue contraceptive use until menopause or age 50–55 years (*301*,*302*). The median age of menopause is approximately 51 years in North America (*301*) but can vary from ages 40 to 60 years (*303*). The median age of definitive loss of natural fertility is 41 years but can range up to age 51 years (*304*,*305*). No reliable laboratory tests are available to confirm definitive loss of fertility in a woman. The assessment of follicle-stimulating hormone levels to determine when a woman is no longer fertile might not be accurate (*301*).

Health-care providers should consider the risks for becoming pregnant in a woman of advanced reproductive age, as well as any risks of continuing contraception until menopause. Pregnancies among women of advanced reproductive age are at higher risk for maternal complications, such as hemorrhage, venous thromboembolism, and death, and fetal complications, such as spontaneous abortion, stillbirth, and congenital anomalies (*306–308*). Risks associated with continuing contraception, in particular risks for acute cardiovascular events (venous thromboembolism, myocardial infarction, or stroke) or breast cancer, also are important to consider. U.S. MEC states that on the basis of age alone, women aged >45 years can use POPs, implants, the LNG-IUD, or the Cu-IUD (U.S. MEC 1) (*5*). Women aged >45 years generally can use combined hormonal contraceptives and DMPA (U.S. MEC 2) (*5*). However, women in this age group might have chronic conditions or other risk factors that might render use of hormonal contraceptive methods unsafe; U.S. MEC might be helpful in guiding the safe use of contraceptives in these women.

The incidence of venous thromboembolism was higher among oral contraceptive users aged ≥45 years compared with younger oral contraceptive users in two studies (*309–311*); however, an interaction between hormonal contraception and increased age compared with baseline risk was not demonstrated (*309*,*310*) or was not examined (*311*). The relative risk for myocardial infarction was higher among all oral contraceptive users than in nonusers, although a trend of increased relative risk with increasing age was not demonstrated (*312*,*313*). No studies were found regarding the risk for stroke in COC users aged ≥45 years (Level of evidence: II-2, good to poor, direct).

A pooled analysis by the Collaborative Group on Hormonal Factors and Breast Cancer in 1996 (*314*) found small increased relative risks for breast cancer among women aged ≥45 years whose last use of combined hormonal contraceptives was <5 years previously and for those whose last use was 5–9 years previously. Seven more recent studies suggested small but nonsignificant increased relative risks for breast carcinoma in situ or breast cancer among women who had used oral contraceptives or DMPA when they were aged ≥40 years compared with those who had never used either method (*315–321*) (Level of evidence: II-2, fair, direct).

Conclusion

Women, men, and couples have increasing numbers of safe and effective choices for contraceptive methods, including LARC methods such as IUDs and implants, to reduce the risk for unintended pregnancy. However, with these expanded options

comes the need for evidence-based guidance to help health-care providers offer quality family planning care to their patients, including choosing the most appropriate contraceptive method for individual circumstances and using that method correctly, consistently, and continuously to maximize effectiveness. Removing unnecessary barriers can help patients access and successfully use contraceptive methods. Several medical barriers to initiating and continuing contraceptive methods might exist, such as unnecessary screening examinations and tests before starting the method (e.g., a pelvic examination before initiation of COCs), inability to receive the contraceptive on the same day as the visit (e.g., waiting for test results that might not be needed or waiting until the woman's next menstrual period to start use), and difficulty obtaining continued contraceptive supplies (e.g., restrictions on number of pill packs dispensed at one time). Removing unnecessary steps, such as providing prophylactic antibiotics at the time of IUD insertion or requiring unnecessary follow-up procedures, also can help patients access and successfully use contraception.

Most women can start most contraceptive methods at any time, and few examinations or tests, if any, are needed before starting a contraceptive method. Routine follow-up for most women includes assessment of her satisfaction with the contraceptive method, concerns about method use, and changes in health status or medications that could affect medical eligibility for continued use of the method. Because changes in bleeding patterns are one of the major reasons for discontinuation of contraception, recommendations are provided for the management of bleeding irregularities with various contraceptive methods. In addition, because women and health-care providers can be confused about the procedures for missed pills and dosing errors with the contraceptive patch and ring, the instructions are streamlined for easier use. ECPs and emergency use of the Cu-IUD are important options for women, and recommendations on using these methods, as well as starting regular contraception after use of emergency contraception, are provided. Male and female sterilization are highly effective methods of contraception for men, women, and couples who have completed childbearing; for men undergoing vasectomy and women undergoing a hysteroscopic sterilization procedure, additional contraceptive protection is needed until the success of the procedure can be confirmed.

CDC is committed to working with partners at the federal, national, and local levels to disseminate, implement, and evaluate the recommendations in U.S. SPR so that the information reaches health-care providers. Strategies for dissemination and implementation include collaborating with other federal agencies and professional and service organizations to widely distribute the recommendations through presentations, electronic distribution, newsletters, and other publications; development of provider tools and job aids to assist providers in implementing the new recommendations; and training activities for students, as well as for continuing education. CDC will conduct a survey of family planning health-care providers before and after release of this report to assess attitudes and practices related to contraceptive use. Results from this survey will assist CDC in evaluating the impact of these recommendations on the provision of contraceptives in the United States. Finally, CDC will continually monitor new scientific evidence and will update these recommendations as warranted by new evidence. Updates to the recommendations, as well as provider tools and other resources, are available on the CDC U.S. SPR website (http://www.cdc.gov/reproductivehealth/UnintendedPregnancy/USSPR.htm).

Acknowledgment

This report is based, in part, on the work of the Promoting Family Planning Team, Department of Reproductive Health and Research, World Health Organization, and its development of the *Selected Practice Recommendations for Contraceptive Use*.

References

1. Finer LB, Zolna MR. Unintended pregnancy in the United States: incidence and disparities, 2006. Contraception 2011;84:478–85.
2. Gipson JD, Koenig MA, Hindin MJ. The effects of unintended pregnancy on infant, child, and parental health: a review of the literature. Stud Fam Plann 2008;39:18–38.
3. Trussell J. The cost of unintended pregnancy in the United States. Contraception 2007;75:168–70.
4. Finer LB, Henshaw SK. Disparities in rates of unintended pregnancy in the United States, 1994 and 2001. Perspect Sex Reprod Health 2006;38:90–6.
5. CDC. U.S. medical eligibility criteria for contraceptive use. MMWR 2010;59(No. RR-4).
6. World Health Organization. Selected practice recommendations for contraceptive use. Second ed. Geneva: World Health Organization; 2004.
7. World Health Organization. Selected practice recommendations for contraceptive use. 2008 update. Geneva, Switzerland: World Health Organization; 2008. Available at http://whqlibdoc.who.int/hq/2008/WHO_RHR_08.17_eng.pdf.
8. Faculty of Sexual and Reproductive Health Care. UK Selected Practice Recommendations for Contraceptive Use. London, England: Faculty of Sexual and Reproductive Health Care; 2002. Available at http://www.fsrh.org/pdfs/SelectedPracticeRecommendations2002.pdf.
9. Moher D, Liberati A, Tetzlaff J, Altman DG. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. Int J Surg 2010;8:336–41.
10. Stroup DF, Berlin JA, Morton SC, et al. Meta-analysis of observational studies in epidemiology: a proposal for reporting. Meta-analysis Of Observational Studies in Epidemiology (MOOSE) group. JAMA 2000;283:2008–12.
11. Harris RP, Helfand M, Woolf SH, et al. Current methods of the US Preventive Services Task Force: a review of the process. Am J Prev Med 2001;20(Suppl):21–35.
12. Tepper NK, Marchbanks PA, Curtis KM. Use of a checklist to rule out pregnancy: a systematic review. Contraception 2013;87:661–5.

13. Whiteman MK, Tyler CP, Folger SG, Gaffield ME, Curtis KM. When can a woman have an intrauterine device inserted? A systematic review. Contraception 2013;87:666–73.
14. Tepper NK, Steenland MW, Marchbanks PA, Curtis KM. Laboratory screening prior to initiating contraception: a systematic review. Contraception 2013;87:645–9.
15. Tepper NK, Curtis KM, Steenland MW, Marchbanks PA. Physical examination prior to initiating hormonal contraception: a systematic review. Contraception 2013;87:650–4.
16. Steenland MW, Zapata LB, Brahmi D, Marchbanks PA, Curtis KM. The effect of follow-up visits or contacts after contraceptive initiation on method continuation and correct use. Contraception 2013;87:625–30.
17. Steenland MW, Zapata LB, Brahmi D, Marchbanks PA, Curtis KM. Appropriate follow up to detect potential adverse events after initiation of select contraceptive methods: a systematic review. Contraception 2013;87:611–24.
18. Godfrey EM, Folger SG, Jeng G, Jamieson DJ, Curtis KM. Treatment of bleeding irregularities in women with copper-containing IUDs: a systematic review. Contraception 2013;87:549–66.
19. Tepper NK, Steenland MW, Gaffield ME, Marchbanks PA, Curtis KM. Retention of intrauterine devices in women who acquire pelvic inflammatory disease: a systematic review. Contraception 2013;87:655–60.
20. Kapp N, Gaffield ME. Initiation of progestogen-only injectables on different days of the menstrual cycle and its effect on contraceptive effectiveness and compliance: a systematic review. Contraception 2013;87:576–82.
21. Tepper NK, Curtis KM, Steenland MW, Marchbanks PA. Blood pressure measurement prior to initiating hormonal contraception: a systematic review. Contraception 2013;87:631–8.
22. Brahmi D, Curtis KM. When can a woman start combined hormonal contraceptives (CHCs)? A systematic review. Contraception 2013; 87:524–38.
23. Steenland MW, Rodriguez MI, Marchbanks PA, Curtis KM. How does the number of oral contraceptive pill packs dispensed or prescribed affect continuation and other measures of consistent and correct use? A systematic review. Contraception 2013;87:605–10.
24. Zapata LB, Steenland MW, Brahmi D, Marchbanks PA, Curtis KM. Effect of missed combined hormonal contraceptives on contraceptive effectiveness: a systematic review. Contraception 2013;87:685–700.
25. Godfrey EM, Whiteman MK, Curtis KM. Treatment of unscheduled bleeding in women using extended- or continuous-use combined hormonal contraception: a systematic review. Contraception 2013;87:567–75.
26. Rodriguez MI, Curtis KM, Gaffield ML, Jackson E, Kapp N. Advance supply of emergency contraception: a systematic review. Contraception 2013;87:590–601.
27. Salcedo J, Rodriguez MI, Curtis KM, Kapp N. When can a woman resume or initiate contraception after taking emergency contraceptive pills? A systematic review. Contraception 2013;87:602–4.
28. Rodriguez MI, Godfrey EM, Warden M, Curtis KM. Prevention and management of nausea and vomiting with emergency contraception: a systematic review. Contraception 2013;87:583–9.
29. Cleary TP, Tepper NK, Cwiak C, et al. Pregnancies after hysteroscopic sterilization: a systematic review. Contraception 2013;87:539–48.
30. Tepper NK, Steenland MW, Marchbanks PA, Curtis KM. Hemoglobin measurement prior to initiating copper intrauterine devices: a systematic review. Contraception 2013;87:639–44.
31. Folger SG, Jamieson DJ, Godfrey EM, Zapata LB, Curtis KM. Evidence-based guidance on selected practice recommendations for contraceptive use: identification of research gaps. Contraception 2013;87:517–23.
32. CDC. Sexually transmitted diseases treatment guidelines. MMWR 2010;59(No. RR-12).
33. Mohllajee AP, Curtis KM, Flanagan RG, Rinehart W, Gaffield ML, Peterson HB. Keeping up with evidence a new system for WHO's evidence-based family planning guidance. Am J Prev Med 2005;28:483–90.
34. Stanback J, Nakintu N, Qureshi Z, Nasution M. Does assessment of signs and symptoms add to the predictive value of an algorithm to rule out pregnancy? J Fam Plann Reprod Health Care 2006;32:27–9.
35. Stanback J, Nanda K, Ramirez Y, Rountree W, Cameron SB. Validation of a job aid to rule out pregnancy among family planning clients in Nicaragua. Rev Panam Salud Publica 2008;23:116–8.
36. Stanback J, Qureshi Z, Sekadde-Kigondu C, Gonzalez B, Nutley T. Checklist for ruling out pregnancy among family-planning clients in primary care. Lancet 1999;354:566.
37. Torpey K, Mwenda L, Kabaso M, et al. Excluding pregnancy among women initiating antiretroviral therapy: efficacy of a family planning job aid. BMC Public Health 2010;10:249.
38. Cole LA, Ladner DG, Byrn FW. The normal variabilities of the menstrual cycle. Fertil Steril 2009;91:522–7.
39. Wilcox AJ, Dunson D, Baird DD. The timing of the "fertile window" in the menstrual cycle: day specific estimates from a prospective study. BMJ 2000;321:1259–62.
40. Donnet ML, Howie PW, Marnie M, Cooper W, Lewis M. Return of ovarian function following spontaneous abortion. Clin Endocrinol (Oxf) 1990;33:13–20.
41. Lahteenmaki P. Postabortal contraception. Ann Med 1993;25:185–9.
42. Stoddard A, Eisenberg DL. Controversies in family planning: timing of ovulation after abortion and the conundrum of postabortion intrauterine device insertion. Contraception 2011;84:119–21.
43. Jackson E, Glasier A. Return of ovulation and menses in postpartum, non-lactating women: a systematic review. Obstet Gynecol 2011;117:657–62.
44. Kennedy KI, Rivera R, McNeilly AS. Consensus statement on the use of breastfeeding as a family planning method. Contraception 1989;39:477–96.
45. Food and Drug Administration. 510(k) premarket notification. Silver Spring, MD: Food and Drug Administration; 2013. Available at http://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfPMN/pmn.cfm.
46. Cervinski MA, Gronowski AM. Reproductive-endocrine point-of-care testing: current status and limitations. Clin Chem Lab Med 2010;48:935–42.
47. Cole LA. Human chorionic gonadotropin tests. Expert Rev Mol Diagn 2009;9:721–47.
48. Eichner SF, Timpe EM. Urinary-based ovulation and pregnancy: point-of-care testing. Ann Pharmacother 2004;38:325–31.
49. Wilcox AJ, Baird DD, Dunson D, McChesney R, Weinberg CR. Natural limits of pregnancy testing in relation to the expected menstrual period. JAMA 2001;286:1759–61.
50. Korhonen J, Alfthan H, Ylostalo P, Veldhuis J, Stenman UH. Disappearance of human chorionic gonadotropin and its alpha- and beta-subunits after term pregnancy. Clin Chem 1997;43:2155–63.
51. Reyes FI, Winter JS, Faiman C. Postpartum disappearance of chorionic gonadotropin from the maternal and neonatal circulations. Am J Obstet Gynecol 1985;153:486–9.
52. Steier JA, Bergsjo P, Myking OL. Human chorionic gonadotropin in maternal plasma after induced abortion, spontaneous abortion, and removed ectopic pregnancy. Obstet Gynecol 1984;64:391–4.
53. Bracken MB. Oral contraception and congenital malformations in offspring: a review and meta-analysis of the prospective studies. Obstet Gynecol 1990;76:552–7.
54. Gray RH, Pardthaisong T. In utero exposure to steroid contraceptives and survival during infancy. Am J Epidemiol 1991;134:804–11.
55. Pardthaisong T, Gray RH. In utero exposure to steroid contraceptives and outcome of pregnancy. Am J Epidemiol 1991;134:795–803.
56. Jaffe B, Harlap S, Baras M, et al. Long-term effects of MPA on human progeny: intellectual development. Contraception 1988;37:607–19.
57. Pardthaisong T, Yenchit C, Gray R. The long-term growth and development of children exposed to Depo-Provera during pregnancy or lactation. Contraception 1992;45:313–24.
58. Brahmi D, Steenland MW, Renner RM, Gaffield ME, Curtis KM. Pregnancy outcomes with an IUD in situ: a systematic review. Contraception 2012;85:131–9.

59. Trussell J. Contraceptive failure in the United States. Contraception 2011;83:397–404.
60. Labbok M, Perez A, Valdes V, et al. The Lactational Amenorrhea Method (LAM): a postpartum introductory family planning method with policy and program implications. Adv Contracept 1994;10:93–109.
61. Teva Women's Health. ParaGard physicians prescribing information. Sellersville, PA: Teva Women's Health; 2013. Available at http://www.paragard.com/images/ParaGard_info.pdf.
62. Bayer HealthCare Pharmaceuticals. Mirena physicians prescribing information. Wayne, NJ: Bayer HealthCare Pharmaceuticals; 2013. Available at http://www.berlex.com/html/products/pi/Mirena_PI.pdf.
63. Farley TM, Rosenberg MJ, Rowe PJ, Chen JH, Meirik O. Intrauterine devices and pelvic inflammatory disease: an international perspective. Lancet 1992;339:785–8.
64. Mohllajee AP, Curtis KM, Peterson HB. Does insertion and use of an intrauterine device increase the risk of pelvic inflammatory disease among women with sexually transmitted infection? A systematic review. Contraception 2006;73:145–53.
65. Rivera R, Almonte H, Arreola M, et al. The effects of three different regimens of oral contraceptives and three different intrauterine devices on the levels of hemoglobin, serum iron and iron binding capacity in anemic women. Contraception 1983;27:311–27.
66. Effects of contraceptives on hemoglobin and ferritin. Task Force for Epidemiological Research on Reproductive Health, United Nations Development Programme/United Nations Population Fund/World Health Organization/World Bank Special Programme of Research, Development and Research Training in Human Reproduction, World Health Organization, Geneva, Switzerland. Contraception 1998;58:262–73.
67. Calzolari E, Guglielmo R, Viola F, Migliore L. Hematological parameters and iron therapy in women with IUDs: experimental study. Minerva Ginecol 1981;33:355–62.
68. Hassan EO, El-Husseini M, El-Nahal N. The effect of 1-year use of the CuT 380A and oral contraceptive pills on hemoglobin and ferritin levels. Contraception 1999;60:101–5.
69. Andersson K, Odlind V, Rybo G. Levonorgestrel-releasing and copper-releasing (Nova T) IUDs during five years of use: a randomized comparative trial. Contraception 1994;49:56–72.
70. Andrade AT, Pizarro E, Shaw STJ, Souza JP, Belsey EM, Rowe PJ. Consequences of uterine blood loss caused by various intrauterine contraceptive devices in South American women. World Health Organization Special Programme of Research, Development and Research Training in Human Reproduction. Contraception 1988;38:1–18.
71. Blum M, Ariel J, Zacharowitch D. Ferritin, a faithful reflection of iron deficiency in IUD wearers with mild vaginal spotting. Adv Contracept 1991;7:39–42.
72. El-sheikha Z, Hamza A, Mahmoud M. Menstrual blood loss of TCu-380 A and TCu-200 B IUDs. Popul Sci 1990;9:55–62.
73. Gallegost AJ, Aznar R, Merino G, Guizer E. Intrauterine devices and menstrual blood loss: a comparative study of eight devices during the first six months of use. Contraception 1978;17:153–61.
74. Gao J, Zeng S, Sun BL, et al. Menstrual blood loss, haemoglobin and ferritin concentration of Beijing women wearing steel ring, VCu 200, and TCu 220c IUDs. Contraception 1986;34:559–71.
75. Goh TH, Hariharan M. Effect of laparoscopic sterilization and insertion of Multiload Cu 250 and Progestasert IUDs on serum ferritin levels. Contraception 1983;28:329–36.
76. Goh TH, Hariharan M, Tan CH. A longitudinal study of serum iron indices and haemoglobin concentration following copper-IUD insertion. Contraception 1980;22:389–95.
77. Guillebaud J, Bonnar J, Morehead J, Matthews A. Menstrual blood-loss with intrauterine devices. Lancet 1976;1:387–90.
78. Haugan T, Skjeldestad FE, Halvorsen LE, Kahn H. A randomized trial on the clinical performance of Nova T380 and Gyne T380 Slimline copper IUDs. Contraception 2007;75:171–6.
79. Kivijarvi A, Timonen H, Rajamaki A, Gronroos M. Iron deficiency in women using modern copper intrauterine devices. Obstet Gynecol 1986;67:95–8.
80. Larsson B, Hamberger L, Rybo G. Influence of copper intrauterine contraceptive devices (Cu-7-IUD) on the menstrual blood-loss. Acta Obstet Gynecol Scand 1975;54:315–8.
81. Larsson G, Milsom I, Jonasson K, Lindstedt G, Rybo G. The long-term effects of copper surface area on menstrual blood loss and iron status in women fitted with an IUD. Contraception 1993;48:471–80.
82. Malmqvist R, Petersohn L, Bengtsson LP. Menstrual bleeding with copper-covered intrauterine contraceptive devices. Contraception 1974;9:627–33.
83. Milsom I, Andersson K, Jonasson K, Lindstedt G, Rybo G. The influence of the Gyne-T 380S IUD on menstrual blood loss and iron status. Contraception 1995;52:175–9.
84. Milsom I, Rybo G, Lindstedt G. The influence of copper surface area on menstrual blood loss and iron status in women fitted with an IUD. Contraception 1990;41:271–81.
85. Piedras J, Cordova MS, Perez-Toral MC, Lince E, Garza-Flores J. Predictive value of serum ferritin in anemia development after insertion of T Cu 220 intrauterine device. Contraception 1983;27:289–97.
86. Sivin I, Alvarez F, Diaz J, et al. Intrauterine contraception with copper and with levonorgestrel: a randomized study of the TCu 380Ag and levonorgestrel 20 mcg/day devices. Contraception 1984;30:443–56.
87. Sivin I. Two years of intrauterine contraception with levonorgestrel and with copper: a randomized comparison of the TCu 380Ag and levonorgestrel 20 mcg/day devices. Contraception 1987;35:245–55.
88. Tchai BS, Kim SW, Han JH, Im MW. Menstrual blood loss, iron nutriture, and the effects of Alza-T IPCS 52, T-Cu 220C and Lippes Loop D in Korean women. Seoul J Med 1987;28:51–9.
89. Wright EA, Kapu MM, Isichei UP. Zinc depletion and menorrhagia in Nigerians using copper T-200 intrauterine device. Trace Elem Med 1989;6:147–9.
90. Pleis JR, Lucas JW, Ward BW. Summary health statistics for U.S. adults: National Health Interview Survey, 2008. Vital Health Stat 10 2009;10:1–157.
91. CDC. Viral hepatitis surveillance, United States, 2009. Atlanta, GA: CDC; 2011. Available at http://www.cdc.gov/hepatitis/Statistics/2009Surveillance/PDFs/2009HepSurveillanceRpt.pdf.
92. Kohler BA, Ward E, McCarthy BJ, et al. Annual report to the nation on the status of cancer, 1975–2007, featuring tumors of the brain and other nervous system. J Natl Cancer Inst 2011;103:714–36.
93. Kapp N, Curtis KM. Hormonal contraceptive use among women with liver tumors: a systematic review. Contraception 2009;80:387–90.
94. Kapp N, Tilley IB, Curtis KM. The effects of hormonal contraceptive use among women with viral hepatitis or cirrhosis of the liver: a systematic review. Contraception 2009;80:381–6.
95. National Cancer Institute. National Cancer Institute at the National Institutes for Health [homepage]. Bethesda, MD: National Cancer Institute. Available at http://www.cancer.gov.
96. Watson M, Saraiya M, Benard V, et al. Burden of cervical cancer in the United States, 1998–2003. Cancer 2008;113(Suppl):2855–64.
97. CDC. Cervical cancer screening among women aged 18-30 years—United States, 2000–2010. MMWR 2013;61:1038–42.
98. Curtis KM, Nanda K, Kapp N. Safety of hormonal and intrauterine methods of contraception for women with HIV/AIDS: a systematic review. AIDS 2009;23(Suppl 1):S55–67.
99. Grimes DA, Schulz KF. Prophylactic antibiotics for intrauterine device insertion: a metaanalysis of the randomized controlled trials. Contraception 1999;60:57–63.

100. Wilson W, Taubert KA, Gewitz M, et al. Prevention of infective endocarditis: guidelines from the American Heart Association: a guideline from the American Heart Association Rheumatic Fever, Endocarditis, and Kawasaki Disease Committee, Council on Cardiovascular Disease in the Young, and the Council on Clinical Cardiology, Council on Cardiovascular Surgery and Anesthesia, and the Quality of Care and Outcomes Research Interdisciplinary Working Group. Circulation 2007;116:1736–54.
101. Canto De Cetina TE, Canto P, Ordonez Luna M. Effect of counseling to improve compliance in Mexican women receiving depot-medroxyprogesterone acetate. Contraception 2001;63:143–6.
102. Lei ZW, Wu SC, Garceau RJ, et al. Effect of pretreatment counseling on discontinuation rates in Chinese women given depo-medroxyprogesterone acetate for contraception. Contraception 1996;53:357–61.
103. Toppozada M, Anwar M, Abdel Rahman H, Gaweesh S. Control of IUD-induced bleeding by three non-steroidal anti-inflammatory drugs. Contracept Deliv Syst 1982;3:117–25.
104. Toppozada M, El-Attar A, El-Ayyat MA, Khamis Y. Management of uterine bleeding by PGs or their synthesis inhibitors. Adv Prostaglandin Thromboxane Res 1980;8:1459–63.
105. Wu S, Wang C, Cheng W, et al. Randomized multi-center study of baofuxin for treatment of bleeding side-effect induced by IUD. Reprod Contracept 2000;11:152–7.
106. Mercorio F, De Simone R, Di Carlo C, et al. Effectiveness and mechanism of action of desmopressin in the treatment of copper intrauterine device-related menorrhagia: a pilot study. Hum Reprod 2003;18:2319–22.
107. Pedron N, Lozano M, Aznar R. Treatment of hypermenorrhea with mefenamic acid in women using IUDs. Contracept Deliv Syst 1982;3:135–9.
108. Pizarro E, Mehech G, Hidalgo M, Munoz G, Romero C. Effect of meclofenamic acid on menstruation in hypermenorrheic women using intrauterine devices. Rev Chil Obstet Ginecol 1988;53:43–56.
109. Chinese National IUD Research Working Group. Prevention and treatment of IUD-induced menorrhagia with antifibrinolytic and antiprostaglandin drugs. Zhonghua Fu Chan Ke Za Zhi 1987;22:291–4, 312.
110. Di Lieto A, Catalano D, Miranda L, Paladini A. Action of a prostaglandin synthetase inhibitor on IUD associated uterine bleeding. Clin Exp Obstet Gynecol 1987;14:41–4.
111. Ylikorkala O, Viinikka L. Comparison between antifibrinolytic and antiprostaglandin treatment in the reduction of increased menstrual blood loss in women with intrauterine contraceptive devices. Br J Obstet Gynaecol 1983;90:78–83.
112. Food and Drug Administration. FDA prescribing information. Cyklokapron: tranexamic acid injection. New York, New York; 2011. Available at http://www.accessdata.fda.gov/drugsatfda_docs/label/2011/019281s030lbl.pdf.
113. Food and Drug Administration. FDA prescribing information. Lysteda: tranexamic acid tablets. Parsippany, New Jersey; 2011. Available at http://www.accessdata.fda.gov/drugsatfda_docs/label/2011/022430s002lbl.pdf.
114. Pedron N, Lozano M, Gallegos AJ. The effect of acetylsalicylic acid on menstrual blood loss in women with IUDs. Contraception 1987;36:295–303.
115. Hidalgo M, Bahamondes L, Perrotti M, Diaz J, Dantas-Monteiro C, Petta C. Bleeding patterns and clinical performance of the levonorgestrel-releasing intrauterine system (Mirena) up to two years. Contraception 2002; 65:129–32.
116. Larsson B, Wennergren M. Investigation of a copper-intrauterine device (Cu-IUD) for possible effect on frequency and healing of pelvic inflammatory disease. Contraception 1977;15:143–9.
117. Soderberg G, Lindgren S. Influence of an intrauterine device on the course of an acute salpingitis. Contraception 1981;24:137–43.
118. Teisala K. Removal of an intrauterine device and the treatment of acute pelvic inflammatory disease. Ann Med 1989;21:63–5.
119. Altunyurt S, Demir N, Posaci C. A randomized controlled trial of coil removal prior to treatment of pelvic inflammatory disease. Eur J Obstet Gynecol Reprod Biol 2003;107:81–4.
120. Stewart FH, Harper CC, Ellertson CE, Grimes DA, Sawaya GF, Trussell J. Clinical breast and pelvic examination requirements for hormonal contraception: current practice vs evidence. JAMA 2001;285:2232–9.
121. Mansour D, Korver T, Marintcheva-Petrova M, Fraser IS. The effects of Implanon on menstrual bleeding patterns. Eur J Contracept Reprod Health Care 2008;13(Suppl 1):13–28.
122. Abdel-Aleem H, D'Arcangues C, Vogelsong K, Gulmezoglu AM. Treatment of vaginal bleeding irregularities induced by progestin only contraceptives. Cochrane Database Syst Rev 2007;CD003449.
123. Archer DF, Philput CB, Levine AS, et al. Effects of ethinyl estradiol and ibuprofen compared to placebo on endometrial bleeding, cervical mucus and the postcoital test in levonorgestrel subcutaneous implant users. Contraception 2008;78:106–12.
124. Buasang K, Taneepanichskul S. Efficacy of celecoxib on controlling irregular uterine bleeding secondary to Jadelle use. J Med Assoc Thai 2009;92:301–7.
125. Phaliwong P, Taneepanichskul S. The effect of mefenamic acid on controlling irregular uterine bleeding second to Implanon use. J Med Assoc Thai 2004;87(Suppl 3):S64–8.
126. Weisberg E, Hickey M, Palmer D, et al. A randomized controlled trial of treatment options for troublesome uterine bleeding in Implanon users. Hum Reprod 2009;24:1852–61.
127. Weisberg E, Hickey M, Palmer D, et al. A pilot study to assess the effect of three short-term treatments on frequent and/or prolonged bleeding compared to placebo in women using Implanon. Hum Reprod 2006;21:295–302.
128. Cheng L, Zhu H, Wang A, Ren F, Chen J, Glasier A. Once a month administration of mifepristone improves bleeding patterns in women using subdermal contraceptive implants releasing levonorgestrel. Hum Reprod 2000;15:1969–72.
129. Alvarez-Sanchez F, Brache V, Thevenin F, Cochon L, Faundes A. Hormonal treatment for bleeding irregularities in Norplant implant users. Am J Obstet Gynecol 1996;174:919–22.
130. Diaz S, Croxatto HB, Pavez M, Belhadj H, Stern J, Sivin I. Clinical assessment of treatments for prolonged bleeding in users of Norplant implants. Contraception 1990;42:97–109.
131. Witjaksono J, Lau TM, Affandi B, Rogers PA. Oestrogen treatment for increased bleeding in Norplant users: preliminary results. Hum Reprod 1996;11(Suppl 2):109–14.
132. Abdel-Aleem H, Shaaban OM, Amin AF, Abdel-Aleem AM. Tamoxifen treatment of bleeding irregularities associated with Norplant use. Contraception 2005;72:432–7.
133. Phupong V, Sophonsritsuk A, Taneepanichskul S. The effect of tranexamic acid for treatment of irregular uterine bleeding secondary to Norplant use. Contraception 2006;73:253–6.
134. D'Arcangues C, Piaggio G, Brache V, et al. Effectiveness and acceptability of vitamin E and low-dose aspirin, alone or in combination, on Norplant-induced prolonged bleeding. Contraception 2004;70:451–62.
135. Subakir SB, Setiadi E, Affandi B, Pringgoutomo S, Freisleben HJ. Benefits of vitamin E supplementation to Norplant users–in vitro and in vivo studies. Toxicology 2000;148:173–8.
136. Petta C, Faundes A, Dunson T, Ramos M, DeLucio M, Faundes D. Timing of onset of contraceptive effectiveness in Depo-Provera users. II. Effects on ovarian function. Fertil Steril 1998;70:817–20.

137. Petta C, Faundes A, Dunson T, Ramos M, DeLucio M, Faundes D. Timing of onset of contraceptive effectiveness in Depo-Provera users: Part I. Changes in cervical mucus. Fertil Steril 1998;69:252–7.
138. Siriwongse T, Snidvongs W, Tantayaporn P, Leepipatpaiboon S. Effect of depo-medroxyprogesterone acetate on serum progesterone levels when administered on various cycle days. Contraception 1982;26:487–93.
139. Balkus J, Miller L. Same-day administration of depot-medroxyprogesterone acetate injection: a retrospective chart review. Contraception 2005; 71:395–8.
140. Morroni C, Grams M, Tiezzi L, Westhoff C. Immediate monthly combination contraception to facilitate initiation of the depot medroxyprogesterone acetate contraceptive injection. Contraception 2004;70:19–23.
141. Nelson A, Katz T. Initiation and continuation rates seen in 2-year experience with Same Day injections of DMPA. Contraception 2007;75:84–7.
142. Rickert V, Tiezzi L, Lipshutz J, Leon J, Vaughan R, Westhoff C. Depo Now: preventing unintended pregnancies among adolescents and young adults. J Adolesc Health 2007;40:22–8.
143. Sneed R, Westhoff C, Morroni C, Tiezzi L. A prospective study of immediate initiation of depo medroxyprogesterone acetate contraceptive injection. Contraception 2005;71:99–103.
144. Cox S, Dietz P, Bowman B, Posner S. Prevalence of chronic conditions among women of reproductive age. Third National Summit on Preconception Health and Health Care; Tampa, FL.
145. Diab KM, Zaki MM. Contraception in diabetic women: comparative metabolic study of Norplant, depot medroxyprogesterone acetate, low dose oral contraceptive pill and CuT380A. J Obstet Gynaecol Res 2000;26:17–26.
146. Garg SK, Chase HP, Marshall G, Hoops SL, Holmes DL, Jackson WE. Oral contraceptives and renal and retinal complications in young women with insulin-dependent diabetes mellitus. JAMA 1994;271:1099–102.
147. Grigoryan OR, Grodnitskaya EE, Andreeva EN, Chebotnikova TV, Melnichenko GA. Use of the NuvaRing hormone-releasing system in late reproductive-age women with type 1 diabetes mellitus. Gynecol Endocrinol 2008;24:99–104.
148. Kahn HS, Curtis KM, Marchbanks PA. Effects of injectable or implantable progestin-only contraceptives on insulin-glucose metabolism and diabetes risk. Diabetes Care 2003;26:216–25.
149. Lopez LM, Grimes DA, Schulz KF. Steroidal contraceptives: effect on carbohydrate metabolism in women without diabetes mellitus. Cochrane Database Syst Rev 2009;CD006133.
150. Rogovskaya S, Rivera R, Grimes DA, et al. Effect of a levonorgestrel intrauterine system on women with type 1 diabetes: a randomized trial. Obstet Gynecol 2005;105:811–5.
151. Troisi RJ, Cowie CC, Harris MI. Oral contraceptive use and glucose metabolism in a national sample of women in the united states. Am J Obstet Gynecol 2000;183:389–95.
152. Bonny A, Secic M, Cromer B. Early weight gain related to later weight gain in adolescents on depot medroxyprogesterone acetate. Obstet Gynecol 2011;117:793–7.
153. Le Y, Rahman M, Berenson A. Early weight gain predicting later weight gain among depot medroxyprogesterone acetate users. Obstet Gynecol 2009;114:279–84.
154. Risser WL. Weight change in adolescents who used hormonal contraception. J Adolesc Health 1999;24:433–6.
155. Paulen ME, Curtis KM. When can a woman have repeat progestogen-only injectables—depot medroxyprogesterone acetate or norethisterone enantate? Contraception 2009;80:391–408.
156. Pardthaisong T. Return of fertility after use of the injectable contraceptive Depo Provera: up-dated data analysis. J Biosoc Sci 1984;16:23–34.
157. Schwallie PC, Assenzo JR. The effect of depo-medroxyprogesterone acetate on pituitary and ovarian function, and the return of fertility following its discontinuation: a review. Contraception 1974;10:181–202.
158. Steiner MJ, Kwok C, Stanback J, et al. Injectable contraception: what should the longest interval be for reinjections? Contraception 2008;77:410–4.
159. Bassol S, Garza-Flores J, Cravioto MC, et al. Ovarian function following a single administration of depo-medroxyprogesterone acetate (DMPA) at different doses. Fertil Steril 1984;42:216–22.
160. Fotherby K, Koetsawang S, Mathrubutham M. Pharmacokinetic study of different doses of Depo Provera. Contraception 1980;22:527–36.
161. Fotherby K, Saxena BN, Shrimanker K, et al. A preliminary pharmacokinetic and pharmacodynamic evaluation of depot-medroxyprogesterone acetate and norethisterone oenanthate. Fertil Steril 1980;34:131–9.
162. Garza-Flores J, Cardenas S, Rodriguez V, Cravioto MC, Diaz-Sanchez V, Perez-Palacios G. Return to ovulation following the use of long-acting injectable contraceptives: a comparative study. Contraception 1985;31:361–6.
163. Jain J, Dutton C, Nicosia A, Wajszczuk C, Bode FR, Mishell DR Jr. Pharmacokinetics, ovulation suppression and return to ovulation following a lower dose subcutaneous formulation of Depo-Provera. Contraception 2004;70:11–8.
164. Lan PT, Aedo AR, Landgren BM, Johannisson E, Diczfalusy E. Return of ovulation following a single injection of depo-medroxyprogesterone acetate: a pharmacokinetic and pharmacodynamic study. Contraception 1984;29:1–18.
165. Ortiz A, Hirol M, Stanczyk FZ, Goebelsmann U, Mishell DR. Serum medroxyprogesterone acetate (MPA) concentrations and ovarian function following intramuscular injection of depo-MPA. J Clin Endocrinol Metab 1977;44:32–8.
166. Saxena BN, Dusitsin N, Tankeyoon M, Chaudhury RR. Return of ovulation after the cessation of depot-medroxy progesterone acetate treatment in Thai women. J Med Assoc Thai 1980;63:66–9.
167. Toh YC, Jain J, Rahnny MH, Bode FR, Ross D. Suppression of ovulation by a new subcutaneous depot medroxyprogesterone acetate (104 mg/0.65 mL) contraceptive formulation in Asian women. Clin Ther 2004;26:1845–54.
168. Hubacher D, Lopez L, Steiner MJ, Dorflinger L. Menstrual pattern changes from levonorgestrel subdermal implants and DMPA: systematic review and evidence-based comparisons. Contraception 2009;80:113–8.
169. Arias RD, Jain JK, Brucker C, Ross D, Ray A. Changes in bleeding patterns with depot medroxyprogesterone acetate subcutaneous injection 104 mg. Contraception 2006;74:234–8.
170. Dempsey A, Roca C, Westhoff C. Vaginal estrogen supplementation during Depo-Provera initiation: a randomized controlled trial. Contraception 2010;82:250–5.
171. Harel Z, Biro F, Kollar L, Riggs S, Flanagan P, Vaz R. Supplementation with vitamin C and/or vitamin B(6) in the prevention of Depo-Provera side effects in adolescents. J Pediatr Adolesc Gynecol 2002;15:153–8.
172. Nathirojanakun P, Taneepanichskul S, Sappakitkumjorn N. Efficacy of a selective COX-2 inhibitor for controlling irregular uterine bleeding in DMPA users. Contraception 2006;73:584–7.
173. Tantiwattanakul P, Taneepanichskul S. Effect of mefenamic acid on controlling irregular uterine bleeding in DMPA users. Contraception 2004;70:277–9.

174. Said S, Sadek W, Rocca M, et al. Clinical evaluation of the therapeutic effectiveness of ethinyl oestradiol and oestrone sulphate on prolonged bleeding in women using depot medroxyprogesterone acetate for contraception. World Health Organization, Special Programme of Research, Development and Research Training in Human Reproduction, Task Force on Long-acting Systemic Agents for Fertility Regulation. Hum Reprod 1996;11(Suppl 2):1–13.
175. Sadeghi-Bazargani H, Ehdaeivand F, Arshi S, Eftekhar H, Sezavar H, Amanati L. Low-dose oral contraceptive to re-induce menstrual bleeding in amenorrheic women on DMPA treatment: a randomized clinical trial. Med Sci Monit 2006;12:CR420–5.
176. CDC. Update to CDC's U.S. Medical Eligibility Criteria for Contraceptive Use, 2010: Revised recommendations for the use of contraceptive methods during the postpartum period. MMWR 2011;60:878–83.
177. Westhoff C, Morroni C, Kerns J, Murphy PA. Bleeding patterns after immediate vs. conventional oral contraceptive initiation: a randomized, controlled trial. Fertil Steril 2003;79:322–9.
178. Westhoff C, Heartwell S, Edwards S, et al. Initiation of oral contraceptives using a quick start compared with a conventional start—A randomized controlled trial. Obstet Gynecol 2007;109:1270–6.
179. Edwards SM, Zieman M, Jones K, Diaz A, Robilotto C, Westhoff C. Initiation of oral contraceptives—start now! J Adolesc Health 2008;43:432–6.
180. Baerwald AR, Olatunbosun OA, Pierson RA. Ovarian follicular development is initiated during the hormone-free interval of oral contraceptive use. Contraception 2004;70:371–7.
181. Baerwald AR, Pierson RA. Ovarian follicular development during the use of oral contraception: a review. J Obstet Gynaecol Can 2004;26:19–24.
182. Duijkers IJM, Klipping C, Verhoeven CHJ, Dieben TOM. Ovarian function with the contraceptive vaginal ring or an oral contraceptive: a randomized study. Human Reproduction 2004;19):2668–73.
183. Killick S, Eyong E, Elstein M. Ovarian follicular development in oral contraceptive cycles. Fertil Steril 1987;48:409–13.
184. Molloy BG, Coulson KA, Lee JM, Watters JK. "Missed pill" conception: fact or fiction? Br Med J (Clin Res Ed) 1985;290:1474–5.
185. Mulders TM, Dieben TO, Bennink HJ. Ovarian function with a novel combined contraceptive vaginal ring. Hum Reprod 2002;17:2594–9.
186. Schwartz JL, Creinin MD, Pymar HC, Reid L. Predicting risk of ovulation in new start oral contraceptive users. Obstet Gynecol 2002;99:177–82.
187. Sitavarin S, Jaisamrarn U, Taneepanichskul S. A randomized trial on the impact of starting day on ovarian follicular activity in very low dose oral contraceptive pills users. J Med Assoc Thai 2003;86:442–8.
188. Smith SK, Kirkman RJ, Arce BB, McNeilly AS, Loudon NB, Baird DT. The effect of deliberate omission of Trinordiol or Microgynon on the hypothalamo-pituitary-ovarian axis. Contraception 1986;34:513–22.
189. Taylor DR, Anthony FW, Dennis KJ. Suppression of ovarian function by Microgynon 30 in day 1 and day 5 "starters." Contraception 1986; 33:463–71.
190. Lara-Torre E, Schroeder B. Adolescent compliance and side effects with Quick Start initiation of oral contraceptive pills. Contraception 2002;66:81–5.
191. Murthy AS, Creinin MD, Harwood B, Schreiber CA. Same-day initiation of the transdermal hormonal delivery system (contraceptive patch) versus traditional initiation methods. Contraception 2005;72:333–6.
192. Westhoff C, Osborne L, Schafer J, Morroni C. Bleeding patterns after immediate initiation of an oral compared with a vaginal hormonal contraceptive. Obstet Gynecol 2005;106:89–96.
193. Yeshaya A, Orvieto R, Kaplan B, et al. Flexible starting schedule for oral contraception: effect on the incidence of breakthrough bleeding and compliance. Eur J Contracept Reprod Health Care 1998;3:121–3.
194. Yeshaya A, Orvieto R, Kauschansky A, et al. A delayed starting schedule of oral contraception: the effect on the incidence of breakthrough bleeding and compliance in women. Eur J Contracept Reprod Health Care 1996;1:263–5.
195. Westhoff C, Kerns J, Morroni C, Cushman LF, Tiezzi L, Murphy PA. Quick start: novel oral contraceptive initiation method. Contraception 2002;66:141–5.
196. Acute myocardial infarction and combined oral contraceptives: results of an international multicentre case-control study. WHO Collaborative Study of Cardiovascular Disease and Steroid Hormone Contraception. Lancet 1997;349:1202–9.
197. Dunn N, Thorogood M, Faragher B, et al. Oral contraceptives and myocardial infarction: results of the MICA case-control study. BMJ 1999;318:1579–83.
198. Lewis MA, Heinemann LA, Spitzer WO, MacRae KD, Bruppacher R. The use of oral contraceptives and the occurrence of acute myocardial infarction in young women. Results from the Transnational Study on Oral Contraceptives and the Health of Young Women. Contraception 1997;56:129–40.
199. Ischaemic stroke and combined oral contraceptives: results of an international, multicentre, case-control study. WHO Collaborative Study of Cardiovascular Disease and Steroid Hormone Contraception. Lancet 1996;348:498–505.
200. Heinemann LA, Lewis MA, Spitzer WO, Thorogood M, Guggenmoos-Holzmann I, Bruppacher R. Thromboembolic stroke in young women. A European case-control study on oral contraceptives. Transnational Research Group on Oral Contraceptives and the Health of Young Women. Contraception 1998;57:29–37.
201. Haemorrhagic stroke, overall stroke risk, and combined oral contraceptives: results of an international, multicentre, case-control study. WHO Collaborative Study of Cardiovascular Disease and Steroid Hormone Contraception. Lancet 1996;348:505–10.
202. Berenson AB, Rahman M, Wilkinson G. Effect of injectable and oral contraceptives on serum lipids. Obstet Gynecol 2009;114:786–94.
203. Dilbaz B, Ozdegirmenci O, Caliskan E, Dilbaz S, Haberal A. Effect of etonogestrel implant on serum lipids, liver function tests and hemoglobin levels. Contraception 2010;81:510–4.
204. Nelson A. Combined oral contraceptives. In: Hatcher RA, Trussel J, Nelson A, Cates W Jr, Stewart FH, Kowal D, eds. Contraceptive technology. 19th ed. New York, NY: Ardent Media, Inc; 2007:193–270.
205. Mohllajee AP, Curtis KM, Martins SL, Peterson HB. Does use of hormonal contraceptives among women with thrombogenic mutations increase their risk of venous thromboembolism? A systematic review. Contraception 2006;73:166–78.
206. Wu O, Robertson L, Twaddle S, et al. Screening for thrombophilia in high-risk situations: systematic review and cost-effectiveness analysis. The Thrombosis: Risk and Economic Assessment of Thrombophilia Screening (TREATS) study. Health technology assessment (Winchester, England) 2006;10:1–110.
207. Blickstein D, Blickstein I. Oral contraception and thrombophilia. Curr Opin Obstet Gynecol 2007;19:370–6.
208. Wu O, Greer IA. Is screening for thrombophilia cost-effective? Curr Opin Hematol 2007;14:500–3.
209. Foster D, Hulett D, Bradsberry M, Darney P, Policar M. Number of oral contraceptive pill packages dispensed and subsequent unintended pregnancies. Obstet Gynecol 2011;117:566–72.
210. Foster D, Parvataneni R, de Bocanegra H, Lewis C, Bradsberry M, Darney P. Number of oral contraceptive pill packages dispensed, method continuation, and costs. Obstet Gynecol 2006;108:1107–14.

211. White KO, Westhoff C. The effect of pack supply on oral contraceptive pill continuation: a randomized controlled trial. Obstet Gynecol 2011; 118:615–22.
212. Chin Quee D, Cuthbertson C, Janowitz B. Over-the-counter pill provision: evidence from Jamaica. Stud Fam Plann 2006;37:99–110.
213. Anttila L, Kunz M, Marr J. Bleeding pattern with drospirenone 3 mg+ethinyl estradiol 20 mcg 24/4 combined oral contraceptive compared with desogestrel 150 mcg+ethinyl estradiol 20 mcg 21/7 combined oral contraceptive. Contraception 2009;80:445–51.
214. Chowdhury V, Joshi UM, Gopalkrishna K, Betrabet S, Mehta S, Saxena BN. 'Escape' ovulation in women due to the missing of low dose combination oral contraceptive pills. Contraception 1980;22:241–7.
215. Christin-Maitre S, Serfaty D, Chabbert-Buffet N, Ochsenbein E, Chassard D, Thomas JL. Comparison of a 24-day and a 21-day pill regimen for the novel combined oral contraceptive, nomegestrol acetate and 17beta-estradiol (NOMAC/E2): a double-blind, randomized study. Hum Reprod 2011;26:1338–47.
216. Creinin MD, Lippman JS, Eder SE, Godwin AJ, Olson W. The effect of extending the pill-free interval on follicular activity: triphasic norgestimate/35 micro g ethinyl estradiol versus monophasic levonorgestrel/20 micro g ethinyl estradiol. Contraception 2002;66:147–52.
217. Dinger J, Minh TD, Buttmann N, Bardenheuer K. Effectiveness of oral contraceptive pills in a large U.S. cohort comparing progestogen and regimen. Obstet Gynecol 2011;117:33–40.
218. Elomaa K, Lahteenmaki P. Ovulatory potential of preovulatory sized follicles during oral contraceptive treatment. Contraception 1999;60:275–9.
219. Elomaa K, Rolland R, Brosens I, et al. Omitting the first oral contraceptive pills of the cycle does not automatically lead to ovulation. Am J Obstet Gynecol 1998;179:41–6.
220. Endrikat J, Wessel J, Rosenbaum P, Dusterberg B. Plasma concentrations of endogenous hormones during one regular treatment cycle with a low-dose oral contraceptive and during two cycles with deliberate omission of two tablets. Gynecol Endocrinol 2004;18:318–26.
221. Hamilton CJ, Hoogland HJ. Longitudinal ultrasonographic study of the ovarian suppressive activity of a low-dose triphasic oral contraceptive during correct and incorrect pill intake. Am J Obstet Gynecol 1989;161:1159–62.
222. Hedon B, Cristol P, Plauchut A, et al. Ovarian consequences of the transient interruption of combined oral contraceptives. Int J Fertil 1992;37:270–6.
223. Killick SR. Ovarian follicles during oral contraceptive cycles: their potential for ovulation. Fertil Steril 1989;52:580–2.
224. Killick SR, Bancroft K, Oelbaum S, Morris J, Elstein M. Extending the duration of the pill-free interval during combined oral contraception. Adv Contracept 1990;6:33–40.
225. Klipping C, Duijkers I, Trummer D, Marr J. Suppression of ovarian activity with a drospirenone-containing oral contraceptive in a 24/4 regimen. Contraception 2008;78:16–25.
226. Klipping C, Marr J. Effects of two combined oral contraceptives containing ethinyl estradiol 20 microg combined with either drospirenone or desogestrel on lipids, hemostatic parameters and carbohydrate metabolism. Contraception 2005;71:409–16.
227. Landgren BM, Csemiczky G. The effect of follicular growth and luteal function of "missing the pill." A comparison between a monophasic and a triphasic combined oral contraceptive. Contraception 1991;43:149–59.
228. Landgren BM, Diczfalusy E. Hormonal consequences of missing the pill during the first two days of three consecutive artificial cycles. Contraception 1984;29:437–46.
229. Letterie GS. A regimen of oral contraceptives restricted to the periovulatory period may permit folliculogenesis but inhibit ovulation. Contraception 1998;57:39–44.
230. Letterie GS, Chow GE. Effect of "missed" pills on oral contraceptive effectiveness. Obstet Gynecol 1992;79:979–82.
231. Morris SE, Groom GV, Cameron ED, Buckingham MS, Everitt JM, Elstein M. Studies on low dose oral contraceptives: plasma hormone changes in relation to deliberate pill ('Microgynon 30') omission. Contraception 1979;20:61–9.
232. Nakajima ST, Archer DF, Ellman H. Efficacy and safety of a new 24-day oral contraceptive regimen of norethindrone acetate 1 mg/ethinyl estradiol 20 micro g (Loestrin 24 Fe). Contraception 2007;75:16–22.
233. Nuttall ID, Elstein M, McCafferty E, Seth J, Cameron ED. The effect of ethinyl estradiol 20 mcg and levonorgestrel 250 mcg on the pituitary-ovarian function during normal tablet-taking and when tablets are missed. Contraception 1982;26:121–35.
234. Pierson RA, Archer DF, Moreau M, Shangold GA, Fisher AC, Creasy GW. Ortho Evra/Evra versus oral contraceptives: follicular development and ovulation in normal cycles and after an intentional dosing error. Fertil Steril 2003;80:34–42.
235. Rible RD, Taylor D, Wilson ML, Stanczyk FZ, Mishell DR Jr. Follicular development in a 7-day versus 4-day hormone-free interval with an oral contraceptive containing 20 mcg ethinyl estradiol and 1 mg norethindrone acetate. Contraception 2009;79:182–8.
236. Schlaff WD, Lynch AM, Hughes HD, Cedars MI, Smith DL. Manipulation of the pill-free interval in oral contraceptive pill users: the effect on follicular suppression. Am J Obstet Gynecol 2004;190:943–51.
237. Spona J, Elstein M, Feichtinger W, et al. Shorter pill-free interval in combined oral contraceptives decreases follicular development. Contraception 1996;54:71–7.
238. Sullivan H, Furniss H, Spona J, Elstein M. Effect of 21-day and 24-day oral contraceptive regimens containing gestodene (60 microg) and ethinyl estradiol (15 microg) on ovarian activity. Fertil Steril 1999;72:115–20.
239. Wang E, Shi S, Cekan SZ, Landgren BM, Diczfalusy E. Hormonal consequences of "missing the pill." Contraception 1982;26:545–66.
240. Willis SA, Kuehl TJ, Spiekerman AM, Sulak PJ. Greater inhibition of the pituitary–ovarian axis in oral contraceptive regimens with a shortened hormone-free interval. Contraception 2006;74:100–3.
241. Abrams LS, Skee DM, Natarajan J, et al. Pharmacokinetics of norelgestromin and ethinyl estradiol delivered by a contraceptive patch (Ortho Evra/Evra) under conditions of heat, humidity, and exercise. J Clin Pharmacol 2001;41:1301–9.
242. Ahrendt HJ, Nisand I, Bastianelli C, et al. Efficacy, acceptability and tolerability of the combined contraceptive ring, NuvaRing, compared with an oral contraceptive containing 30 microg of ethinyl estradiol and 3 mg of drospirenone. Contraception 2006;74:451–7.
243. Bjarnadottir RI, Tuppurainen M, Killick SR. Comparison of cycle control with a combined contraceptive vaginal ring and oral levonorgestrel/ethinyl estradiol. Am J Obstet Gynecol 2002;186:389–95.
244. Brucker C, Karck U, Merkle E. Cycle control, tolerability, efficacy and acceptability of the vaginal contraceptive ring, NuvaRing: results of clinical experience in Germany. Eur J Contracept Reprod Health Care 2008;13:31–8.
245. Dieben TO, Roumen FJ, Apter D. Efficacy, cycle control, and user acceptability of a novel combined contraceptive vaginal ring. Obstet Gynecol 2002;100:585–93.
246. Mulders TM, Dieben TO. Use of the novel combined contraceptive vaginal ring NuvaRing for ovulation inhibition. Fertil Steril 2001;75:865–70.
247. Guilbert E, Boroditsky R, Black A, et al. Canadian Consensus Guideline on Continuous and Extended Hormonal Contraception, 2007. J Obstet Gynaecol Can 2007;29(Suppl 2):S1–32.

248. Wiegratz I, Stahlberg S, Manthey T, et al. Effect of extended-cycle regimen with an oral contraceptive containing 30 mcg ethinylestradiol and 2 mg dienogest on bleeding patterns, safety, acceptance and contraceptive efficacy. Contraception 2011;84:133–43.
249. Sulak PJ, Kuehl TJ, Coffee A, Willis S. Prospective analysis of occurrence and management of breakthrough bleeding during an extended oral contraceptive regimen. Am J Obstet Gynecol 2006;195:935–41.
250. Sulak PJ, Smith V, Coffee A, Witt I, Kuehl AL, Kuehl TJ. Frequency and management of breakthrough bleeding with continuous use of the transvaginal contraceptive ring: a randomized controlled trial. Obstet Gynecol 2008;112:563–71.
251. Kaneshiro B, Edelman A, Carlson N, Morgan K, Nichols M, Jensen J. Treatment of unscheduled bleeding in continuous oral contraceptive users with doxycycline: a randomized controlled trial. Obstet Gynecol 2010; 115:1141–9.
252. McCann MF, Potter LS. Progestin-only oral contraception: a comprehensive review. Contraception 1994;50:S1–195.
253. Arevalo M, Jennings V, Sinai I. Efficacy of a new method of family planning: the Standard Days Method. Contraception 2002;65:333–8.
254. Arevalo M, Sinai I, Jennings V. A fixed formula to define the fertile window of the menstrual cycle as the basis of a simple method of natural family planning. Contraception 1999;60:357–60.
255. Wilcox AJ, Dunson DB, Weinberg CR, Trussell J, Baird DD. Likelihood of conception with a single act of intercourse: providing benchmark rates for assessment of post-coital contraceptives. Contraception 2001;63:211–5.
256. Cleland K, Zhu H, Goldstuck N, Cheng L, Trussell J. The efficacy of intrauterine devices for emergency contraception: a systematic review of 35 years of experience. Hum Reprod 2012;27:1994–2000.
257. Glasier AF, Cameron ST, Fine PM, et al. Ulipristal acetate versus levonorgestrel for emergency contraception: a randomised non-inferiority trial and meta-analysis. Lancet 2010;375:555–62.
258. Raymond E, Taylor D, Trussell JMJS. Minimum effectiveness of the levonorgestrel regimen of emergency contraception. Contraception 2004;69:79–81.
259. Fine P, Mathe H, Ginde S, Cullins V, Morfesis J, Gainer E. Ulipristal acetate taken 48–120 hours after intercourse for emergency contraception. Obstet Gynecol 2010;115:257–63.
260. Dada OA, Godfrey EM, Piaggio G, von Hertzen H. A randomized, double-blind, noninferiority study to compare two regimens of levonorgestrel for emergency contraception in Nigeria. Contraception 2010;82:373–8.
261. Ngai SW, Fan S, Li S, et al. A randomized trial to compare 24 h versus 12 h double dose regimen of levonorgestrel for emergency contraception. Hum Reprod 2005;20:307–11.
262. Ellertson C, Evans M, Ferden S, et al. Extending the time limit for starting the Yuzpe regimen of emergency contraception to 120 hours. Obstet Gynecol 2003;101:1168–71.
263. Von Hertzen H, Piaggio G, Ding J, et al. Low dose mifepristone and two regimens of levonorgestrel for emergency contraception: a WHO multicentre randomised trial. Lancet 2002;360:1803–10.
264. Rodrigues I, Grou F, Joly J. Effectiveness of emergency contraceptive pills between 72 and 120 hours after unprotected sexual intercourse. Am J Obstet Gynecol 2001;184:531–7.
265. Piaggio G, Kapp N, von Hertzen H. Effect on pregnancy rates of the delay in the administration of levonorgestrel for emergency contraception: a combined analysis of four WHO trials. Contraception 2011;84:35–9.
266. Watson Pharma Inc. Prescribing Information for Ella. 2010. Morristown, NJ.
267. Emergency contraceptive pills. Medical and service delivery guidelines. 2012. Available at http://www.cecinfo.org/custom-content/uploads/2013/03/Medical-and-Service-Delivery-Guidelines-English-2012.pdf.
268. Creinin MD, Schlaff W, Archer DF, et al. Progesterone receptor modulator for emergency contraception - A randomized controlled trial. Obstet Gynecol 2006;108:1089–97.
269. Farajkhoda T, Khoshbin A, Enjezab B, Bokaei M, Zarchi MK. Assessment of two emergency contraceptive regimens in Iran: levonorgestrel versus the Yuzpe. Niger J Clin Pract 2009;12:450–2.
270. Ho PC, Kwan MSW. A prospective randomized comparison of levonorgestrel with the Yuzpe regimen in post-coital contraception. Hum Reprod 1993;8:389–92.
271. Arowojolu AO, Okewole IA, Adekunle AO. Comparative evaluation of the effectiveness and safety of two regimens of levonorgestrel for emergency contraception in Nigerians. Contraception 2002;66:269–73.
272. Raymond EG, Creinin MD, Barnhart KT, Lovvorn AE, Rountree RW, Trussell J. Meclizine for prevention of nausea associated with use of emergency contraceptive pills: a randomized trial. Obstet Gynecol 2000;95:271–7.
273. Ragan RE, Rock RW, Buck HW. Metoclopramide pretreatment attenuates emergency contraceptive-associated nausea. Am J Obstet Gynecol 2003;188:330–3.
274. Peterson HB, Xia Z, Hughes JM, Wilcox LS, Tylor LR, Trussell J. The risk of pregnancy after tubal sterilization: findings from the U.S. Collaborative Review of Sterilization. Am J Obstet Gynecol 1996;174:1161–8, 8–70.
275. Peterson HB. Sterilization. Obstet Gynecol 2008;111:189–203.
276. Lawrie TA, Nardin JM, Kulier R, Boulvain M. Techniques for the interruption of tubal patency for female sterilisation. Cochrane Database Syst Rev 2011; (2):CD003034.
277. Veersema S, Vleugels MP, Moolenaar LM, Janssen CA, Brolmann HA. Unintended pregnancies after Essure sterilization in the Netherlands. Fertil Steril 2010;93:35–8.
278. Levy B, Levie MD, Childers ME. A summary of reported pregnancies after hysteroscopic sterilization. J Minim Invasive Gynecol 2007;14:271–4.
279. Legendre G, Levaillant JM, Faivre E, Deffieux X, Gervaise A, Fernandez H. 3D ultrasound to assess the position of tubal sterilization microinserts. Hum Reprod 2011;26:2683–9.
280. Duffy S, Marsh F, Rogerson L, et al. Female sterilisation: a cohort controlled comparative study of ESSURE versus laparoscopic sterilisation. BJOG 2005;112:1522–8.
281. Levie MD, Chudnoff SG. Prospective analysis of office-based hysteroscopic sterilization. J Minim Invasive Gynecol 2006;13:98–101.
282. Arjona JE, Mino M, Cordon J, Povedano B, Pelegrin B, Castelo-Branco C. Satisfaction and tolerance with office hysteroscopic tubal sterilization. Fertil Steril 2008;90:1182–6.
283. Grosdemouge I, Engrand JB, Dhainault C, et al. La pratique francaise de la pose des implants de sterilisation tubaire Essure. Gynecol Obstet Fertil 2009;37:389–95.
284. Shavell VI, Abdallah ME, Diamond MP, Berman JM. Placement of a permanent birth control device at a university medical center. J Reprod Med 2009;54:218–22.
285. American Urological Association. Vasectomy guideline 2012. Available at http://www.auanet.org/education/vasectomy.cfm.
286. Bedford J, Zelikovsky G. Viability of spermatozoa in the human ejaculate after vasectomy. Fertil Steril 1979;32:460–3.
287. Edwards I. Earlier testing after vasectomy, based on the absence of motile sperm. Fertil Steril 1993;59:431–6.
288. Jouannet P, David G. Evolution of the properties of semen immediately following vasectomy. Fertil Steril 1978;29:435–41.
289. Labrecque M, Hays M, Chen-Mok M, Barone M, Sokal D. Frequency and patterns of early recanalization after vasectomy. BMC Urol 2006;6:25.
290. Philp T, Guillebaud J, Budd D. Complications of vasectomy: review of 16,000 patients. Br J Urol 1984;56:745–8.

291. Alderman PM. The lurking sperm. A review of failures in 8879 vasectomies performed by one physician. JAMA 1988;259:3142–4.
292. Black T, Francome C. The evolution of the Marie Stopes electrocautery no-scalpel vasectomy procedure. J Fam Plann Reprod Health Care 2002;28:137–8.
293. Davies AH, Sharp RJ, Cranston D, Mitchell RG. The long-term outcome following "special clearance" after vasectomy. Br J Urol 1990;66:211–2.
294. Philp T, Guillebaud J, Budd D. Late failure of vasectomy after two documented analyses showing azoospermic semen. Br Med J (Clin Res Ed) 1984;289:77–9.
295. Belker A, Sexter M, Sweitzer S, Raff M. The high rate of noncompliance for post-vasectomy semen examination: medical and legal considerations. J Urol 1990;144:284–6.
296. Chawla A, Bowles B, Zini A. Vasectomy follow-up: clinical significance of rare nonmotile sperm in postoperative semen analysis. Urology 2004;64:1212–5.
297. Labrecque M, Bedard L, Laperriere L. Efficacy and complications associated with vasectomies in two clinics in the Quebec region. Can Fam Physician 1998;44:1860–6.
298. Labrecque M, Nazerali H, Mondor M, Fortin V, Nasution M. Effectiveness and complications associated with 2 vasectomy occlusion techniques. J Urol 2002;168:2495–8.
299. Maatman TJ, Aldrin L, Carothers GG. Patient noncompliance after vasectomy. Fertil Steril 1997;68:552–5.
300. Dhar NB, Jones JS, Bhatt A, Babineau D. A prospective evaluation of the impact of scheduled follow-up appointments with compliance rates after vasectomy. BJU Int 2007;99:1094–7.
301. Hillard PJ, Berek JS, Barss VA, et al. Guidelines for women's health care: a resource manual. 3rd ed. Washington, DC: American College of Obstetricians and Gynecologists; 2007.
302. The North American Menopause Society. Prescription hormonal therapies. Menopause practice: a clinician's guide. 4th ed. 2010.
303. Te Velde ER, Pearson PL. The variability of female reproductive ageing. Hum Reprod Update 2002;8:141–54.
304. Broekmans FJ, Soules MR, Fauser BC. Ovarian aging: mechanisms and clinical consequences. Endocr Rev 2009;30:465–93.
305. Wood JW. Fecundity and natural fertility in humans. Oxf Rev Reprod Biol 1989;11:61–109.
306. Bateman BT, Simpson LL. Higher rate of stillbirth at the extremes of reproductive age: a large nationwide sample of deliveries in the United States. Am J Obstet Gynecol 2006;194:840–5.
307. Berg CJ, Callaghan WM, Syverson C, Henderson Z. Pregnancy-related mortality in the United States, 1998 to 2005. Obstet Gynecol 2010; 116:1302–9.
308. Balasch J, Gratacós E. Delayed childbearing: effects on fertility and the outcome of pregnancy. Curr Opin Obstet Gynecol 2012;24:187–93.
309. Lidegaard O, Lokkegaard E, Svendsen AL, Agger C. Hormonal contraception and risk of venous thromboembolism: national follow-up study. BMJ 2009;339:b2890.
310. Lidegaard O, Nielsen LH, Skovlund CW, Skjeldestad FE, Lokkegaard E. Risk of venous thromboembolism from use of oral contraceptives containing different progestogens and oestrogen doses: Danish cohort study, 2001–9. BMJ 2011;343:d6423.
311. Nightingale AL, Lawrenson RA, Simpson EL, Williams TJ, MacRae KD, Farmer RD. The effects of age, body mass index, smoking and general health on the risk of venous thromboembolism in users of combined oral contraceptives. Eur J Contracept Reprod Health Care 2000;5:265–74.
312. Slone D, Shapiro S, Kaufman DW, Rosenberg L, Miettinen OS, Stolley PD. Risk of myocardial infarction in relation to current and discontinued use of oral contraceptives. N Engl J Med 1981;305:420–4.
313. Tanis BC, van den Bosch MA, Kemmeren JM, et al. Oral contraceptives and the risk of myocardial infarction. N Engl J Med 2001;345:1787–93.
314. Collaborative Group on Hormonal Factors in Breast Cancer. Breast cancer and hormonal contraceptives: collaborative reanalysis of individual data on 53 297 women with breast cancer and 100 239 women without breast cancer from 54 epidemiological studies. Lancet 1996;347:1713–27.
315. Gill JK, Press MF, Patel AV, Bernstein L. Oral contraceptive use and risk of breast carcinoma in situ (United States). Cancer Causes Control 2006;17:1155–62.
316. Kumle M, Weiderpass E, Braaten T, Persson I, Adami HO, Lund E. Use of oral contraceptives and breast cancer risk: the Norwegian-Swedish Women's Lifestyle and Health Cohort Study. Cancer Epidemiol Biomarkers Prev 2002;11:1375–81.
317. Marchbanks PA, McDonald JA, Wilson HG, et al. Oral contraceptives and the risk of breast cancer. N Engl J Med 2002;346:2025–32.
318. Newcomb PA, Longnecker MP, Storer BE, et al. Recent oral contraceptive use and risk of breast cancer (United States). Cancer Causes Control 1996;7:525–32.
319. Rosenberg L, Palmer JR, Rao RS, et al. Case-control study of oral contraceptive use and risk of breast cancer. Am J Epidemiol 1996;143:25–37.
320. Rosenberg L, Zhang Y, Coogan PF, Strom BL, Palmer JR. A case-control study of oral contraceptive use and incident breast cancer. Am J Epidemiol 2009;169:473–9.
321. Shapiro S, Rosenberg L, Hoffman M, et al. Risk of breast cancer in relation to the use of injectable progestogen contraceptives and combined estrogen/progestogen contraceptives. Am J Epidemiol 2000;151:396–403.

Appendix A

Summary Chart of *U.S. Medical Eligibility Criteria for Contraceptive Use, 2010*

Key:
1. No restriction (method can be used)
2. Advantages generally outweigh theoretical or proven risks
3. Theoretical or proven risks usually outweigh the advantages
4. Unacceptable health risk (method not to be used)

Updated June 2012. This summary sheet only contains a subset of the recommendations from the US MEC. For complete guidance, see: http://www.cdc.gov/reproductivehealth/unintendedpregnancy/USMEC.htm.

Most contraceptive methods do not protect against sexually transmitted infections (STIs). Consistent and correct use of the male latex condom reduces the risk of STIs and HIV.

Condition	Sub-condition	Combined pill, patch, ring		Progestin-only pill		Injection		Implant		LNG-IUD		Copper-IUD	
		I	C	I	C	I	C	I	C	I	C	I	C
Age		Menarche to <40=1		Menarche to <18=1		Menarche to <18=2		Menarche to <18=1		Menarche to <20=2		Menarche to <20=2	
		≥40=2		18-45=1		18-45=1		18-45=1		≥20=1		≥20=1	
				>45=1		>45=2		>45=1					
Anatomic abnormalities	a) Distorted uterine cavity									4		4	
	b) Other abnormalities									2		2	
Anemias	a) Thalassemia	1		1		1		1		1		2	
	b) Sickle cell disease†	2		1		1		1		1		2	
	c) Iron-deficiency anemia	1		1		1		1		1		2	
Benign ovarian tumors	(including cysts)	1		1		1		1		1		1	
Breast disease	a) Undiagnosed mass	2*		2*		2*		2*		2		1	
	b) Benign breast disease	1		1		1		1		1		1	
	c) Family history of cancer	1		1		1		1		1		1	
	d) Breast cancer†												
	i) current	4		4		4		4		4		1	
	ii) past and no evidence of current disease for 5 years	3		3		3		3		3		1	
Breastfeeding (see also Postpartum)	a) < 1 month postpartum	3*		2*		2*		2*					
	b) 1 month or more postpartum	2*		1*		1*		1*					
Cervical cancer	Awaiting treatment	2		1		2		2		4	2	4	2
Cervical ectropion		1		1		1		1		1		1	
Cervical intraepithelial neoplasia		2		1		2		2		2		1	

See table footnotes on page 54.

Appendix A (*Continued*)

Summary Chart of *U.S. Medical Eligibility Criteria for Contraceptive Use, 2010*

Condition	Sub-condition	Combined pill, patch, ring		Progestin-only pill		Injection		Implant		LNG-IUD		Copper-IUD	
		I	C	I	C	I	C	I	C	I	C	I	C
Cirrhosis	a) Mild (compensated)	1		1		1		1		1		1	
	b) Severe† (decompensated)	4		3		3		3		3		1	
DVT/PE	a) History of DVT/PE, not on anticoagulant therapy												
	i) higher risk for recurrent DVT/PE	4		2		2		2		2		1	
	ii) lower risk for recurrent DVT/PE	3		2		2		2		2		1	
	b) Acute DVT/PE	4		2		2		2		2		2	
	c) DVT/PE and established on anticoagulant therapy for at least 3 months												
	i) higher risk for recurrent DVT/PE	4*		2		2		2		2		2	
	ii) lower risk for recurrent DVT/PE	3*		2		2		2		2		2	
	d) Family history (first-degree relatives)	2		1		1		1		1		1	
	e) Major surgery												
	(i) with prolonged immobilization	4		2		2		2		2		1	
	(ii) without prolonged immobilization	2		1		1		1		1		1	
	f) Minor surgery without immobilization	1		1		1		1		1		1	
Depressive disorders		1*		1*		1*		1*		1*		1*	

See table footnotes on page 54.

Appendix A (*Continued*)

Summary Chart of *U.S. Medical Eligibility Criteria for Contraceptive Use, 2010*

Condition	Sub-condition	Combined pill, patch, ring		Progestin-only pill		Injection		Implant		LNG-IUD		Copper-IUD	
		I	C	I	C	I	C	I	C	I	C	I	C
Diabetes mellitus	a) History of gestational diabetes mellitus only	1		1		1		1		1		1	
	b) Non-vascular disease												
	(i) non-insulin dependent	2		2		2		2		2		1	
	(ii) insulin dependent†	2		2		2		2		2		1	
	c) Nephropathy/ retinopathy/ neuropathy†	3/4*		2		3		2		2		1	
	d) Other vascular disease or diabetes of >20 years' duration†	3/4*		2		3		2		2		1	
Endometrial cancer†		1		1		1		1		4	2	4	2
Endometrial hyperplasia		1		1		1		1		1		1	
Endometriosis		1		1		1		1		1		2	
Epilepsy†	(see also Drug Interactions)	1*		1*		1*		1*		1		1	
Gallbladder disease	a) Symptomatic												
	(i) treated by cholecystectomy	2		2		2		2		2		1	
	(ii) medically treated	3		2		2		2		2		1	
	(iii) current	3		2		2		2		2		1	
	b) Asymptomatic	2		2		2		2		2		1	
Gestational trophoblastic disease	a) Decreasing or undetectable ß-hCG levels	1		1		1		1		3		3	
	b) Persistently elevated ß-hCG levels or malignant disease†	1		1		1		1		4		4	
Headaches	a) Non-migrainous	1*	2*	1*	1*	1*	1*	1*	1*	1*	1*	1*	
	b) Migraine												
	i) without aura, age <35	2*	3*	1*	2*	2*	2*	2*	2*	2*	2*	1*	
	ii) without aura, age ≥35	3*	4*	1*	2*	2*	2*	2*	2*	2*	2*	1*	
	iii) with aura, any age	4*	4*	2*	3*	2*	3*	2*	3*	2*	3*	1*	

See table footnotes on page 54.

Appendix A (*Continued*)

Summary Chart of *U.S. Medical Eligibility Criteria for Contraceptive Use, 2010*

Condition	Sub-condition	Combined pill, patch, ring		Progestin-only pill		Injection		Implant		LNG-IUD		Copper-IUD	
		I	C	I	C	I	C	I	C	I	C	I	C
History of bariatric surgery†	a) Restrictive procedures	1		1		1		1		1		1	
	b) Malabsorptive procedures	COCs: 3 P/R: 1		3		1		1		1		1	
History of cholestasis	a) Pregnancy-related	2		1		1		1		1		1	
	b) Past COC-related	3		2		2		2		2		1	
History of high blood pressure during pregnancy		2		1		1		1		1		1	
History of pelvic surgery		1		1		1		1		1		1	
HIV	High risk	1		1		1*		1		2	2	2	2
	HIV infected (see also Drug Interactions)†	1*		1*		1*		1*		2	2	2	2
	AIDS (see also Drug Interactions) †	1*		1*		1*		1*		3	2*	3	2*
	Clinically well on therapy	If on treatment, see Drug Interactions								2	2	2	2
Hyperlipidemias		2/3*		2*		2*		2*		2*		1*	
Hypertension	a) Adequately controlled hypertension	3*		1*		2*		1*		1		1	
	b) Elevated blood pressure levels (properly taken measurements)												
	(i) systolic 140-159 or diastolic 90-99	3		1		2		1		1		1	
	(ii) systolic ≥160 or diastolic ≥100†	4		2		3		2		2		1	
	c) Vascular disease	4		2		3		2		2		1	
Inflammatory bowel disease	(Ulcerative colitis, Crohn's disease)	2/3*		2		2		1		1		1	
Ischemic heart disease†	Current and history of	4		2	3	3		2	3	2	3	1	
Liver tumors	a) Benign												
	i) Focal nodular hyperplasia	2		2		2		2		2		1	
	ii) Hepatocellular adenoma†	4		3		3		3		3		1	
	b) Malignant†	4		3		3		3		3		1	
Malaria		1		1		1		1		1		1	

See table footnotes on page 54.

Appendix A (*Continued*)

Summary Chart of *U.S. Medical Eligibility Criteria for Contraceptive Use, 2010*

Condition	Sub-condition	Combined pill, patch, ring		Progestin-only pill		Injection		Implant		LNG-IUD		Copper-IUD	
		I	C	I	C	I	C	I	C	I	C	I	C
Multiple risk factors for arterial cardiovascular disease	(such as older age, smoking, diabetes and hypertension)	3/4*		2*		3*		2*		2		1	
Obesity	a) ≥30 kg/m^2 BMI	2		1		1		1		1		1	
	b) Menarche to <18 years and ≥30 kg/m^2 BMI	2		1		2		1		1		1	
Ovarian cancer†		1		1		1		1		1		1	
Parity	a) Nulliparous	1		1		1		1		2		2	
	b) Parous	1		1		1		1		1		1	
Past ectopic pregnancy		1		2		1		1		1		1	
Pelvic inflammatory disease	a) Past, (assuming no current risk factors of STIs)												
	(i) with subsequent pregnancy	1		1		1		1		1	1	1	1
	(ii) without subsequent pregnancy	1		1		1		1		2	2	2	2
	b) Current	1		1		1		1		4	2*	4	2*
Peripartum cardiomyopathy†	a) Normal or mildly impaired cardiac function												
	(i) <6 months	4		1		1		1		2		2	
	(ii) ≥6 months	3		1		1		1		2		2	
	b) Moderately or severely impaired cardiac function	4		2		2		2		2		2	
Postabortion	a) First trimester	1*		1*		1*		1*		1*		1*	
	b) Second trimester	1*		1*		1*		1*		2		2	
	c) Immediately post-septic abortion	1*		1*		1*		1*		4		4	
Postpartum (see also Breastfeeding)	a) <21 days	4		1		1		1					
	b) 21 days to 42 days												
	(i) with other risk factors for VTE	3*		1		1		1					
	(ii) without other risk factors for VTE	2		1		1		1					
	c) >42 days	1		1		1		1					

See table footnotes on page 54.

Appendix A (*Continued*)

Summary Chart of *U.S. Medical Eligibility Criteria for Contraceptive Use, 2010*

Condition	Sub-condition	Combined pill, patch, ring		Progestin-only pill		Injection		Implant		LNG-IUD		Copper-IUD	
		I	C	I	C	I	C	I	C	I	C	I	C
Postpartum (in breastfeeding or non-breastfeeding women, including post-cesarean section)	a) <10 minutes after delivery of the placenta									2		1	
	b) 10 minutes after delivery of the placenta to < 4 weeks									2		2	
	c) ≥4 weeks									1		1	
	d) Puerperal sepsis									4		4	
Pregnancy		NA*		NA*		NA*		NA*		4*		4*	
Rheumatoid arthritis	a) On immunosuppressive therapy	2		1		2/3*		1		2	1	2	1
	b) Not on immunosuppressive therapy	2		1		2		1		1		1	
Schistosomiasis	a) Uncomplicated	1		1		1		1		1		1	
	b) Fibrosis of the liver†	1		1		1		1		1		1	
Severe dysmenorrhea		1		1		1		1		1		2	
STIs	a) Current purulent cervicitis or chlamydial infection or gonorrhea	1		1		1		1		4	2*	4	2*
	b) Other STIs (excluding HIV and hepatitis)	1		1		1		1		2	2	2	2
	c) Vaginitis (including trichomonas vaginalis and bacterial vaginosis)	1		1		1		1		2	2	2	2
	d) Increased risk of STIs	1		1		1		1		2/3*	2	2/3*	2
Smoking	a) Age <35	2		1		1		1		1		1	
	b) Age ≥35, <15 cigarettes/day	3		1		1		1		1		1	
	c) Age ≥35, ≥15 cigarettes/day	4		1		1		1		1		1	
Solid organ transplantation†	a) Complicated	4		2		2		2		3	2	3	2
	b) Uncomplicated	2*		2		2		2		2		2	
Stroke†	History of cerebrovascular accident	4		2	3	3		2	3	2		1	

See table footnotes on page 54.

Appendix A (*Continued*)

Summary Chart of *U.S. Medical Eligibility Criteria for Contraceptive Use, 2010*

Condition	Sub-condition	Combined pill, patch, ring		Progestin-only pill		Injection		Implant		LNG-IUD		Copper-IUD	
		I	C	I	C	I	C	I	C	I	C	I	C
Superficial venous thrombosis	a) Varicose veins	1		1		1		1		1		1	
	b) Superficial thrombophlebitis	2		1		1		1		1		1	
Systemic lupus erythematosus†	a) Positive (or unknown) antiphospholipid antibodies	4		3		3	3	3		3		1	1
	b) Severe thrombocytopenia	2		2		3	2	2		2*		3*	2*
	c) Immunosuppressive treatment	2		2		2	2	2		2		2	1
	d) None of the above	2		2		2	2	2		2		1	1
Thrombogenic mutations†		4*		2*		2*		2*		2*		1*	
Thyroid disorders	Simple goiter/ hyperthyroid/ hypothyroid	1		1		1		1		1		1	
Tuberculosis† (see also Drug Interactions)	a) Non-pelvic	1*		1*		1*		1*		1		1	
	b) Pelvic	1*		1*		1*		1*		4	3	4	3
Unexplained vaginal bleeding	(suspicious for serious condition) before evaluation	2*		2*		3*		3*		4*	2*	4*	2*
Uterine fibroids		1		1		1		1		2		2	
Valvular heart disease	a) Uncomplicated	2		1		1		1		1		1	
	b) Complicated†	4		1		1		1		1		1	
Vaginal bleeding patterns	a) Irregular pattern without heavy bleeding	1		2		2		2		1	1	1	
	b) Heavy or prolonged bleeding	1*		2*		2*		2*		1*	2*	2*	
Viral hepatitis	a) Acute or flare	3/4*	2	1		1		1		1		1	
	b) Carrier/Chronic	1	1	1		1		1		1		1	

See table footnotes on page 54.

Appendix A (*Continued*)

Summary Chart of *U.S. Medical Eligibility Criteria for Contraceptive Use, 2010*

Condition	Sub-condition	Combined pill, patch, ring		Progestin-only pill		Injection		Implant		LNG-IUD		Copper-IUD	
		I	C	I	C	I	C	I	C	I	C	I	C
Drug Interactions													
Antiretroviral therapy	a) Nucleoside reverse transcriptase inhibitors	**1***		**1**		**1**		**1**		**2/3***	**2***	**2/3***	**2***
	b) Non-nucleoside reverse transcriptase inhibitors	**2***		**2***		**1**		**2***		**2/3***	**2***	**2/3***	**2***
	c) Ritonavir-boosted protease inhibitors	**3***		**3***		**1**		**2***		**2/3***	**2***	**2/3***	**2***
Anticonvulsant therapy	a) Certain anticonvulsants (phenytoin, carbamazepine, barbiturates, primidone, topiramate, oxcarbazepine)	**3***		**3***		**1**		**2***		**1**		**1**	
	b) Lamotrigine	**3***		**1**		**1**		**1**		**1**		**1**	
Antimicrobial therapy	a) Broad spectrum antibiotics	**1**		**1**		**1**		**1**		**1**		**1**	
	b) Antifungals	**1**		**1**		**1**		**1**		**1**		**1**	
	c) Antiparasitics	**1**		**1**		**1**		**1**		**1**		**1**	
	d) Rifampicin or rifabutin therapy	**3***		**3***		**1**		**2***		**1**		**1**	

Abbreviations: AIDS = acquired immunodeficiency syndrome; BMI = body mass index; C = continuation of contraceptive method; COC = combined oral contraceptive; Cu-IUD = copper-containing intrauterine device; DVT = deep venous thrombosis; hCG = human chorionic gonadotropin; HIV = human immunodeficiency virus; I = initiation of contraceptive method; LNG-IUD = levonorgestrel-releasing intrauterine device; NA = not applicable; PE = pulmonary embolism; STI = sexually transmitted infection; VTE = venous thromboembolism.

Source: Modified from CDC. Summary chart of U.S. medical eligibility criteria for contraceptive use. Atlanta, GA: CDC; 2012. (Available at http://www.cdc.gov/reproductivehealth/UnintendedPregnancy/USMEC.htm.)

* Please see the complete guidance for a clarification to this classification: www.cdc.gov/reproductivehealth/unintendedpregnancy/USMEC.htm.

† Condition that exposes a woman to increased risk as a result of unintended pregnancy.

Appendix B

When To Start Using Specific Contraceptive Methods

Contraceptive method	When to start (if the provider is reasonably certain that the woman is not pregnant)	Additional contraception (i.e., back-up) needed	Examinations or tests needed before initiation*
Copper-containing IUD	Anytime	Not needed	Bimanual examination and cervical inspection†
Levonorgestrel-releasing IUD	Anytime	If >7 days after menses started, use back-up method or abstain for 7 days.	Bimanual examination and cervical inspection†
Implant	Anytime	If >5 days after menses started, use back-up method or abstain for 7 days.	None
Injectable	Anytime	If >7 days after menses started, use back-up method or abstain for 7 days.	None
Combined hormonal contraceptive	Anytime	If >5 days after menses started, use back-up method or abstain for 7 days.	Blood pressure measurement
Progestin-only pill	Anytime	If >5 days after menses started, use back-up method or abstain for 2 days.	None

Abbreviations: BMI = body mass index; HIV = human immunodeficiency virus; IUD = intrauterine device; STD = sexually transmitted disease; U.S. MEC = *U.S. Medical Eligibility Criteria for Contraceptive Use, 2010*.

* Weight (BMI) measurement is not needed to determine medical eligibility for any methods of contraception because all methods can be used (U.S. MEC 1) or generally can be used (U.S. MEC 2) among obese women (Box 2). However, measuring weight and calculating BMI (weight [kg]/height [m]^2) at baseline might be helpful for monitoring any changes and counseling women who might be concerned about weight change perceived to be associated with their contraceptive method.

† Most women do not require additional STD screening at the time of IUD insertion if they have already been screened according to CDC's *STD Treatment Guidelines* (available at http://www.cdc.gov/std/treatment). If a woman has not been screened according to guidelines, screening can be performed at the time of IUD insertion, and insertion should not be delayed. Women with purulent cervicitis or current chlamydial infection or gonorrhea should not undergo IUD insertion (U.S. MEC 4). Women who have a very high individual likelihood of STD exposure (e.g., those with a currently infected partner) generally should not undergo IUD insertion (U.S. MEC 3) (Box 2). For these women, IUD insertion should be delayed until appropriate testing and treatment occurs.

Appendix C

Examinations and Tests Needed Before Initiation of Contraceptive Methods

The examinations or tests noted apply to women who are presumed to be healthy. Those with known medical problems or other special conditions might need additional examinations or tests before being determined to be appropriate candidates for a particular method of contraception. The *U.S. Medical Eligibility Criteria for Contraceptive Use, 2010* (U.S. MEC), might be useful in such circumstances (*5*). The following classification was considered useful in differentiating the applicability of the various examinations or tests:

Class A: essential and mandatory in all circumstances for safe and effective use of the contraceptive method.

Class B: contributes substantially to safe and effective use, but implementation may be considered within the public health and/or service context; risk of not performing an examination or test should be balanced against the benefits of making the contraceptive method available.

Class C: does not contribute substantially to safe and effective use of the contraceptive method.

These classifications focus on the relationship of the examinations or tests to safe initiation of a contraceptive method. They are not intended to address the appropriateness of these examinations or tests in other circumstances. For example, some of the examinations or tests that are not deemed necessary for safe and effective contraceptive use might be appropriate for good preventive health care or for diagnosing or assessing suspected medical conditions.

No examinations or tests are needed before initiating condoms or spermicides. A bimanual examination is necessary for diaphragm fitting. A bimanual examination and cervical inspection are needed for cervical cap fitting.

TABLE. Examinations and tests needed before initiation of contraceptive methods

	Contraceptive method and class							
Examination or test	**Cu-IUD and LNG-IUD**	**Implant**	**Injectable**	**CHC**	**POP**	**Condom**	**Diaphragm or cervical cap**	**Spermicide**
Examination								
Blood pressure	C	C	C	A*	C	C	C	C
Weight (BMI) (weight [kg]/ height [m]^2)	—†	—†	—†	—†	—†	C	C	C
Clinical breast examination	C	C	C	C	C	C	C	C
Bimanual examination and cervical inspection	A	C	C	C	C	C	A§	C
Laboratory test								
Glucose	C	C	C	C	C	C	C	C
Lipids	C	C	C	C	C	C	C	C
Liver enzymes	C	C	C	C	C	C	C	C
Hemoglobin	C	C	C	C	C	C	C	C
Thrombogenic mutations	C	C	C	C	C	C	C	C
Cervical cytology (Papanicolaou smear)	C	C	C	C	C	C	C	C
STD screening with laboratory tests	—¶	C	C	C	C	C	C	C
HIV screening with laboratory tests	C	C	C	C	C	C	C	C

Abbreviations: BMI = body mass index; CHC = combined hormonal contraceptive; Cu-IUD = copper-containing intrauterine device; DMPA = depot medroxyprogesterone acetate; HIV = human immunodeficiency virus; LNG-IUD = levonorgestrel-releasing intrauterine device; POP = progestin-only pill; STD = sexually transmitted disease; U.S. MEC = *U.S. Medical Eligibility Criteria for Contraceptive Use, 2010*.

* In cases in which access to health care might be limited, the blood pressure measurement can be obtained by the woman in a nonclinical setting (e.g., pharmacy or fire station) and self-reported to the provider.

† Weight (BMI) measurement is not needed to determine medical eligibility for any methods of contraception because all methods can be used (U.S. MEC 1) or generally can be used (U.S. MEC 2) among obese women (Box 2). However, measuring weight and calculating BMI at baseline might be helpful for monitoring any changes and counseling women who might be concerned about weight change perceived to be associated with their contraceptive method.

§ A bimanual examination (not cervical inspection) is needed for diaphragm fitting.

¶ Most women do not require additional STD screening at the time of IUD insertion if they have already been screened according to CDC's *STD Treatment Guidelines* (available at http://www.cdc.gov/std/treatment). If a woman has not been screened according to guidelines, screening can be performed at the time of IUD insertion and insertion should not be delayed. Women with purulent cervicitis or current chlamydial infection or gonorrhea should not undergo IUD insertion (U.S. MEC 4). Women who have a very high individual likelihood of STD exposure (e.g., those with a currently infected partner) generally should not undergo IUD insertion (U.S. MEC 3). For these women, IUD insertion should be delayed until appropriate testing and treatment occurs.

Appendix D

Routine Follow-Up After Contraceptive Initiation

These recommendations address when routine follow-up is recommended for safe and effective continued use of contraception for healthy women. The recommendations refer to general situations and might vary for different users and different situations. Specific populations that might benefit from more frequent follow-up visits include adolescents, those with certain medical conditions or characteristics, and those with multiple medical conditions.

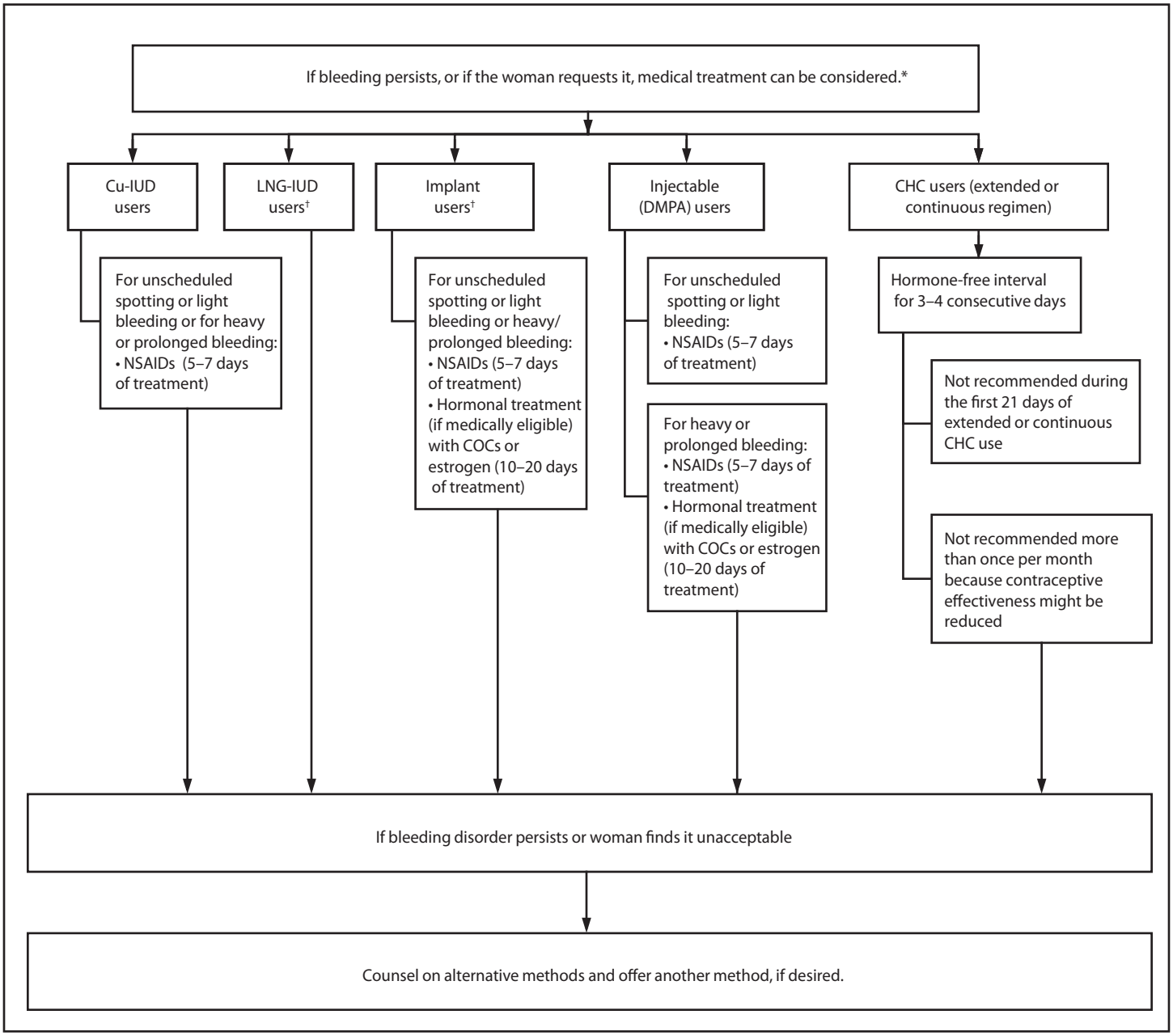
TABLE. Routine follow-up after contraceptive initiation

	Contraceptive method				
Action	**Cu-IUD or LNG-IUD**	**Implant**	**Injectable**	**CHC**	**POP**
General follow-up					
Advise women to return at any time to discuss side effects or other problems or if they want to change the method. Advise women using IUDs, implants, or injectables when the IUD or implant needs to be removed or when a reinjection is needed. No routine follow-up visit is required.	X	X	X	X	X
Other routine visits					
Assess the woman's satisfaction with her current method and whether she has any concerns about method use.	X	X	X	X	X
Assess any changes in health status, including medications, that would change the method's appropriateness for safe and effective continued use based on U.S. MEC (i.e., category 3 and 4 conditions and characteristics) (Box 2).	X	X	X	X	X
Consider performing an examination to check for the presence of IUD strings.	X	—	—	—	—
Consider assessing weight changes and counseling women who are concerned about weight change perceived to be associated with their contraceptive method.	X	X	X	X	X
Measure blood pressure.	—	—	—	X	—

Abbreviations: CHC = combined hormonal contraceptive; Cu-IUD = copper-containing intrauterine device; HIV = human immunodeficiency virus; IUD = intrauterine device; LNG-IUD = levonorgestrel-releasing intrauterine device; POP = progestin-only pill; U.S. MEC = *U.S. Medical Eligibility Criteria for Contraceptive Use, 2010*.

Appendix E

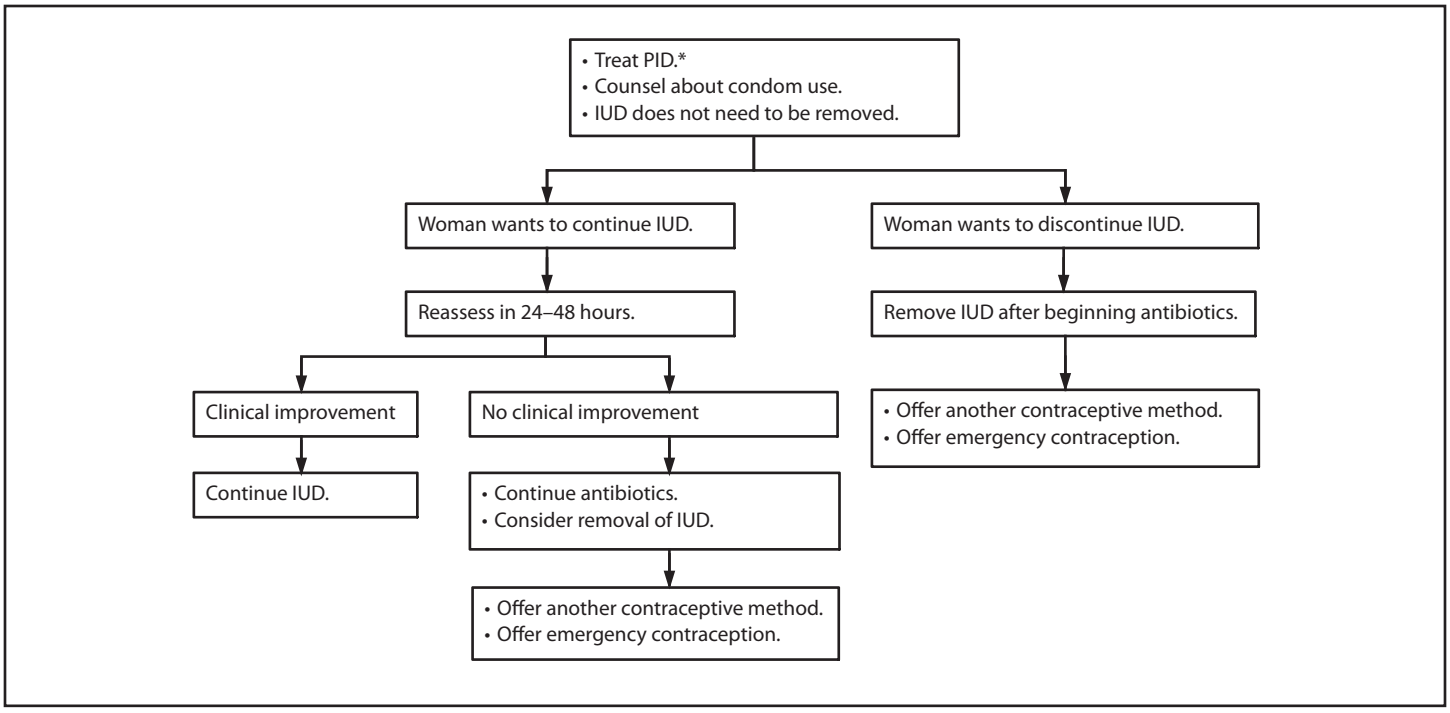
Management of Women with Bleeding Irregularities While Using Contraception



Abbreviations: CHC = combined hormonal contraceptive; COC = combined oral contraceptive; Cu-IUD = copper-containing intrauterine device; DMPA = depot medroxyprogesterone acetate; LNG-IUD = levonorgestrel-releasing intrauterine device; NSAIDs = nonsteroidal antiinflammatory drugs.
* If clinically warranted, evaluate for underlying condition. Treat the condition or refer for care.
† Heavy or prolonged bleeding, either unscheduled or menstrual, is uncommon.

Appendix F

Management of the IUD when a Cu-IUD or an LNG-IUD User Is Found To Have Pelvic Inflammatory Disease



Abbreviations: Cu-IUD = copper-containing IUD; IUD = intrauterine device; LNG-IUD = levonorgestrel-releasing IUD; PID = pelvic inflammatory disease.
* Treat according to CDC's *STD Treatment Guidelines* (available at http://www.cdc.gov/std/treatment).

U.S. Selected Practice Recommendations for Contraceptive Use Participants

CDC Steering Committee

Kathryn M. Curtis, PhD (Chair), Denise J. Jamieson, MD, Polly A. Marchbanks, PhD, Naomi K. Tepper, MD, CDC, Atlanta, Georgia.

Invited Meeting Participants, October 21–22, 2010, Atlanta, Georgia

Herbert B. Peterson, MD, University of North Carolina, Chapel Hill, North Carolina (Chair); Abbey Berenson, MD, University of Texas Medical Branch, Nassau Bay, Texas; Philip Darney, MD, University of California, San Francisco, California; David Eisenberg, MD, Washington University, St. Louis, Missouri; Paula Hillard, MD, Stanford University, Palo Alto, California; Andrew Kaunitz, MD, University of Florida, Jacksonville, Florida; Trent MacKay, MD, National Institute of Child Health and Human Development, National Institutes of Health, Bethesda, Maryland; Mary Mitchell, American College of Obstetricians and Gynecologists, Washington, DC; Michael Policar, MD, University of California, San Francisco, California; Sarah Prager, University of Washington, Seattle, Washington.

Systematic Review Presenters and Authors Meeting, October 4–7, 2011, Atlanta, Georgia

Kathryn M. Curtis, PhD, Suzanne G. Folger, PhD, Emily M. Godfrey, MD, Denise J. Jamieson, MD, Gary Jeng, PhD, Polly A. Marchbanks, PhD, Sarah Murtaza, MPH, Maria W. Steenland, MPH, Naomi K. Tepper, MD, Crystal P. Tyler, PhD, Maura K. Whiteman, PhD, Lauren B. Zapata, PhD, CDC, Atlanta, Georgia; Dalia Brahmi, MD, Ipas, Carrboro, NC; Nathalie Kapp, MD, Mary Lyn E. Gaffield, PhD, Maria I. Rodriguez, MD, World Health Organization, Geneva, Switzerland; Tara P. Cleary, MD, Carrie Cwiak, MD, Emory University, Atlanta, Georgia; Jennifer Salcedo, MD, University of California, Los Angeles, California; Ira Sharlip, MD, American Urological Association and University of California, San Francisco, California.

Invited Meeting Participants, October 4–7, 2011, Atlanta, Georgia

Willard Cates, Jr., MD, FHI360, Research Triangle Park, North Carolina (Chair); Herbert B. Peterson, MD, University of North Carolina, Chapel Hill, North Carolina (Chair); Rebecca Allen, MD, American Society for Reproductive Medicine and Brown University, Providence, Rhode Island; Abbey Berenson, MD, University of Texas Medical Branch, Nassau Bay, Texas; Paul Blumenthal, MD, Stanford University, Palo Alto, California; Mitchell Creinin, MD, University of California, Davis, California; Vanessa Cullins, MD, Planned Parenthood Federation of America, New York, New York; Jennifer Dietrich, MD, Baylor College of Medicine, Houston, Texas; Linda Dominguez, Southwest Women's Health, Albuquerque, New Mexico; Alison Edelman, MD, Oregon Health Sciences University, Portland, Oregon; David Eisenberg, MD, Washington University, St. Louis, Missouri; Melissa Gilliam, MD, The University of Chicago, Chicago, Illinois; Marji Gold, MD, Albert Einstein College of Medicine, Bronx, New York; Alisa Goldberg, MD, Brigham and Women's Hospital and Planned Parenthood of Massachusetts, Boston, Massachusetts; Robert Hatcher, MD, Emory University, Atlanta, Georgia; Mark Hathaway, MD, Association for Reproductive Health Professionals and Washington Hospital Center, Washington, DC; Stephen Heartwell, DrPH, Susan Thompson Buffett Foundation, Omaha, Nebraska; Paula Hillard, MD, Stanford University, Palo Alto, California; Andrew Kaunitz, MD, University of Florida, Jacksonville, Florida; Hal Lawrence, MD, American College of Obstetricians and Gynecologists, Washington, DC; Laura MacIsaac, MD, Albert Einstein School of Medicine, New York, New York; Trent MacKay, MD, National Institute of Child Health and Human Development, National Institutes of Health, Bethesda, Maryland; Mary Mitchell, American College of Obstetricians and Gynecologists, Washington, DC; Susan Moskosky, MS, U.S. Department of Health and Human Services, Rockville, Maryland; Patricia Murphy, DrPH, University of Utah, Salt Lake City, Utah; Kavita Nanda, MD, FHI360, Research Triangle Park, North Carolina; Deborah Nucatola, MD, Planned Parenthood Federation of America, New York, New York; Jeffrey Peipert, MD, Washington University, St. Louis, Missouri; Michael Policar, MD, University of California, San Francisco, California; Sarah Prager, University of Washington, Seattle, Washington; Ira Sharlip, MD, American Urological Association and University of California, San Francisco, California; David Soper, MD, University of South Carolina, Charleston, South Carolina; Lisa Soule, MD, Food and Drug Administration, Silver Spring, Maryland; Maria Trent, MD, Johns Hopkins University, Baltimore, Maryland; James Trussell, PhD, Princeton University, Princeton, New Jersey; Carolyn Westhoff, MD, Columbia University, New York, New York; Mimi Zieman, MD, Emory University, Atlanta, Georgia.

External Reviewers

Courtney Benedict, MSN, Planned Parenthood Federation of America, Sonoma, California; Gale Burstein, MD, Erie County Department of Health, Buffalo, New York; Lucy Chie, MD, Harvard Medical School, Boston, Massachusetts; Kelly Culwell, MD, University of California, Davis, California; Sharon Schnare, MSN, University of Washington, Seattle, Washington.

The *Morbidity and Mortality Weekly Report (MMWR)* Series is prepared by the Centers for Disease Control and Prevention (CDC) and is available free of charge in electronic format. To receive an electronic copy each week, visit MMWR's free subscription page at *http://www.cdc.gov/mmwr/mmwrsubscribe.html*. Paper copy subscriptions are available through the Superintendent of Documents, U.S. Government Printing Office, Washington, DC 20402; telephone 202-512-1800.

Address all inquiries about the *MMWR* Series, including material to be considered for publication, to Editor, *MMWR* Series, Mailstop E-90, CDC, 1600 Clifton Rd., N.E., Atlanta, GA 30333 or to *mmwrq@cdc.gov.*

U.S. Government Printing Office: 2013-623-210/81600 Region IV ISSN: 1057-5987

Centers for Disease Control and Prevention
MMWR
Morbidity and Mortality Weekly Report
Recommendations and Reports / Vol. 65 / No. 4
July 29, 2016

U.S. Selected Practice Recommendations for Contraceptive Use, 2016

Continuing Education Examination available at http://www.cdc.gov/mmwr/cme/conted.html.

U.S. Department of Health and Human Services
Centers for Disease Control and Prevention

CONTENTS

CONTENTS (*Continued*)

Disclosure of Relationship

CDC, our planners, and our content experts wish to disclose they have no financial interest or other relationships with the manufacturers of commercial products, suppliers of commercial services, or commercial supporters. Planners have reviewed content to ensure there is no bias. This document will not include any discussion of the unlabeled use of a product or a product under investigational use, with the exception that some of the recommendations in this document might be inconsistent with package labeling.

The *MMWR* series of publications is published by the Center for Surveillance, Epidemiology, and Laboratory Services, Centers for Disease Control and Prevention (CDC), U.S. Department of Health and Human Services, Atlanta, GA 30329-4027.

Suggested citation: [Author names; first three, then et al., if more than six.] [Title]. MMWR Recomm Rep 2016;65(No. RR-#):[inclusive page numbers].

U.S. Selected Practice Recommendations for Contraceptive Use, 2016

Kathryn M. Curtis, PhD[1]
Tara C. Jatlaoui, MD[1]
Naomi K. Tepper, MD[1]
Lauren B. Zapata, PhD[1]
Leah G. Horton, MSPH[1]
Denise J. Jamieson, MD[1]
Maura K. Whiteman, PhD[1]
[1]*Division of Reproductive Health, National Center for Chronic Disease Prevention and Health Promotion*

Summary

The 2016 U.S. Selected Practice Recommendations for Contraceptive Use *(U.S. SPR) addresses a select group of common, yet sometimes controversial or complex, issues regarding initiation and use of specific contraceptive methods. These recommendations for health care providers were updated by CDC after review of the scientific evidence and consultation with national experts who met in Atlanta, Georgia, during August 26–28, 2015. The information in this report updates the 2013 U.S. SPR* (CDC. U.S. selected practice recommendations for contraceptive use, 2013. MMWR 2013;62[No. RR-5]). *Major updates include 1) revised recommendations for starting regular contraception after the use of emergency contraceptive pills and 2) new recommendations for the use of medications to ease insertion of intrauterine devices. The recommendations in this report are intended to serve as a source of clinical guidance for health care providers and provide evidence-based guidance to reduce medical barriers to contraception access and use. Health care providers should always consider the individual clinical circumstances of each person seeking family planning services. This report is not intended to be a substitute for professional medical advice for individual patients. Persons should seek advice from their health care providers when considering family planning options.*

Introduction

Unintended pregnancy rates remain high in the United States; approximately 45% of all pregnancies are unintended, with higher proportions among adolescent and young women, women who are racial/ethnic minorities, and women with lower levels of education and income (*1*). Unintended pregnancies increase the risk for poor maternal and infant outcomes (*2*) and in 2010, resulted in U.S. government health care expenditures of $21 billion (*3*). Approximately half of unintended pregnancies are among women who were not using contraception at the time they became pregnant; the other half are among women who became pregnant despite reported use of contraception (*4*). Strategies to prevent unintended pregnancy include assisting women at risk for unintended pregnancy and their partners with choosing appropriate contraceptive methods and helping them use methods correctly and consistently to prevent pregnancy.

In 2013, CDC published the first *U.S. Selected Practice Recommendations for Contraceptive Use* (U.S. SPR), adapted from global guidance developed by the World Health Organization (WHO SPR), which provided evidence-based guidance on how to use contraceptive methods safely and effectively once they are deemed to be medically appropriate.

Corresponding author: Kathryn M. Curtis, PhD, Division of Reproductive Health, National Center for Chronic Disease Prevention and Health Promotion, CDC. Telephone: 770-488-5200; E-mail: kmc6@cdc.gov.

U.S. SPR is a companion document to *U.S. Medical Eligibility Criteria for Contraceptive Use* (U.S. MEC) (http://www.cdc.gov/reproductivehealth/unintendedpregnancy/usmec.htm), which provides recommendations on safe use of contraceptive methods for women with various medical conditions and other characteristics (*5*). WHO intended for the global guidance to be used by local or national policy makers, family planning program managers, and the scientific community as a reference when they develop family planning guidance at the country or program level. During 2012–2013, CDC went through a formal process to adapt the global guidance for best implementation in the United States, which included rigorous identification and critical appraisal of the scientific evidence through systematic reviews, and input from national experts on how to translate that evidence into recommendations for U.S. health care providers (*6*). At that time, CDC committed to keeping this guidance up to date and based on the best available evidence, with full review every few years (*6*).

This document updates the 2013 U.S. SPR (*6*) with new evidence and input from experts. Major updates include 1) revised recommendations for starting regular contraception after the use of emergency contraceptive pills and 2) new recommendations for the use of medications to ease insertion of intrauterine devices (IUDs). Recommendations are provided for health care providers on the safe and effective use of contraceptive methods and address provision of contraceptive methods and management of side effects and other problems

with contraceptive method use, within the framework of removing unnecessary medical barriers to accessing and using contraception. These recommendations are meant to serve as a source of clinical guidance for health care providers; health care providers should always consider the individual clinical circumstances of each person seeking family planning services. This report is not intended to be a substitute for professional medical advice for individual patients, who should seek advice from their health care providers when considering family planning options.

Summary of Changes from the 2013 U.S. SPR

Updated Recommendations

Recommendations have been updated regarding when to start regular contraception after ulipristal acetate (UPA) emergency contraceptive pills:

- Advise the woman to start or resume hormonal contraception no sooner than 5 days after use of UPA, and provide or prescribe the regular contraceptive method as needed. For methods requiring a visit to a health care provider, such as depo-medroxyprogesterone acetate (DMPA), implants, and IUDs, starting the method at the time of UPA use may be considered; the risk that the regular contraceptive method might decrease the effectiveness of UPA must be weighed against the risk of not starting a regular hormonal contraceptive method.
- The woman needs to abstain from sexual intercourse or use barrier contraception for the next 7 days after starting or resuming regular contraception or until her next menses, whichever comes first.
- Any nonhormonal contraceptive method can be started immediately after the use of UPA.
- Advise the woman to have a pregnancy test if she does not have a withdrawal bleed within 3 weeks.

New Recommendations

New recommendations have been made for medications to ease IUD insertion:

- Misoprostol is not recommended for routine use before IUD insertion. Misoprostol might be helpful in select circumstances (e.g., in women with a recent failed insertion).
- Paracervical block with lidocaine might reduce patient pain during IUD insertion.

Methods

Since publication of the 2013 U.S. SPR, CDC has monitored the literature for new evidence relevant to the recommendations through the WHO/CDC continuous identification of research evidence (CIRE) system (*7*). This system identifies new evidence as it is published and allows WHO and CDC to update systematic reviews and facilitate updates to recommendations as new evidence warrants. Automated searches are run in PubMed weekly, and the results are reviewed. Abstracts that meet specific criteria are added to the web-based CIRE system, which facilitates coordination and peer review of systematic reviews for both WHO and CDC. In 2014, CDC reviewed all of the existing recommendations in the 2013 U.S. SPR for new evidence identified by CIRE that had the potential to lead to a changed recommendation. During August 27–28, 2014, CDC held a meeting in Atlanta, Georgia, of 11 family planning experts and representatives from partner organizations to solicit their input on the scope of and process for updating both the 2010 U.S. MEC and the 2013 U.S. SPR. The participants were experts in family planning and represented different provider types and organizations that represent health care providers. A list of participants is provided at the end of this report. The meeting related to topics to be addressed in the update of U.S. SPR based on new scientific evidence published since 2013 (identified though the CIRE system), topics addressed at a 2014 WHO meeting to update global guidance, and suggestions CDC received from providers for the addition of recommendations not included in the 2013 U.S. SPR (e.g., from provider feedback through e-mail, public inquiry, and questions received at conferences). CDC identified one topic to consider adding to the guidance: the use of medications to ease IUD insertion (evidence question: "Among women of reproductive age, does use of medications before IUD insertion improve the safety or effectiveness of the procedure [ease of insertion, need for adjunctive insertion measures, or insertion success] or affect patient outcomes [pain or side effects] compared with nonuse of these medications?"). CDC also identified one topic for which new evidence warranted a review of an existing recommendation: initiation of regular contraception after emergency contraceptive pills (evidence question: "Does ulipristal acetate for emergency contraception interact with regular use of hormonal contraception leading to decreased effectiveness of either contraceptive method?"). CDC determined that all other recommendations in the 2013 U.S. SPR were up to date and consistent with the current body of evidence for that recommendation.

In preparation for a subsequent expert meeting August 26–28, 2015, to review the scientific evidence

for potential recommendations, CDC staff conducted independent systematic reviews for each of the topics being considered. The purpose of these systematic reviews was to identify direct evidence related to the common clinical challenges associated with the recommendations. Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines were followed for reporting systematic reviews (*8*,*9*), and strength and quality of the evidence were assigned using the system of the U.S. Preventive Services Task Force (*10*). When direct evidence was limited or not available, indirect evidence (e.g., evidence on surrogate outcomes) and theoretical issues were considered and either added to direct evidence within a systematic review or separately compiled for presentation to the meeting participants. Completed systematic reviews were peer reviewed by two or three experts and then provided to participants before the expert meeting. Reviews are referenced throughout this document; the full reviews have been published and contain the details of each review, including systematic review question, literature search protocol, inclusion and exclusion criteria, evidence tables, and quality assessment. CDC staff continued to monitor new evidence identified through the CIRE system during the preparation for the August 2015 meeting.

During August 26–28, 2015, CDC held a meeting in Atlanta, Georgia, of 29 participants who were invited to provide their individual perspectives on the scientific evidence presented and to discuss potential recommendations that followed. Participants represented a wide range of expertise in family planning provision and research and included obstetrician/gynecologists, pediatricians, family physicians, nurse practitioners, epidemiologists, and others with research and clinical practice expertise in contraceptive safety, effectiveness, and management. Lists of participants and any potential conflicts of interest are provided at the end of this report. During the meeting, the evidence from the systematic review for each topic was presented, including direct evidence and any indirect evidence or theoretical concerns. Participants provided their perspectives on using the evidence to develop the recommendations that would meet the needs of U.S. health care providers. After the meeting, CDC determined the recommendations in this report, taking into consideration the perspectives provided by the meeting participants. Feedback also was received from four external reviewers, composed of health care providers and researchers who had not participated in the update meetings. These providers were asked to provide comments on the accuracy, feasibility, and clarity of the recommendations. Areas of research that need additional investigation also were considered during the meeting (*11*).

Maintaining Updated Guidance

As with any evidence-based guidance document, a key challenge is keeping the recommendations up to date as new scientific evidence becomes available. Working with WHO, CDC uses the CIRE system to ensure that WHO and CDC guidance is based on the best available evidence and that a mechanism is in place to update guidance when new evidence becomes available (*7*). CDC will continue to work with WHO to identify and assess all new relevant evidence and determine whether changes in the recommendations are warranted. In most cases, U.S. SPR will follow any updates in the WHO guidance, which typically occurs every 5 years (or sooner if warranted by new data). In addition, CDC will review any interim WHO updates for their application in the United States. CDC also will identify and assess any new literature for the recommendations that are not included in the WHO guidance and will completely review U.S. SPR every 5 years. Updates to the guidance can be found on the U.S. SPR website (http://www.cdc.gov/reproductivehealth/UnintendedPregnancy/USSPR.htm).

How To Use This Document

The recommendations in this report are intended to help health care providers address issues related to use of contraceptives, such as how to help a woman initiate use of a contraceptive method, which examinations and tests are needed before initiating use of a contraceptive method, what regular follow-up is needed, and how to address problems that often arise during use, including missed pills and side effects such as unscheduled bleeding. Each recommendation addresses what a woman or health care provider can do in specific situations. For situations in which certain groups of women might be medically ineligible to follow the recommendations, comments and reference to U.S. MEC are provided (*5*). The full U.S. MEC recommendations and the evidence supporting those recommendations have been updated in 2016 (*5*) and are summarized (Appendix A).

The information in this document is organized by contraceptive method, and the methods generally are presented in order of effectiveness, from highest to lowest. However, the recommendations are not intended to provide guidance on every aspect of provision and management of contraceptive method use. Instead, they incorporate the best available evidence to address specific issues regarding common, yet sometimes complex, clinical issues. Each contraceptive method section generally includes information about initiation of the method, regular follow-up, and management of problems with use (e.g., usage errors and side effects). Each section first

provides the recommendation and then includes comments and a brief summary of the scientific evidence on which the recommendation is based. The level of evidence from the systematic reviews for each evidence summary are provided based on the U.S. Preventive Services Task Force system, which includes ratings for study design (I: randomized controlled trials; II-1: controlled trials without randomization; II-2: observational studies; and II-3: multiple time series or descriptive studies), ratings for internal validity (good, fair, or poor), and categorization of the evidence as direct or indirect for the specific review question (*10*).

Recommendations in this document are provided for permanent methods of contraception, such as vasectomy and female sterilization, as well as for reversible methods of contraception, including the copper-containing intrauterine device (Cu-IUD); levonorgestrel-releasing IUDs (LNG-IUDs); the etonogestrel implant; progestin-only injectables; progestin-only pills (POPs); combined hormonal contraceptive methods that contain both estrogen and a progestin, including combined oral contraceptives (COCs), a transdermal contraceptive patch, and a vaginal contraceptive ring; and the standard days method (SDM). Recommendations also are provided for emergency use of the Cu-IUD and emergency contraceptive pills (ECPs).

For each contraceptive method, recommendations are provided on the timing for initiation of the method and indications for when and for how long additional contraception, or a back-up method, is needed. Many of these recommendations include guidance that a woman can start a contraceptive method at any time during her menstrual cycle if it is reasonably certain that she is not pregnant. Guidance for health care providers on how to be reasonably certain that a woman is not pregnant also is provided.

For each contraceptive method, recommendations include the examinations and tests needed before initiation of the method. These recommendations apply to persons who are presumed to be healthy. Those with known medical problems or other special conditions might need additional examinations or tests before being determined to be appropriate candidates for a particular method of contraception. U.S. MEC might be useful in such circumstances (*5*). Most women need no or very few examinations or tests before initiating a contraceptive method although they might be needed to address other noncontraceptive health needs (*12*). Any additional screening needed for preventive health care can be performed at the time of contraception initiation, and initiation should not be delayed for test results. The following classification system was developed by WHO and adopted by CDC to categorize the applicability of the various examinations or tests before initiation of contraceptive methods (*13*):

Class A: These tests and examinations are essential and mandatory in all circumstances for safe and effective use of the contraceptive method.

Class B: These tests and examinations contribute substantially to safe and effective use, although implementation can be considered within the public health context, service context, or both. The risk for not performing an examination or test should be balanced against the benefits of making the contraceptive method available.

Class C: These tests and examinations do not contribute substantially to safe and effective use of the contraceptive method.

These classifications focus on the relation of the examinations or tests to safe initiation of a contraceptive method. They are not intended to address the appropriateness of these examinations or tests in other circumstances. For example, some of the examinations or tests that are not deemed necessary for safe and effective contraceptive use might be appropriate for good preventive health care or for diagnosing or assessing suspected medical conditions. Systematic reviews were conducted for several different types of examinations and tests to assess whether a screening test was associated with safe use of contraceptive methods. Because no single convention exists for screening panels for certain diseases, including diabetes, lipid disorders, and liver diseases, the search strategies included broad terms for the tests and diseases of interest.

Summary charts and clinical algorithms that summarize the guidance for the various contraceptive methods have been developed for many of the recommendations, including when to start using specific contraceptive methods (Appendix B), examinations and tests needed before initiating the various contraceptive methods (Appendix C), routine follow-up after initiating contraception (Appendix D), management of bleeding irregularities (Appendix E), and management of IUDs when users are found to have pelvic inflammatory disease (PID) (Appendix F). These summaries might be helpful to health care providers when managing family planning patients. Additional tools are available on the U.S. SPR website (http://www.cdc.gov/reproductivehealth/UnintendedPregnancy/USSPR.htm).

Contraceptive Method Choice

Many elements need to be considered individually by a woman, man, or couple when choosing the most appropriate contraceptive method. Some of these elements include safety, effectiveness, availability (including accessibility and affordability), and acceptability. Although most contraceptive methods are safe for use by most women, U.S. MEC provides recommendations on the safety of specific contraceptive methods for women with certain characteristics and medical conditions (*5*); a U.S. MEC summary (Appendix A) and the categories of medical eligibility criteria for contraceptive use (Box 1) are provided.

Voluntary informed choice of contraceptive methods is an essential guiding principle, and contraceptive counseling, where applicable, might be an important contributor to the successful use of contraceptive methods.

Contraceptive method effectiveness is critically important in minimizing the risk for unintended pregnancy, particularly among women for whom an unintended pregnancy would pose additional health risks. The effectiveness of contraceptive methods depends both on the inherent effectiveness of the method itself and on how consistently and correctly it is used (Figure 1). Both consistent and correct use can vary greatly with characteristics such as age, income, desire to prevent or delay pregnancy, and culture. Methods that depend on consistent and correct use by clients have a wide range of effectiveness between typical use (actual use, including incorrect or inconsistent use) and perfect use (correct and consistent use according to directions) (*14*). IUDs and implants are considered long-acting, reversible contraception (LARC); these methods are highly effective because they do not depend on regular compliance from the user. LARC methods are appropriate for most women, including adolescents and nulliparous women. All women should be counseled about the full range and effectiveness of contraceptive options for which they are medically eligible so that they can identify the optimal method.

In choosing a method of contraception, dual protection from the simultaneous risk for human immunodeficiency virus (HIV) and other sexually transmitted diseases (STDs) also should be considered. Although hormonal contraceptives and IUDs are highly effective at preventing pregnancy, they do not protect against STDs, including HIV. Consistent and correct use of the male latex condom reduces the risk for HIV infection and other STDs, including chlamydial infection, gonococcal infection, and trichomoniasis (*15*). Although evidence is limited, use of female condoms can provide protection from acquisition and transmission of STDs (*15*). All patients, regardless of contraceptive choice, should be counseled about the use of condoms and the risk for STDs, including HIV infection (*15*). Additional information about prevention and treatment of STDs is available from the CDC *Sexually Transmitted Diseases Treatment Guidelines* (http://www.cdc.gov/std/treatment) (*15*).

Women, men, and couples have increasing numbers of safe and effective choices for contraceptive methods, including LARC methods such as IUDs and implants, to reduce the risk for unintended pregnancy. However, with these expanded options comes the need for evidence-based guidance to help health care providers offer quality family planning care to their patients, including assistance in choosing the most appropriate contraceptive method for individual circumstances and using that method correctly, consistently, and continuously to maximize effectiveness. Removing unnecessary barriers can help patients access and successfully use contraceptive methods. Several medical barriers to initiating and continuing contraceptive methods might exist, such as unnecessary screening examinations and tests before starting the method (e.g., a pelvic examination before initiation of COCs), inability to receive the contraceptive on the same day as the visit (e.g., waiting for test results that might not be needed or waiting until the woman's next menstrual cycle to start use), and difficulty obtaining continued contraceptive supplies (e.g., restrictions on number of pill packs dispensed at one time). Removing unnecessary steps, such as providing prophylactic antibiotics at the time of IUD insertion or requiring unnecessary follow-up procedures, also can help patients access and successfully use contraception.

BOX 1. Categories of medical eligibility criteria for contraceptive use

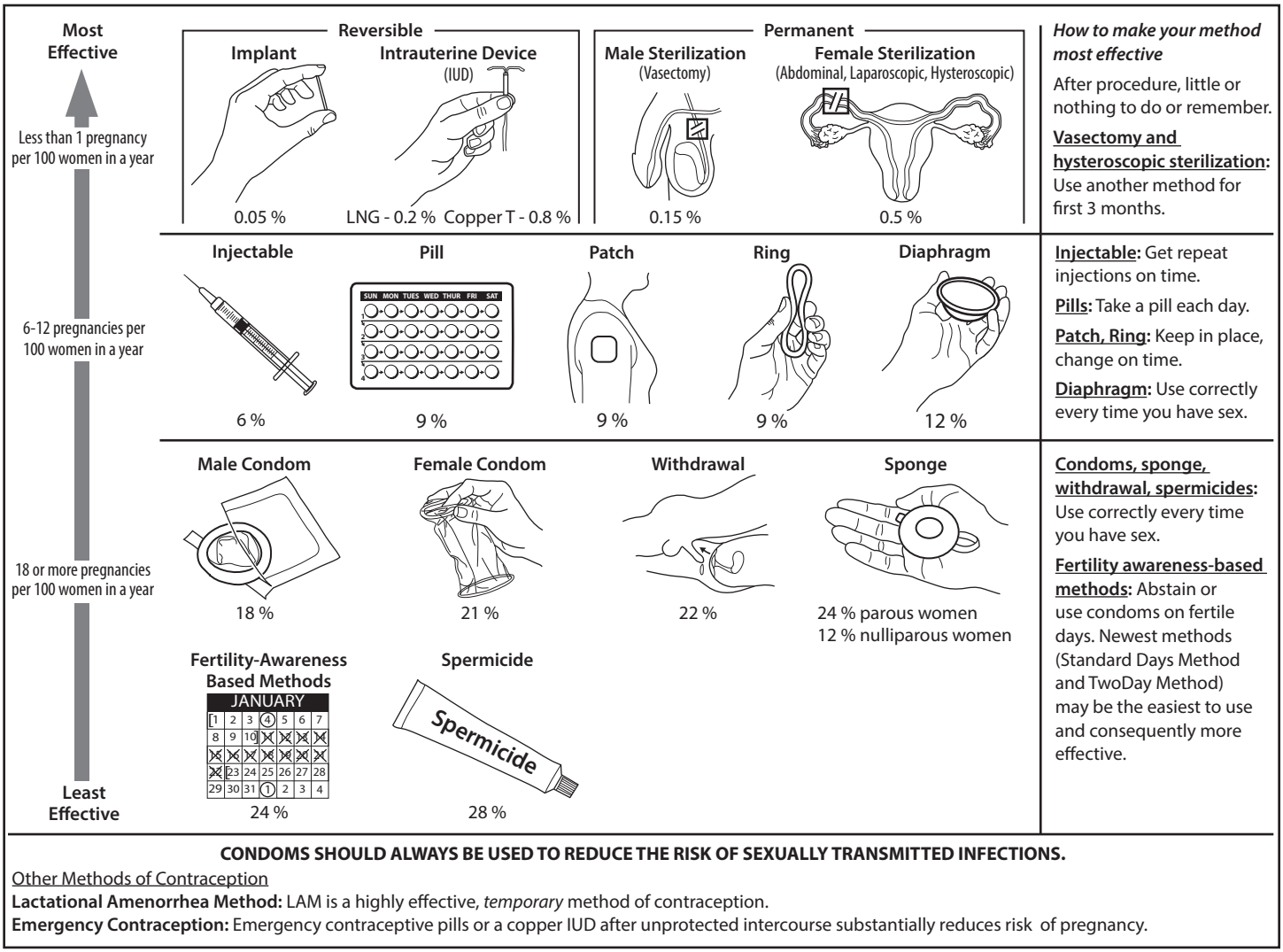
- U.S. MEC 1 = A condition for which there is no restriction for the use of the contraceptive method.
- U.S. MEC 2 = A condition for which the advantages of using the method generally outweigh the theoretical or proven risks.
- U.S. MEC 3 = A condition for which the theoretical or proven risks usually outweigh the advantages of using the method.
- U.S. MEC 4 = A condition that represents an unacceptable health risk if the contraceptive method is used.

Source: Curtis KM, Jatlaoui TC, Tepper NK, et al. U.S. medical eligibility criteria for contraceptive use. MMWR 2016;65(No. RR-3).
Abbreviation: U.S. MEC = *U.S. Medical Eligibility Criteria for Contraceptive Use.*

How To Be Reasonably Certain that a Woman Is Not Pregnant

In most cases, a detailed history provides the most accurate assessment of pregnancy risk in a woman who is about to start using a contraceptive method. Several criteria for assessing pregnancy risk are listed in the recommendation that follows. These criteria are highly accurate (i.e., a negative predictive value of 99%–100%) in ruling out pregnancy among women who are not pregnant (*16*–*19*). Therefore, CDC recommends that health care providers use these criteria to assess pregnancy status in a woman who is about to start using contraceptives (Box 2). If a woman meets one of these criteria (and therefore the health care provider can be reasonably certain that she is not pregnant), a urine pregnancy

FIGURE 1. Effectiveness of family planning methods*



Sources: Adapted from World Health Organization (WHO) Department of Reproductive Health and Research, Johns Hopkins Bloomberg School of Public Health/ Center for Communication Programs (CCP). Knowledge for health project. Family planning: a global handbook for providers (2011 update). Baltimore, MD; Geneva, Switzerland: CCP and WHO; 2011; and Trussell J. Contraceptive failure in the United States. Contraception 2011;83:397–404.

* The percentages indicate the number out of every 100 women who experienced an unintended pregnancy within the first year of typical use of each contraceptive method.

test might be considered in addition to these criteria (based on clinical judgment), bearing in mind the limitations of the accuracy of pregnancy testing. If a woman does not meet any of these criteria, then the health care provider cannot be reasonably certain that she is not pregnant, even with a negative pregnancy test. Routine pregnancy testing for every woman is not necessary.

On the basis of clinical judgment, health care providers might consider the addition of a urine pregnancy test; however, they should be aware of the limitations, including accuracy of the test relative to the time of last sexual intercourse, recent delivery, or spontaneous or induced abortion. Routine pregnancy testing for every woman is not necessary. If a woman has had recent (i.e., within the last 5 days) unprotected sexual intercourse, consider offering emergency contraception (either a Cu-IUD or ECPs) if pregnancy is not desired.

Comments and Evidence Summary. The criteria for determining whether a woman is pregnant depend on the assurance that she has not ovulated within a certain amount of time after her last menses, spontaneous or induced abortion, or delivery. Among menstruating women, the timing of ovulation can vary widely. During an average 28-day cycle, ovulation generally occurs during days 9–20 (*20*). In addition, the likelihood of ovulation is low from days 1–7 of the menstrual cycle (*21*). After a spontaneous or an induced abortion, ovulation can occur within 2–3 weeks and has been found to occur as early as 8–13 days after the end of the pregnancy. Therefore, the likelihood of ovulation is low ≤7 days after

BOX 2. How to be reasonably certain that a woman is not pregnant

A health care provider can be reasonably certain that a woman is not pregnant if she has no symptoms or signs of pregnancy and meets any one of the following criteria:

- is ≤7 days after the start of normal menses
- has not had sexual intercourse since the start of last normal menses.
- has been correctly and consistently using a reliable method of contraception
- is ≤7 days after spontaneous or induced abortion
- is within 4 weeks postpartum
- is fully or nearly fully breastfeeding (exclusively breastfeeding or the vast majority [≥85%] of feeds are breastfeeds), amenorrheic, and <6 months postpartum

an abortion (*22*–*24*). A systematic review reported that the mean day of first ovulation among postpartum nonlactating women occurred 45–94 days after delivery (*25*). In one study, the earliest ovulation was reported at 25 days after delivery. Among women who are within 6 months postpartum, are fully or nearly fully breastfeeding (exclusively breastfeeding or the vast majority [≥85%] of feeds are breastfeeds), and are amenorrheic, the risk for pregnancy is <2% (*26*,*27*).

Although pregnancy tests often are performed before initiating contraception, the accuracy of qualitative urine pregnancy tests varies depending on the timing of the test relative to missed menses, recent sexual intercourse, or recent pregnancy. The sensitivity of a pregnancy test is defined as the concentration of human chorionic gonadotropin (hCG) at which 95% of tests are positive. Most qualitative pregnancy tests approved by the U.S. Food and Drug Administration (FDA) report a sensitivity of 20–25 mIU/mL in urine (*28*–*31*). However, pregnancy detection rates can vary widely because of differences in test sensitivity and the timing of testing relative to missed menses (*30*,*32*). Some studies have shown that an additional 11 days past the day of expected menses are needed to detect 100% of pregnancies using qualitative tests (*29*). In addition, pregnancy tests cannot detect a pregnancy resulting from recent sexual intercourse. Qualitative tests also might have positive results for several weeks after termination of pregnancy because hCG can be present for several weeks after delivery or abortion (spontaneous or induced) (*33*–*35*).

For contraceptive methods other than IUDs, the benefits of starting to use a contraceptive method likely exceed any risk, even in situations in which the health care provider is uncertain whether the woman is pregnant. Therefore, the health care provider can consider having patients start using contraceptive methods other than IUDs at any time, with a follow-up pregnancy test in 2–4 weeks. The risks of not starting to use contraception should be weighed against the risks of initiating contraception use in a woman who might be already pregnant. Most studies have shown no increased risk for adverse outcomes, including congenital anomalies or neonatal or infant death, among infants exposed in utero to COCs (*36*–*38*). Studies also have shown no increased risk for neonatal or infant death or developmental abnormalities among infants exposed in utero to DMPA (*37*,*39*,*40*).

In contrast, for women who want to begin using an IUD (Cu-IUD or LNG-IUD), in situations in which the health care provider is uncertain whether the woman is pregnant, the woman should be provided with another contraceptive method to use until the health care provider is reasonably certain that she is not pregnant and can insert the IUD. Pregnancies among women with IUDs are at higher risk for complications such as spontaneous abortion, septic abortion, preterm delivery, and chorioamnionitis (*41*).

A systematic review identified four analyses of data from three diagnostic accuracy studies that evaluated the performance of the listed criteria (Box 2) through use of a pregnancy checklist compared with a urine pregnancy test conducted concurrently (*42*). The performance of the checklist to diagnose or exclude pregnancy varied, with sensitivity of 55%–100% and specificity of 39%–89%. The negative predictive value was consistent across studies at 99%–100%; the pregnancy checklist correctly ruled out women who were not pregnant. One of the studies assessed the added usefulness of signs and symptoms of pregnancy and found that these criteria did not substantially improve the performance of the pregnancy checklist, although the number of women with signs and symptoms was small (*16*) (Level of evidence: Diagnostic accuracy studies, fair, direct).

Intrauterine Contraception

Four IUDs are available in the United States, the copper-containing IUD and three levonorgestrel-releasing IUDs (containing a total of either 13.5 mg or 52 mg levonorgestrel). Fewer than 1 woman out of 100 becomes pregnant in the first year of using IUDs (with typical use) (*14*). IUDs are long-acting, are reversible, and can be used by women of all ages, including adolescents, and by parous and nulliparous women. IUDs do not protect against STDs; consistent and correct use of male latex condoms reduces the risk for STDs, including HIV.

Initiation of Cu-IUDs

Timing

- The Cu-IUD can be inserted at any time if it is reasonably certain that the woman is not pregnant (Box 2).
- The Cu-IUD also can be inserted within 5 days of the first act of unprotected sexual intercourse as an emergency contraceptive. If the day of ovulation can be estimated, the Cu-IUD also can be inserted >5 days after sexual intercourse as long as insertion does not occur >5 days after ovulation.

Need for Back-Up Contraception

- No additional contraceptive protection is needed after Cu-IUD insertion.

Special Considerations

Amenorrhea (Not Postpartum)

- **Timing**: The Cu-IUD can be inserted at any time if it is reasonably certain that the woman is not pregnant (Box 2).
- **Need for back-up contraception:** No additional contraceptive protection is needed.

Postpartum (Including After Cesarean Delivery)

- **Timing:** The Cu-IUD can be inserted at any time postpartum, including immediately postpartum (U.S. MEC 1 or 2) (Box 1), if it is reasonably certain that the woman is not pregnant (Box 2). The Cu-IUD should not be inserted in a woman with postpartum sepsis (e.g., chorioamnionitis or endometritis) (U.S. MEC 4).
- **Need for back-up contraception**: No additional contraceptive protection is needed.

Postabortion (Spontaneous or Induced)

- **Timing:** The Cu-IUD can be inserted within the first 7 days, including immediately postabortion (U.S. MEC 1 for first-trimester abortion and U.S. MEC 2 for second-trimester abortion). The Cu-IUD should not be inserted immediately after a septic abortion (U.S. MEC 4).
- **Need for back-up contraception:** No additional contraceptive protection is needed.

Switching from Another Contraceptive Method

- **Timing:** The Cu-IUD can be inserted immediately if it is reasonably certain that the woman is not pregnant (Box 2). Waiting for her next menstrual cycle is unnecessary.
- **Need for back-up contraception:** No additional contraceptive protection is needed.

Comments and Evidence Summary. In situations in which the health care provider is not reasonably certain that the woman is not pregnant, the woman should be provided with another contraceptive method to use until the health care provider can be reasonably certain that she is not pregnant and can insert the Cu-IUD.

A systematic review identified eight studies that suggested that timing of Cu-IUD insertion in relation to the menstrual cycle in non-postpartum women had little effect on long-term outcomes (rates of continuation, removal, expulsion, or pregnancy) or on short-term outcomes (pain at insertion, bleeding at insertion, or immediate expulsion) (*43*) (Level of evidence: II-2, fair, direct).

Initiation of LNG-IUDs

Timing of LNG-IUD Insertion

- The LNG-IUD can be inserted at any time if it is reasonably certain that the woman is not pregnant (Box 2).

Need for Back-Up Contraception

- If the LNG-IUD is inserted within the first 7 days since menstrual bleeding started, no additional contraceptive protection is needed.
- If the LNG-IUD is inserted >7 days since menstrual bleeding started, the woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.

Special Considerations

Amenorrhea (Not Postpartum)

- **Timing:** The LNG-IUD can be inserted at any time if it is reasonably certain that the woman is not pregnant (Box 2).
- **Need for back-up contraception:** The woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.

Postpartum (Including After Cesarean Delivery)

- **Timing:** The LNG-IUD can be inserted at any time, including immediately postpartum (U.S. MEC 1 or 2) if it is reasonably certain that the woman is not pregnant (Box 2). The LNG-IUD should not be inserted in a woman with postpartum sepsis (e.g., chorioamnionitis or endometritis) (U.S. MEC 4).
- **Need for back-up contraception:** If the woman is <6 months postpartum, amenorrheic, and fully or nearly fully breastfeeding (exclusively breastfeeding or the vast majority [≥85%] of feeds are breastfeeds) (*27*), no

additional contraceptive protection is needed. Otherwise, a woman who is ≥21 days postpartum and has not experienced return of her menstrual cycle needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days. If her menstrual cycles have returned and it has been >7 days since menstrual bleeding began, she needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.

Postabortion (Spontaneous or Induced)

- **Timing:** The LNG-IUD can be inserted within the first 7 days, including immediately postabortion (U.S. MEC 1 for first-trimester abortion and U.S. MEC 2 for second-trimester abortion). The LNG-IUD should not be inserted immediately after a septic abortion (U.S. MEC 4).
- **Need for back-up contraception:** The woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days unless the IUD is placed at the time of a surgical abortion.

Switching from Another Contraceptive Method

- **Timing:** The LNG-IUD can be inserted immediately if it is reasonably certain that the woman is not pregnant (Box 2). Waiting for her next menstrual cycle is unnecessary.
- **Need for back-up contraception:** If it has been >7 days since menstrual bleeding began, the woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.
- **Switching from a Cu-IUD:** If the woman has had sexual intercourse since the start of her current menstrual cycle and it has been >5 days since menstrual bleeding started, theoretically, residual sperm might be in the genital tract, which could lead to fertilization if ovulation occurs. A health care provider may consider providing any type of ECPs at the time of LNG-IUD insertion.

Comments and Evidence Summary. In situations in which the health care provider is uncertain whether the woman might be pregnant, the woman should be provided with another contraceptive method to use until the health care provider can be reasonably certain that she is not pregnant and can insert the LNG-IUD. If a woman needs to use additional contraceptive protection when switching to an LNG-IUD from another contraceptive method, consider continuing her previous method for 7 days after LNG-IUD insertion. No direct evidence was found regarding the effects of inserting LNG-IUDs on different days of the cycle on short- or long-term outcomes (*43*).

Examinations and Tests Needed Before Initiation of a Cu-IUD or an LNG-IUD

Among healthy women, few examinations or tests are needed before initiation of an IUD (Table 1). Bimanual examination and cervical inspection are necessary before IUD insertion. A baseline weight and BMI measurement might be useful for monitoring IUD users over time. If a woman has not been screened for STDs according to STD screening guidelines, screening can be performed at the time of insertion. Women with known medical problems or other special conditions might need additional examinations or tests before being determined to be appropriate candidates for a particular method of contraception. U.S. MEC might be useful in such circumstances (*5*).

Comments and Evidence Summary. Weight (BMI): Obese women can use IUDs (U.S. MEC 1) (*5*); therefore, screening for obesity is not necessary for the safe initiation of IUDs. However, measuring weight and calculating

TABLE 1. Classification of examinations and tests needed before IUD insertion

Examination or test	Class*	
	Copper-containing IUD	Levonorgestrel-releasing IUD
Examination		
Blood pressure	C	C
Weight (BMI) (weight [kg] / height [m]^2)	—†	—†
Clinical breast examination	C	C
Bimanual examination and cervical inspection	A	A
Laboratory test		
Glucose	C	C
Lipids	C	C
Liver enzymes	C	C
Hemoglobin	C	C
Thrombogenic mutations	C	C
Cervical cytology (Papanicolaou smear)	C	C
STD screening with laboratory tests	—§	—§
HIV screening with laboratory tests	C	C

Abbreviations: BMI = body mass index; HIV = human immunodeficiency virus; IUD = intrauterine device; STD = sexually transmitted disease; U.S. MEC = *U.S. Medical Eligibility Criteria for Contraceptive Use.*

* **Class A:** essential and mandatory in all circumstances for safe and effective use of the contraceptive method. **Class B:** contributes substantially to safe and effective use, but implementation may be considered within the public health and/or service context; the risk of not performing an examination or test should be balanced against the benefits of making the contraceptive method available. **Class C:** does not contribute substantially to safe and effective use of the contraceptive method.

† Weight (BMI) measurement is not needed to determine medical eligibility for any methods of contraception because all methods can be used (U.S. MEC 1) or generally can be used (U.S. MEC 2) among obese women (Box 1). However, measuring weight and calculating BMI at baseline might be helpful for monitoring any changes and counseling women who might be concerned about weight change perceived to be associated with their contraceptive method.

§ Most women do not require additional STD screening at the time of IUD insertion. If a woman with risk factors for STDs has not been screened for gonorrhea and chlamydia according to CDC's *STD Treatment Guidelines* (http://www.cdc.gov/std/treatment), screening can be performed at the time of IUD insertion, and insertion should not be delayed. Women with current purulent cervicitis or chlamydial infection or gonococcal infection should not undergo IUD insertion (U.S. MEC 4).

BMI (weight [kg] / height [m^2]) at baseline might be helpful for monitoring any changes and counseling women who might be concerned about weight change perceived to be associated with their contraceptive method.

Bimanual examination and cervical inspection: Bimanual examination and cervical inspection are necessary before IUD insertion to assess uterine size and position and to detect any cervical or uterine abnormalities that might indicate infection or otherwise prevent IUD insertion (*44*,*45*).

STDs: Women should be routinely screened for chlamydial infection and gonorrhea according to national screening guidelines. The CDC *Sexually Transmitted Diseases Treatment Guidelines* provide information on screening eligibility, timing, and frequency of screening and on screening for persons with risk factors (*15*) (http://www.cdc.gov/std/treatment). If STD screening guidelines have been followed, most women do not need additional STD screening at the time of IUD insertion, and insertion should not be delayed. If a woman with risk factors for STDs has not been screened for gonorrhea and chlamydia according to CDC STD treatment guidelines, screening can be performed at the time of IUD insertion, and insertion should not be delayed. Women with current purulent cervicitis or chlamydial infection or gonococcal infection should not undergo IUD insertion (U.S. MEC 4). A systematic review identified two studies that demonstrated no differences in PID rates among women who screened positive for gonorrhea or chlamydia and underwent concurrent IUD insertion compared with women who screened positive and initiated other contraceptive methods (*46*). Indirect evidence demonstrates women who undergo same-day STD screening and IUD insertion have similar PID rates compared with women who have delayed IUD insertion. Women who undergo same-day STD screening and IUD insertion have low incidence rates of PID. Algorithms for predicting PID among women with risk factors for STDs have poor predictive value. Risk for PID among women with risk factors for STDs is low (*15*,*47*–*57*). Although women with STDs at the time of IUD insertion have a higher risk for PID, the overall rate of PID among all IUD users is low (*51*,*54*).

Hemoglobin: Women with iron-deficiency anemia can use the LNG-IUD (U.S. MEC 1) (*5*); therefore, screening for anemia is not necessary for safe initiation of the LNG-IUD. Women with iron-deficiency anemia generally can use Cu-IUDs (U.S. MEC 2) (*5*). Measurement of hemoglobin before initiation of Cu-IUDs is not necessary because of the minimal change in hemoglobin among women with and without anemia using Cu-IUDs. A systematic review identified four studies that provided direct evidence for changes in hemoglobin among women with anemia who received Cu-IUDs (*58*). Evidence from one randomized trial (*59*) and one prospective cohort study (*60*) showed no significant changes in hemoglobin among Cu-IUD users with anemia, whereas two prospective cohort studies (*61*,*62*) showed a statistically significant decrease in hemoglobin levels during 12 months of follow-up; however, the magnitude of the decrease was small and most likely not clinically significant. The systematic review also identified 21 studies that provided indirect evidence by examining changes in hemoglobin among healthy women receiving Cu-IUDs (*63*–*83*), which generally showed no clinically significant changes in hemoglobin levels with up to 5 years of follow up (Level of evidence: I to II-2, fair, direct).

Lipids: Screening for dyslipidemias is not necessary for the safe initiation of Cu-IUD or LNG-IUD because of the low prevalence of undiagnosed disease in women of reproductive age and the low likelihood of clinically significant changes with use of hormonal contraceptives. A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened with lipid measurement before initiation of hormonal contraceptives (*57*). During 2009–2012 among women aged 20–44 years in the United States, 7.6% had high cholesterol, defined as total serum cholesterol ≥240 mg/dL (*84*). During 1999–2008, the prevalence of undiagnosed hypercholesterolemia among women aged 20–44 years was approximately 2% (*85*). Studies have shown mixed results about the effects of hormonal methods on lipid levels among both healthy women and women with baseline lipid abnormalities, and the clinical significance of these changes is unclear (*86*–*89*).

Liver enzymes: Women with liver disease can use the Cu-IUD (U.S. MEC 1) (*5*); therefore, screening for liver disease is not necessary for the safe initiation of the Cu-IUD. Although women with certain liver diseases generally should not use the LNG-IUD (U.S. MEC 3) (*5*), screening for liver disease before initiation of the LNG-IUD is not necessary because of the low prevalence of these conditions and the high likelihood that women with liver disease already would have had the condition diagnosed. A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened with liver enzyme tests before initiation of hormonal contraceptive use (*57*). In 2012, among U.S. women, the percentage with liver disease (not further specified) was 1.3% (*90*). In 2013, the incidence of acute hepatitis A, B, or C was ≤1 per 100,000 U.S. population (*91*). During 2002–2011, the incidence of liver carcinoma among U.S. women was approximately 3.7 per 100,000 population (*92*). Because estrogen and progestins are metabolized in the liver, the use of hormonal contraceptives among women with liver disease might, theoretically, be a concern. The use of hormonal contraceptives, specifically COCs and POPs,

does not affect disease progression or severity in women with hepatitis, cirrhosis, or benign focal nodular hyperplasia (*93*,*94*), although evidence is limited, and no evidence exists for the LNG-IUD.

Clinical breast examination: Women with breast disease can use the Cu-IUD (U.S. MEC 1) (*5*); therefore, screening for breast disease is not necessary for the safe initiation of the Cu-IUD. Although women with current breast cancer should not use the LNG-IUD (U.S. MEC 4) (*5*), screening asymptomatic women with a clinical breast examination before inserting an IUD is not necessary because of the low prevalence of breast cancer among women of reproductive age. A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened with a breast examination before initiation of hormonal contraceptives (*95*). The incidence of breast cancer among women of reproductive age in the United States is low. In 2012, the incidence of breast cancer among women aged 20–49 years was approximately 70.7 per 100,000 women (*96*).

Cervical cytology: Although women with cervical cancer should not undergo IUD insertion (U.S. MEC 4) (*5*), screening asymptomatic women with cervical cytology before IUD insertion is not necessary because of the high rates of cervical screening, low incidence of cervical cancer in the United States, and high likelihood that a woman with cervical cancer already would have had the condition diagnosed. A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened with cervical cytology before initiation of IUDs (*57*). Cervical cancer is rare in the United States, with an incidence rate of 9.8 per 100,000 women during 2012 (*96*). The incidence and mortality rates from cervical cancer have declined dramatically in the United States, largely because of cervical cytology screening (*97*). Overall screening rates for cervical cancer in the United States are high; in 2013 among women aged 18–44 years, approximately 77% reported having cervical cytology screening within the last 3 years (*98*).

HIV screening: Women with HIV infection can use (U.S. MEC 1) or generally can use (U.S. MEC 2) IUDs (*5*). Therefore, HIV screening is not necessary before IUD insertion. A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened for HIV infection before IUD insertion (*57*). Limited evidence suggests that IUDs are not associated with disease progression, increased infection, or other adverse health effects among women with HIV infection (*99*–*114*).

Other screening: Women with hypertension, diabetes, or thrombogenic mutations can use (U.S. MEC 1) or generally can use (U.S. MEC 2) IUDs (*5*). Therefore, screening for these conditions is not necessary for the safe initiation of IUDs.

Provision of Medications to Ease IUD Insertion

- Misoprostol is not recommended for routine use before IUD insertion. Misoprostol might be helpful in select circumstances (e.g., in women with a recent failed insertion).
- Paracervical block with lidocaine might reduce patient pain during IUD insertion.

Comments and Evidence Summary. Potential barriers to IUD use include anticipated pain with insertion and provider concerns about difficult insertion. Identifying effective approaches to ease IUD insertion might increase IUD initiation.

Evidence for misoprostol from two systematic reviews, including a total of 10 randomized controlled trials, suggests that misoprostol does not improve provider ease of insertion, reduce the need for adjunctive insertion measures, or improve insertion success (Level of evidence: I, good to fair, direct) and might increase patient pain and side effects (Level of evidence: I, high quality) (*115*,*116*). However, one randomized controlled trial examined women with a recent failed IUD insertion and found significantly higher insertion success with second insertion attempt among women pretreated with misoprostol versus placebo (Level of evidence: I, good, direct) (*117*).

Limited evidence for paracervical block with lidocaine from one systematic review suggests that it might reduce patient pain (*115*). In this review, two randomized controlled trials found significantly reduced pain at either tenaculum placement or IUD insertion among women receiving paracervical block with 1% lidocaine 3–5 minutes before IUD insertion (*118*,*119*). Neither trial found differences in side effects among women receiving paracervical block compared with controls (Level of evidence: I, moderate to low quality) (*118*,*119*).

Limited evidence on nonsteroidal antiinflammatory drugs (NSAIDs) and nitric oxide donors generally suggested no positive effect; evidence on lidocaine with administration other than paracervical block was limited and inconclusive (Level of evidence for provider ease of insertion: I, good to poor, direct; Level of evidence for need for adjunctive insertion measures: I, fair, direct; Level of evidence for patient pain: I, high to low quality; Level of evidence for side effects: I, high to low quality) (*115*,*116*).

Provision of Prophylactic Antibiotics at the Time of IUD Insertion

- Prophylactic antibiotics are generally not recommended for Cu-IUD or LNG-IUD insertion.

Comments and Evidence Summary. Theoretically, IUD insertion could induce bacterial spread and lead to

complications such as PID or infective endocarditis. A metaanalysis was conducted of randomized controlled trials examining antibiotic prophylaxis versus placebo or no treatment for IUD insertion (*120*). Use of prophylaxis reduced the frequency of unscheduled return visits but did not significantly reduce the incidence of PID or premature IUD discontinuation. Although the risk for PID was higher within the first 20 days after insertion, the incidence of PID was low among all women who had IUDs inserted (*51*). In addition, the American Heart Association recommends that the use of prophylactic antibiotics solely to prevent infective endocarditis is not needed for genitourinary procedures (*121*). Studies have not demonstrated a conclusive link between genitourinary procedures and infective endocarditis or a preventive benefit of prophylactic antibiotics during such procedures (*121*).

Routine Follow-Up After IUD Insertion

These recommendations address when routine follow-up is needed for safe and effective continued use of contraception for healthy women. The recommendations refer to general situations and might vary for different users and different situations. Specific populations who might benefit from more frequent follow-up visits include adolescents, persons with certain medical conditions or characteristics, and persons with multiple medical conditions.

- Advise the woman to return at any time to discuss side effects or other problems, if she wants to change the method being used, and when it is time to remove or replace the contraceptive method. No routine follow-up visit is required.
- At other routine visits, health care providers who see IUD users should do the following:
  - Assess the woman's satisfaction with her contraceptive method and whether she has any concerns about method use.
  - Assess any changes in health status, including medications, that would change the appropriateness of the IUD for safe and effective continued use on the basis of U.S. MEC (e.g., category 3 and 4 conditions and characteristics).
  - Consider performing an examination to check for the presence of the IUD strings.
  - Consider assessing weight changes and counseling women who are concerned about weight changes perceived to be associated with their contraceptive method.

Comments and Evidence Summary. Evidence from a systematic review about the effect of a specific follow-up visit schedule on IUD continuation is very limited and of poor quality. The evidence did not suggest that greater frequency of visits or earlier timing of the first follow-up visit after insertion improves continuation of use (*122*) (Level of evidence: II-2, poor, direct). Evidence from four studies from a systematic review on the incidence of PID among IUD initiators, or IUD removal as a result of PID, suggested that the incidence of PID did not differ between women using Cu- IUDs and those using DMPA, COCs, or LNG-IUDs (*123*) (Level of evidence: I to II-2, good, indirect). Evidence on the timing of PID after IUD insertion is mixed. Although the rate of PID generally was low, the largest study suggested that the rate of PID was significantly higher in the first 20 days after insertion (*51*) (Level of evidence: I to II-3, good to poor, indirect).

Bleeding Irregularities with Cu-IUD Use

- Before Cu-IUD insertion, provide counseling about potential changes in bleeding patterns during Cu-IUD use. Unscheduled spotting or light bleeding, as well as heavy or prolonged bleeding, is common during the first 3–6 months of Cu-IUD use, is generally not harmful, and decreases with continued Cu-IUD use.
- If clinically indicated, consider an underlying gynecological problem, such as Cu-IUD displacement, an STD, pregnancy, or new pathologic uterine conditions (e.g., polyps or fibroids), especially in women who have already been using the Cu-IUD for a few months or longer and who have developed a new onset of heavy or prolonged bleeding. If an underlying gynecological problem is found, treat the condition or refer for care.
- If an underlying gynecological problem is not found and the woman requests treatment, the following treatment option can be considered during days of bleeding:
  - NSAIDs for short-term treatment (5–7 days)
- If bleeding persists and the woman finds it unacceptable, counsel her on alternative contraceptive methods, and offer another method if it is desired.

Comments and Evidence Summary. During contraceptive counseling and before insertion of the Cu-IUD, information about common side effects such as unscheduled spotting or light bleeding or heavy or prolonged menstrual bleeding, especially during the first 3–6 months of use, should be discussed (*64*). These bleeding irregularities are generally not harmful. Enhanced counseling about expected bleeding patterns and reassurance that bleeding irregularities are generally not harmful has been shown to reduce method discontinuation in clinical trials with other contraceptives (i.e., DMPA) (*124*,*125*).

Evidence is limited on specific drugs, doses, and durations of use for effective treatments for bleeding irregularities with Cu-IUD use. Therefore, although this report includes general

recommendations for treatments to consider, evidence for specific regimens is lacking.

A systematic review identified 11 studies that examined various therapeutic treatments for heavy menstrual bleeding, prolonged menstrual bleeding, or both among women using Cu-IUDs (*126*). Nine studies examined the use of various oral NSAIDs for the treatment of heavy or prolonged menstrual bleeding among Cu-IUD users and compared them with either a placebo or a baseline cycle. Three of these trials examined the use of indomethacin (*127–129*), three examined mefenamic acid (*130–132*), and three examined flufenamic acid (*127*,*128*,*133*). Other NSAIDs used in the reported trials included alclofenac (*127*,*128*), suprofen (*134*), and diclofenac sodium (*135*). All but one NSAID study (*131*) demonstrated statistically significant or notable reductions in mean total menstrual blood loss with NSAID use. One study among 19 Cu-IUD users with heavy bleeding suggested that treatment with oral tranexamic acid can significantly reduce mean blood loss during treatment compared with placebo (*135*). Data regarding the overall safety of tranexamic acid are limited; an FDA warning states that tranexamic acid is contraindicated in women with active thromboembolic disease or with a history or intrinsic risk for thrombosis or thromboembolism (*136*,*137*). Treatment with aspirin demonstrated no statistically significant change in mean blood loss among women whose pretreatment menstrual blood loss was >80 ml or 60–80 mL; treatment resulted in a significant increase among women whose pretreatment menstrual blood loss was <60 mL (*138*). One study examined the use of a synthetic form of vasopressin, intranasal desmopressin (300 μg/day), for the first 5 days of menses for three treatment cycles and found a significant reduction in mean blood loss compared with baseline (*130*) (Level of evidence: I to II-3, poor to fair, direct). Only one small study examined treatment of spotting with three separate NSAIDs and did not observe improvements in spotting in any of the groups (*127*) (Level of evidence: I, poor, direct).

Bleeding Irregularities (Including Amenorrhea) with LNG-IUD Use

- Before LNG-IUD insertion, provide counseling about potential changes in bleeding patterns during LNG-IUD use. Unscheduled spotting or light bleeding is expected during the first 3–6 months of LNG-IUD use, is generally not harmful, and decreases with continued LNG-IUD use. Over time, bleeding generally decreases with LNG-IUD use, and many women experience only light menstrual bleeding or amenorrhea. Heavy or prolonged bleeding, either unscheduled or menstrual, is uncommon during LNG-IUD use.

Irregular Bleeding (Spotting, Light Bleeding, or Heavy or Prolonged Bleeding)

- If clinically indicated, consider an underlying gynecological problem, such as LNG-IUD displacement, an STD, pregnancy, or new pathologic uterine conditions (e.g., polyps or fibroids). If an underlying gynecological problem is found, treat the condition or refer for care.
- If bleeding persists and the woman finds it unacceptable, counsel her on alternative contraceptive methods, and offer another method if it is desired.

Amenorrhea

- Amenorrhea does not require any medical treatment. Provide reassurance.
  - If a woman's regular bleeding pattern changes abruptly to amenorrhea, consider ruling out pregnancy if clinically indicated.
- If amenorrhea persists and the woman finds it unacceptable, counsel her on alternative contraceptive methods, and offer another method if it is desired

Comments and Evidence Summary. During contraceptive counseling and before insertion of the LNG-IUD, information about common side effects such as unscheduled spotting or light bleeding, especially during the first 3–6 months of use, should be discussed. Approximately half of LNG-IUD users are likely to experience amenorrhea or oligomenorrhea by 2 years of use (*139*). These bleeding irregularities are generally not harmful. Enhanced counseling about expected bleeding patterns and reassurance that bleeding irregularities are generally not harmful has been shown to reduce method discontinuation in clinical trials with other hormonal contraceptives (i.e., DMPA) (*124*,*125*). No direct evidence was found regarding therapeutic treatments for bleeding irregularities during LNG-IUD use.

Management of the IUD when a Cu-IUD or an LNG-IUD User Is Found To Have PID

- Treat the PID according to the CDC *Sexually Transmitted Diseases Treatment Guidelines* (*15*).
- Provide comprehensive management for STDs, including counseling about condom use.
- The IUD does not need to be removed immediately if the woman needs ongoing contraception.
- Reassess the woman in 48–72 hours. If no clinical improvement occurs, continue antibiotics and consider removal of the IUD.
- If the woman wants to discontinue use, remove the IUD sometime after antibiotics have been started to avoid the

potential risk for bacterial spread resulting from the removal procedure.
- If the IUD is removed, consider ECPs if appropriate. Counsel the woman on alternative contraceptive methods, and offer another method if it is desired.
- A summary of IUD management in women with PID is provided (Appendix F).

Comments and Evidence Summary. Treatment outcomes do not generally differ between women with PID who retain the IUD and those who have the IUD removed; however, appropriate antibiotic treatment and close clinical follow-up are necessary.

A systematic review identified four studies that included women using copper or nonhormonal IUDs who developed PID and compared outcomes between women who had the IUD removed or did not (*140*). One randomized trial showed that women with IUDs removed had longer hospitalizations than those who did not, although no differences in PID recurrences or subsequent pregnancies were observed (*141*). Another randomized trial showed no differences in laboratory findings among women who removed the IUD compared with those who did not (*142*). One prospective cohort study showed no differences in clinical or laboratory findings during hospitalization; however, the IUD removal group had longer hospitalizations (*143*). One randomized trial showed that the rate of recovery for most clinical signs and symptoms was higher among women who had the IUD removed than among women who did not (*144*). No evidence was found regarding women using LNG-IUDs (Level of evidence: I to II-2, fair, direct.)

Management of the IUD when a Cu-IUD or an LNG-IUD User is Found To Be Pregnant

- Evaluate for possible ectopic pregnancy.
- Advise the woman that she has an increased risk for spontaneous abortion (including septic abortion that might be life threatening) and for preterm delivery if the IUD is left in place. The removal of the IUD reduces these risks but might not decrease the risk to the baseline level of a pregnancy without an IUD.
  - If she does not want to continue the pregnancy, counsel her about options.
  - If she wants to continue the pregnancy, advise her to seek care promptly if she has heavy bleeding, cramping, pain, abnormal vaginal discharge, or fever.

IUD Strings Are Visible or Can Be Retrieved Safely from the Cervical Canal

- Advise the woman that the IUD should be removed as soon as possible.
  - If the IUD is to be removed, remove it by pulling on the strings gently.
  - Advise the woman that she should return promptly if she has heavy bleeding, cramping, pain, abnormal vaginal discharge, or fever.
- If she chooses to keep the IUD, advise her to seek care promptly if she has heavy bleeding, cramping, pain, abnormal vaginal discharge, or fever.

IUD Strings Are Not Visible and Cannot Be Safely Retrieved

- If ultrasonography is available, consider performing or referring for ultrasound examination to determine the location of the IUD. If the IUD cannot be located, it might have been expelled or have perforated the uterine wall.
- If ultrasonography is not possible or the IUD is determined by ultrasound to be inside the uterus, advise the woman to seek care promptly if she has heavy bleeding, cramping, pain, abnormal vaginal discharge, or fever.

Comments and Evidence Summary. Removing the IUD improves the pregnancy outcome if the IUD strings are visible or the device can be retrieved safely from the cervical canal. Risks for spontaneous abortion, preterm delivery, and infection are substantial if the IUD is left in place.

Theoretically, the fetus might be affected by hormonal exposure from an LNG-IUD. However, whether this exposure increases the risk for fetal abnormalities is unknown.

A systematic review identified nine studies suggesting that women who did not remove their IUDs during pregnancy were at greater risk for adverse pregnancy outcomes (including spontaneous abortion, septic abortion, preterm delivery, and chorioamnionitis) compared with women who had their IUDs removed or who did not have an IUD (*41*). Cu-IUD removal decreased risks but not to the baseline risk for pregnancies without an IUD. One case series examined LNG-IUDs. When they were not removed, 8 out of 10 pregnancies ended in spontaneous abortions (Level of evidence: II-2, fair, direct).

Implants

The etonogestrel implant, a single rod with 68 mg of etonogestrel, is available in the United States. Fewer than 1 woman out of 100 become pregnant in the first year of use of the etonogestrel implant with typical use (*14*). The implant is long acting, is reversible, and can be used by women of all ages, including adolescents. The implant does not protect against STDs; consistent and correct use of male latex condoms reduces the risk for STDs, including HIV.

Initiation of Implants

Timing

- The implant can be inserted at any time if it is reasonably certain that the woman is not pregnant (Box 2).

Need for Back-Up Contraception

- If the implant is inserted within the first 5 days since menstrual bleeding started, no additional contraceptive protection is needed.
- If the implant is inserted >5 days since menstrual bleeding started, the woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.

Special Considerations

Amenorrhea (Not Postpartum)

- **Timing:** The implant can be inserted at any time if it is reasonably certain that the woman is not pregnant (Box 2).
- **Need for back-up contraception:** The woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.

Postpartum (Breastfeeding)

- **Timing:** The implant can be inserted at any time (U.S. MEC 2 if <1 month postpartum and U.S. MEC 1 if ≥1 month postpartum) if it is reasonably certain that the woman is not pregnant (Box 2).
- **Need for back-up contraception:** If the woman is <6 months postpartum, amenorrheic, and fully or nearly fully breastfeeding (exclusively breastfeeding or the vast majority [≥85%] of feeds are breastfeeds) (*27*), no additional contraceptive protection is needed. Otherwise, a woman who is ≥21 days postpartum and has not experienced return of her menstrual cycle needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days. If her menstrual cycles have returned and it has been >5 days since menstrual bleeding started, she needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.

Postpartum (Not Breastfeeding)

- **Timing:** The implant can be inserted at any time, including immediately postpartum (U.S. MEC 1) if it is reasonably certain that the woman is not pregnant (Box 2).
- **Need for back-up contraception:** If a woman is <21 days postpartum, no additional contraceptive protection is needed. A woman who is ≥21 days postpartum and has not experienced return of her menstrual cycle needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days. If her menstrual cycles have returned and it has been >5 days since menstrual bleeding started, she needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.

Postabortion (Spontaneous or Induced)

- **Timing:** The implant can be inserted within the first 7 days, including immediately after the abortion (U.S. MEC 1).
- **Need for back-up contraception:** The woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days unless the implant is placed at the time of a surgical abortion.

Switching from Another Contraceptive Method

- **Timing:** The implant can be inserted immediately if it is reasonably certain that the woman is not pregnant (Box 2). Waiting for her next menstrual cycle is unnecessary.
- **Need for back-up contraception:** If it has been >5 days since menstrual bleeding started, the woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days after insertion.
- **Switching from an IUD:** If the woman has had sexual intercourse since the start of her current menstrual cycle and it has been >5 days since menstrual bleeding started, theoretically, residual sperm might be in the genital tract, which could lead to fertilization if ovulation occurs. A health care provider may consider any of the following options:
  - Advise the woman to retain the IUD for at least 7 days after the implant is inserted and return for IUD removal.
  - Advise the woman to abstain from sexual intercourse or use barrier contraception for 7 days before removing the IUD and switching to the new method.
  - If the woman cannot return for IUD removal and has not abstained from sexual intercourse or used barrier contraception for 7 days, advise the woman to use ECPs (with the exception of UPA) at the time of IUD removal.

Comments and Evidence Summary. In situations in which the health care provider is uncertain whether the woman might be pregnant, the benefits of starting the implant likely exceed any risk; therefore, starting the implant should be considered at any time, with a follow-up pregnancy test in 2–4 weeks.

If a woman needs to use additional contraceptive protection when switching to an implant from another contraceptive method, consider continuing her previous method for 7 days after implant insertion. No direct evidence was found regarding the effects of starting the etonogestrel implant at different times of the cycle.

Examinations and Tests Needed Before Implant Insertion

Among healthy women, no examinations or tests are needed before initiation of an implant, although a baseline weight and BMI measurement might be useful for monitoring implant users over time (Table 2). Women with known medical problems or other special conditions might need additional examinations or tests before being determined to be appropriate candidates for a particular method of contraception. U.S. MEC might be useful in such circumstances (*5*).

Comments and Evidence Summary. Weight (BMI): Obese women can use implants (U.S. MEC 1) (*5*); therefore, screening for obesity is not necessary for the safe initiation of implants. However, measuring weight and calculating BMI at baseline might be helpful for monitoring any changes and counseling women who might be concerned about weight change perceived to be associated with their contraceptive method.

Bimanual examination and cervical inspection: A pelvic examination is not necessary before initiation of implants because it would not facilitate detection of conditions for which implant use would be unsafe. Women with current breast cancer should not use implants (U.S. MEC 4); women with certain liver diseases generally should not (U.S. MEC 3) use implants (*5*). However, none of these conditions are likely to be detected by pelvic examination (*145*). A systematic review identified two case-control studies that compared delayed and immediate pelvic examination before initiation of hormonal contraceptives, specifically oral contraceptives or DMPA (*95*). No differences in risk factors for cervical neoplasia, incidence of STDs, incidence of abnormal Papanicolaou smears, or incidence of abnormal wet mounts were observed. No evidence was found regarding implants (Level of evidence: II-2 fair, direct).

TABLE 2. Classification of examinations and tests needed before implant insertion

Examination or test	Class*
Examination	
Blood pressure	C
Weight (BMI) (weight [kg] / height [m]^2)	—†
Clinical breast examination	C
Bimanual examination and cervical inspection	C
Laboratory test	
Glucose	C
Lipids	C
Liver enzymes	C
Hemoglobin	C
Thrombogenic mutations	C
Cervical cytology (Papanicolaou smear)	C
STD screening with laboratory tests	C
HIV screening with laboratory tests	C

Abbreviations: BMI = body mass index; HIV = human immunodeficiency virus; STD = sexually transmitted disease; U.S. MEC = *U.S. Medical Eligibility Criteria for Contraceptive Use*.

* **Class A:** essential and mandatory in all circumstances for safe and effective use of the contraceptive method. **Class B:** contributes substantially to safe and effective use, but implementation may be considered within the public health and/or service context; the risk of not performing an examination or test should be balanced against the benefits of making the contraceptive method available. **Class C:** does not contribute substantially to safe and effective use of the contraceptive method.

† Weight (BMI) measurement is not needed to determine medical eligibility for any methods of contraception because all methods can be used (U.S. MEC 1) or generally can be used (U.S. MEC 2) among obese women (Box 1). However, measuring weight and calculating BMI at baseline might be helpful for monitoring any changes and counseling women who might be concerned about weight change perceived to be associated with their contraceptive method.

Lipids: Screening for dyslipidemias is not necessary for the safe initiation of implants because of the low prevalence of undiagnosed disease in women of reproductive age and the low likelihood of clinically significant changes with use of hormonal contraceptives. A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened with lipid measurement before initiation of hormonal contraceptives (*57*). During 2009–2012 among women aged 20–44 years in the United States, 7.6% had high cholesterol, defined as total serum cholesterol ≥240 mg/dL (*84*). During 1999–2008, the prevalence of undiagnosed hypercholesterolemia among women aged 20–44 years was approximately 2% (*85*). Studies have shown mixed results regarding the effects of hormonal methods on lipid levels among both healthy women and women with baseline lipid abnormalities, and the clinical significance of these changes is unclear (*86*–*89*).

Liver enzymes: Although women with certain liver diseases generally should not use implants (U.S. MEC 3) (*5*), screening for liver disease before initiation of implants is not necessary because of the low prevalence of these conditions and the high likelihood that women with liver disease already would have had the condition diagnosed. A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened with liver enzyme tests before initiation of hormonal contraceptives (*57*). In 2012, the percentage of U.S. women with liver disease (not further specified) was 1.3% (*90*). In 2013, the incidence of acute hepatitis A, B, or C was ≤1 per 100,000 U.S. population (*91*). During 2002–2011, the incidence of liver carcinoma among U.S. women was approximately 3.7 per 100,000 population (*92*). Because estrogen and progestins are metabolized in the liver, the use of hormonal contraceptives among women with liver disease might, theoretically, be a concern. The use of hormonal contraceptives, specifically COCs and POPs, does not affect disease progression or severity in women with hepatitis, cirrhosis, or benign focal nodular hyperplasia (*93*,*94*), although evidence is limited and no evidence exists for implants.

Clinical breast examination: Although women with current breast cancer should not use implants (U.S. MEC 4) (*5*), screening asymptomatic women with a clinical breast

examination before initiation of implants is not necessary because of the low prevalence of breast cancer among women of reproductive age (15–49 years). A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened with a breast examination before initiation of hormonal contraceptives (*95*). The incidence of breast cancer among women of reproductive age in the United States is low. In 2012, the incidence of breast cancer among women aged 20–49 years was approximately 70.7 per 100,000 women (*96*).

Other screening: Women with hypertension, diabetes, anemia, thrombogenic mutations, cervical intraepithelial neoplasia, cervical cancer, STDs, or HIV infection can use (U.S. MEC 1) or generally can use (U.S. MEC 2) implants (*5*); therefore, screening for these conditions is not necessary for the safe initiation of implants.

Routine Follow-Up After Implant Insertion

These recommendations address when routine follow-up is needed for safe and effective continued use of contraception for healthy women. The recommendations refer to general situations and might vary for different users and different situations. Specific populations who might benefit from more frequent follow-up visits include adolescents, those with certain medical conditions or characteristics, and those with multiple medical conditions.

- Advise the woman to return at any time to discuss side effects or other problems, if she wants to change the method being used, and when it is time to remove or replace the contraceptive method. No routine follow-up visit is required.
- At other routine visits, health care providers seeing implant users should do the following:
  - Assess the woman's satisfaction with her contraceptive method and whether she has any concerns about method use.
  - Assess any changes in health status, including medications, that would change the appropriateness of the implant for safe and effective continued use based on U.S. MEC (e.g., category 3 and 4 conditions and characteristics).
  - Consider assessing weight changes and counseling women who are concerned about weight change perceived to be associated with their contraceptive method.

Comments and Evidence Summary. A systematic review did not identify any evidence regarding whether a routine follow-up visit after initiating an implant improves correct or continued use (*122*).

Bleeding Irregularities (Including Amenorrhea) During Implant Use

- Before implant insertion, provide counseling about potential changes in bleeding patterns during implant use. Unscheduled spotting or light bleeding is common with implant use, and some women experience amenorrhea. These bleeding changes are generally not harmful and might or might not decrease with continued implant use. Heavy or prolonged bleeding, unscheduled or menstrual, is uncommon during implant use.

Irregular Bleeding (Spotting, Light Bleeding, or Heavy or Prolonged Bleeding)

- If clinically indicated, consider an underlying gynecological problem, such as interactions with other medications, an STD, pregnancy, or new pathologic uterine conditions (e.g., polyps or fibroids). If an underlying gynecological problem is found, treat the condition or refer for care.
- If an underlying gynecologic problem is not found and the woman wants treatment, the following treatment options during days of bleeding can be considered:
  - NSAIDS for short-term treatment (5–7 days)
  - Hormonal treatment (if medically eligible) with low-dose COCs or estrogen for short-term treatment (10–20 days)
- If irregular bleeding persists and the woman finds it unacceptable, counsel her on alternative methods, and offer another method if it is desired.

Amenorrhea

- Amenorrhea does not require any medical treatment. Provide reassurance.
  - If a woman's regular bleeding pattern changes abruptly to amenorrhea, consider ruling out pregnancy if clinically indicated.
- If amenorrhea persists and the woman finds it unacceptable, counsel her on alternative contraceptive methods, and offer another method if it is desired.

Comments and Evidence Summary. During contraceptive counseling and before insertion of the implant, information about common side effects, such as unscheduled spotting or light bleeding and amenorrhea, especially during the first year of use, should be discussed. A pooled analysis of data from 11 clinical trials indicates that a significant proportion of etonogestrel implant users had relatively little bleeding: 22% of women experienced amenorrhea and 34% experienced infrequent spotting, although 7% reported frequent bleeding

and 18% reported prolonged bleeding (*146*). Unscheduled bleeding or amenorrhea is generally not harmful. Enhanced counseling about expected bleeding patterns and reassurance that bleeding irregularities are generally not harmful has been shown to reduce discontinuation in clinical trials with other hormonal contraceptives (i.e., DMPA) (*124*,*125*).

A systematic review and four newly published studies examined several medications for the treatment of bleeding irregularities with primarily levonorgestrel contraceptive implants (*147*–*151*). Two small studies found significant cessation of bleeding within 7 days of start of treatment among women taking oral celecoxib (200 mg) daily for 5 days or oral mefenamic acid (500 mg) 3 times daily for 5 days compared with placebo (*149*,*150*). Differences in bleeding cessation were not found among women with etonogestrel implants taking mifepristone but were found when women with the implants combined mifepristone with either ethinyl estradiol or doxycycline (*151*,*152*). Doxycycline alone or in combination with ethinyl estradiol did not improve bleeding cessation among etonogestrel implant users (*151*). Among LNG implant users, mifepristone reduced the number of bleeding or spotting days but only after 6 months of treatment (*153*). Evidence also suggests that estrogen (*154*–*156*), daily COCs (*154*), LNG pills (*155*), tamoxifen (*157*), or tranexamic acid (*158*) can reduce the number of bleeding or spotting days during treatment among LNG implant users. In one small study, vitamin E was found to significantly reduce the mean number of bleeding days after the first treatment cycle; however, another larger study reported no significant differences in length of bleeding and spotting episodes with vitamin E treatment (*159*,*160*). Use of aspirin did not result in a significant difference in median length of bleeding or bleeding and spotting episodes after treatment (*159*). One study among implant users reported a reduction in number of bleeding days after initiating ibuprofen; however, another trial did not demonstrate any significant differences in the number of spotting and bleeding episodes with ibuprofen compared with placebo (*148*,*155*).

Injectables

Progestin-only injectable contraceptives (DMPA, 150 mg intramuscularly or 104 mg subcutaneously) are available in the United States; the only difference between these two formulations is the route of administration. Approximately 6 out of 100 women will become pregnant in the first year of use of DMPA with typical use (*14*). DMPA is reversible and can be used by women of all ages, including adolescents. DMPA does not protect against STDs; consistent and correct use of male latex condoms reduces the risk for STDs, including HIV.

Initiation of Injectables

Timing

- The first DMPA injection can be given at any time if it is reasonably certain that the woman is not pregnant (Box 2).

Need for Back-Up Contraception

- If DMPA is started within the first 7 days since menstrual bleeding started, no additional contraceptive protection is needed.
- If DMPA is started >7 days since menstrual bleeding started, the woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.

Special Considerations

Amenorrhea (Not Postpartum)

- **Timing:** The first DMPA injection can be given at any time if it is reasonably certain that the woman is not pregnant (Box 2).
- **Need for back-up contraception:** The woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.

Postpartum (Breastfeeding)

- **Timing:** The first DMPA injection can be given at any time, including immediately postpartum (U.S. MEC 2 if <1 month postpartum; U.S. MEC 1 if ≥1 month postpartum) if it is reasonably certain that the woman is not pregnant (Box 2).
- **Need for back-up contraception:** If the woman is <6 months postpartum, amenorrheic, and fully or nearly fully breastfeeding (exclusively breastfeeding or the vast majority [≥85%] of feeds are breastfeeds) (*27*), no additional contraceptive protection is needed. Otherwise, a woman who is ≥21 days postpartum and has not experienced return of her menstrual cycle needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days. If her menstrual cycles have returned and it has been >7 days since menstrual bleeding started, she needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.

Postpartum (Not Breastfeeding)

- **Timing:** The first DMPA injection can be given at any time, including immediately postpartum (U.S. MEC 1) if it is reasonably certain that the woman is not pregnant (Box 2).
- **Need for back-up contraception:** If a woman is <21 days postpartum, no additional contraceptive protection is needed. A woman who is ≥21 days postpartum and has

not experienced return of her menstrual cycle needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days. If her menstrual cycles have returned and it has been >7 days since menstrual bleeding started, she needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.

Postabortion (Spontaneous or Induced)

- **Timing:** The first DMPA injection can be given within the first 7 days, including immediately after the abortion (U.S. MEC 1).
- **Need for back-up contraception:** The woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days unless the injection is given at the time of a surgical abortion.

Switching from Another Contraceptive Method

- **Timing:** The first DMPA injection can be given immediately if it is reasonably certain that the woman is not pregnant (Box 2). Waiting for her next menstrual cycle is unnecessary.
- **Need for back-up contraception:** If it has been >7 days since menstrual bleeding started, the woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.
- **Switching from an IUD:** If the woman has had sexual intercourse since the start of her current menstrual cycle and it has been >5 days since menstrual bleeding started, theoretically, residual sperm might be in the genital tract, which could lead to fertilization if ovulation occurs. A health care provider may consider any of the following options:
  - Advise the women to retain the IUD for at least 7 days after the injection and return for IUD removal.
  - Advise the woman to abstain from sexual intercourse or use barrier contraception for 7 days before removing the IUD and switching to the new method.
  - If the woman cannot return for IUD removal and has not abstained from sexual intercourse or used barrier contraception for 7 days, advise the woman to use ECPs (with the exception of UPA) at the time of IUD removal.

Comments and Evidence Summary. In situations in which the health care provider is uncertain whether the woman might be pregnant, the benefits of starting DMPA likely exceed any risk; therefore, starting DMPA should be considered at any time, with a follow-up pregnancy test in 2–4 weeks. If a woman needs to use additional contraceptive protection when switching to DMPA from another contraceptive method, consider continuing her previous method for 7 days after DMPA injection.

A systematic review identified eight articles examining DMPA initiation on different days of the menstrual cycle (*161*). Evidence from two studies with small sample sizes indicated that DMPA injections given up to day 7 of the menstrual cycle inhibited ovulation; when DMPA was administered after day 7, ovulation occurred in some women. Cervical mucus was of poor quality (i.e., not favorable for sperm penetration) in 90% of women within 24 hours of the injection (Level of evidence: II-2, fair) (*162–164*). Studies found that use of another contraceptive method until DMPA could be initiated (bridging option) did not help women initiate DMPA and was associated with more unintended pregnancies than immediate receipt of DMPA (*165–169*) (Level of evidence: I to II-3, fair to poor, indirect).

Examinations and Tests Needed Before Initiation of an Injectable

Among healthy women, no examinations or tests are needed before initiation of DMPA, although a baseline weight and BMI measurement might be useful to monitor DMPA users over time (Table 3). Women with known medical problems or other special conditions might need additional examinations or tests before being determined to be appropriate candidates for a particular method of contraception. U.S. MEC might be useful in such circumstances (*5*).

Comments and Evidence Summary. Weight (BMI): Obese women can use (U.S. MEC 1) or generally can use (U.S. MEC 2) DMPA (*5*); therefore, screening for obesity is not necessary for the safe initiation of DMPA. However, measuring weight and calculating BMI at baseline might be helpful for monitoring any changes and counseling women who might be concerned about weight change perceived to be associated with their contraceptive method. (See guidance on follow-up for DMPA users for evidence on weight gain with DMPA use).

Bimanual examination and cervical inspection: Pelvic examination is not necessary before initiation of DMPA because it does not facilitate detection of conditions for which DMPA would be unsafe. Although women with current breast cancer should not use DMPA (U.S. MEC 4), and women with severe hypertension, heart disease, vascular disease, or certain liver diseases generally should not use DMPA (U.S. MEC 3) (*5*), none of these conditions are likely to be detected by pelvic examination (*145*). A systematic review identified two case-control studies that compared delayed versus immediate pelvic examination before initiation of hormonal contraceptives, specifically oral contraceptives or DMPA (*95*). No differences in risk factors for cervical neoplasia, incidence of STDs, incidence of abnormal Papanicolaou smears, or incidence of

TABLE 3. Classification of examinations and tests needed before depo-medroxyprogesterone acetate initiation

Examination or test	Class*
Examination	
Blood pressure	C
Weight (BMI) (weight [kg]/height [m]^2)	—†
Clinical breast examination	C
Bimanual examination and cervical inspection	C
Laboratory test	
Glucose	C
Lipids	C
Liver enzymes	C
Hemoglobin	C
Thrombogenic mutations	C
Cervical cytology (Papanicolaou smear)	C
STD screening with laboratory tests	C
HIV screening with laboratory tests	C

Abbreviations: BMI = body mass index; HIV = human immunodeficiency virus; STD = sexually transmitted disease; U.S. MEC = *U.S. Medical Eligibility Criteria for Contraceptive Use.*

* **Class A:** essential and mandatory in all circumstances for safe and effective use of the contraceptive method. **Class B:** contributes substantially to safe and effective use, but implementation may be considered within the public health and/or service context; the risk of not performing an examination or test should be balanced against the benefits of making the contraceptive method available. **Class C:** does not contribute substantially to safe and effective use of the contraceptive method.

† Weight (BMI) measurement is not needed to determine medical eligibility for any methods of contraception because all methods can be used (U.S. MEC 1) or generally can be used (U.S. MEC 2) among obese women (Box 1). However, measuring weight and calculating BMI at baseline might be helpful for monitoring any changes and counseling women who might be concerned about weight change perceived to be associated with their contraceptive method.

abnormal wet mounts were observed (Level of evidence: II-2, fair, direct).

Blood pressure: Women with hypertension generally can use DMPA (U.S. MEC 2), with the exception of women with severe hypertension or vascular disease, who generally should not use DMPA (U.S. MEC 3) (*5*). Screening for hypertension before initiation of DMPA is not necessary because of the low prevalence of undiagnosed severe hypertension and the high likelihood that women with these conditions already would have had them diagnosed. A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened with a blood pressure measurement before initiation of progestin-only contraceptives (*170*). The prevalence of undiagnosed hypertension among women of reproductive age is low. During 2009–2012 among women aged 20–44 years in the United States, the prevalence of hypertension was 8.7% (*84*). During 1999–2008, the percentage of women aged 20–44 years with undiagnosed hypertension was 1.9% (*85*).

Glucose: Although women with complicated diabetes generally should not use DMPA (U.S. MEC 3) (*5*), screening for diabetes before initiation of DMPA is not necessary because of the low prevalence of undiagnosed diabetes and the high likelihood that women with complicated diabetes would already have had the condition diagnosed. A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened with glucose measurement before initiation of hormonal contraceptives (*57*). The prevalence of diabetes among women of reproductive age is low. During 2009–2012 among women aged 20–44 years in the United States, the prevalence of diabetes was 3.3% (*84*). During 1999–2008, the percentage of women aged 20–44 years with undiagnosed diabetes was 0.5% (*85*). Although hormonal contraceptives can have some adverse effects on glucose metabolism in healthy and diabetic women, the overall clinical effect is minimal (*171–177*).

Lipids: Screening for dyslipidemias is not necessary for the safe initiation of injectables because of the low prevalence of undiagnosed disease in women of reproductive age and the low likelihood of clinically significant changes with use of hormonal contraceptives. A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened with lipid measurement before initiation of hormonal contraceptives (*57*). During 2009–2012 among women aged 20–44 years in the United States, 7.6% had high cholesterol, defined as total serum cholesterol ≥240 mg/dL (*84*). During 1999–2008, the prevalence of undiagnosed hypercholesterolemia among women aged 20–44 years was approximately 2% (*85*). Studies have shown mixed results about the effects of hormonal methods on lipid levels among both healthy women and women with baseline lipid abnormalities, and the clinical significance of these changes is unclear (*86–89*).

Liver enzymes: Although women with certain liver diseases generally should not use DMPA (U.S. MEC 3) (*5*), screening for liver disease before initiation of DMPA is not necessary because of the low prevalence of these conditions and the high likelihood that women with liver disease already would have had the condition diagnosed. A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened with liver enzyme tests before initiation of hormonal contraceptives (*57*). In 2012, among U.S. women, the percentage with liver disease (not further specified) was 1.3% (*90*). In 2013, the incidence of acute hepatitis A, B, or C was ≤1 per 100,000 U.S. population (*91*). During 2002–2011, the incidence of liver carcinoma among U.S. women was approximately 3.7 per 100,000 population (*92*). Because estrogen and progestins are metabolized in the liver, the use of hormonal contraceptives among women with liver disease might, theoretically, be a concern. The use of hormonal contraceptives, specifically COCs and POPs, does not affect disease progression or severity in women with hepatitis, cirrhosis, or benign focal nodular hyperplasia (*93,94*), although evidence is limited and no evidence exists for DMPA.

Clinical breast examination: Although women with current breast cancer should not use DMPA (U.S. MEC 4) (*5*), screening asymptomatic women with a clinical breast examination before initiating DMPA is not necessary because of the low prevalence of breast cancer among women of reproductive age. A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened with a clinical breast examination before initiation of hormonal contraceptives (*95*). The incidence of breast cancer among women of reproductive age in the United States is low. In 2012, the incidence of breast cancer among women aged 20–49 years was approximately 70.7 per 100,000 women (*96*).

Other screening: Women with anemia, thrombogenic mutations, cervical intraepithelial neoplasia, cervical cancer, HIV infection, or other STDs can use (U.S. MEC 1) or generally can use (U.S. MEC 2) DMPA (*5*); therefore, screening for these conditions is not necessary for the safe initiation of DMPA.

Routine Follow-Up After Injectable Initiation

These recommendations address when routine follow-up is recommended for safe and effective continued use of contraception for healthy women. The recommendations refer to general situations and might vary for different users and different situations. Specific populations who might benefit from frequent follow-up visits include adolescents, those with certain medical conditions or characteristics, and those with multiple medical conditions.

- Advise the woman to return at any time to discuss side effects or other problems, if she wants to change the method being used, and when it is time for reinjection. No routine follow-up visit is required.
- At other routine visits, health care providers seeing injectable users should do the following:
  - Assess the woman's satisfaction with her contraceptive method and whether she has any concerns about method use.
  - Assess any changes in health status, including medications, that would change the appropriateness of the injectable for safe and effective continued use based on U.S. MEC (e.g., category 3 and 4 conditions and characteristics).
  - Consider assessing weight changes and counseling women who are concerned about weight change perceived to be associated with their contraceptive method.

Comments and Evidence Summary. Although no evidence exists regarding whether a routine follow-up visit after initiating DMPA improves correct or continued use, monitoring weight or BMI change over time is important for DMPA users.

A systematic review identified a limited body of evidence that examined whether weight gain in the few months after DMPA initiation predicted future weight gain (*123*). Two studies found significant differences in weight gain or BMI at follow-up periods ranging from 12 to 36 months between early weight gainers (i.e., those who gained >5% of their baseline body weight within 6 months after initiation) and those who were not early weight gainers (*178*,*179*). The differences between groups were more pronounced at 18, 24, and 36 months than at 12 months. One study found that most adolescent DMPA users who had gained >5% of their baseline weight by 3 months gained even more weight by 12 months (*180*) (Level of evidence: II-2, fair, to II-3, fair, direct).

Timing of Repeat Injections

Reinjection Interval

- Provide repeat DMPA injections every 3 months (13 weeks).

Special Considerations

Early Injection

- The repeat DMPA injection can be given early when necessary.

Late Injection

- The repeat DMPA injection can be given up to 2 weeks late (15 weeks from the last injection) without requiring additional contraceptive protection.
- If the woman is >2 weeks late (>15 weeks from the last injection) for a repeat DMPA injection, she can have the injection if it is reasonably certain that she is not pregnant (Box 2). She needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days. She might consider the use of emergency contraception (with the exception of UPA) if appropriate.

Comments and Evidence Summary. No time limits exist for early injections; injections can be given when necessary (e.g., when a woman cannot return at the routine interval). WHO has extended the time that a woman can have a late reinjection (i.e., grace period) for DMPA use from 2 weeks to 4 weeks on the basis of data from one study showing low pregnancy rates through 4 weeks; however, the CDC expert group did not consider the data to be generalizable to the United States because a large proportion of women in the study were breastfeeding. Therefore, U.S. SPR recommends a grace period of 2 weeks.

A systematic review identified 12 studies evaluating time to pregnancy or ovulation after the last injection of DMPA (*181*). Although pregnancy rates were low during the 2-week interval following the reinjection date and for 4 weeks following the

reinjection date, data were sparse, and one study included a large proportion of breastfeeding women (*182–184*). Studies also indicated a wide variation in time to ovulation after the last DMPA injection, with the majority ranging from 15 to 49 weeks from the last injection (*185–193*) (Level of evidence: level II-2, fair, direct).

Bleeding Irregularities (Including Amenorrhea) During Injectable Use

- Before DMPA initiation, provide counseling about potential changes in bleeding patterns during DMPA use. Amenorrhea and unscheduled spotting or light bleeding is common with DMPA use, and heavy or prolonged bleeding can occur with DMPA use. These bleeding irregularities are generally not harmful and might decrease with continued DMPA use.

Unscheduled Spotting or Light Bleeding

- If clinically indicated, consider an underlying gynecological problem, such as interactions with other medications, an STD, pregnancy, or new pathologic uterine conditions (e.g., polyps or fibroids). If an underlying gynecological problem is found, treat the condition or refer for care.
- If an underlying gynecologic problem is not found and the woman wants treatment, the following treatment option during days of bleeding can be considered:
  - NSAIDs for short-term treatment (5–7 days)
- If unscheduled spotting or light bleeding persists and the woman finds it unacceptable, counsel her on alternative contraceptive methods, and offer another method if it is desired.

Heavy or Prolonged Bleeding

- If clinically indicated, consider an underlying gynecological problem, such as interactions with other medications, an STD, pregnancy, or new pathologic uterine conditions (such as fibroids or polyps). If an underlying gynecologic problem is identified, treat the condition or refer for care.
- If an underlying gynecologic problem is not found and the woman wants treatment, the following treatment options during days of bleeding can be considered:
  - NSAIDS for short-term treatment (5–7 days)
  - Hormonal treatment (if medically eligible) with low-dose COCs or estrogen for short-term treatment (10–20 days)
- If heavy or prolonged bleeding persists and the woman finds it unacceptable, counsel her on alternative contraceptive methods, and offer another method if it is desired.

Amenorrhea

- Amenorrhea does not require any medical treatment. Provide reassurance.
  - If a woman's regular bleeding pattern changes abruptly to amenorrhea, consider ruling out pregnancy if clinically indicated.
- If amenorrhea persists and the woman finds it unacceptable, counsel her on alternative contraceptive methods, and offer another method if it is desired.

Comments and Evidence Summary. During contraceptive counseling and before initiation of DMPA, information about common side effects such as irregular bleeding should be discussed. Unscheduled bleeding or spotting is common with DMPA use (*194*). In addition, amenorrhea is common after ≥1 years of continuous use (*194*,*195*). These bleeding irregularities are generally not harmful. Enhanced counseling among DMPA users detailing expected bleeding patterns and reassurance that these irregularities generally are not harmful has been shown to reduce DMPA discontinuation in clinical trials (*124*,*125*).

A systematic review, as well as two additional studies, examined the treatment of bleeding irregularities during DMPA use (*195–197*). Two small studies found significant cessation of bleeding within 7 days of starting treatment among women taking valdecoxib for 5 days or mefenamic acid for 5 days compared with placebo (*198*,*199*). Treatment with ethinyl estradiol was found to stop bleeding better than placebo during the treatment period, although rates of discontinuation were high and safety outcomes were not examined (*200*). In one small study among DMPA users who had been experiencing amenorrhea for 2 months, treatment with COCs was found to alleviate amenorrhea better than placebo (*201*). No studies examined the effects of aspirin on bleeding irregularities among DMPA users.

Combined Hormonal Contraceptives

Combined hormonal contraceptives contain both estrogen and a progestin and include 1) COCs (various formulations), 2) a transdermal contraceptive patch (which releases 150 μg of norelgestromin and 20 μg ethinyl estradiol daily), and 3) a vaginal contraceptive ring (which releases 120 μg etonogestrel and 15 μg ethinyl estradiol daily). Approximately 9 out of 100 women become pregnant in the first year of use with combined hormonal contraceptives with typical use (*14*). These methods are reversible and can be used by women of all ages. Combined hormonal contraceptives are generally used for

21–24 consecutive days, followed by 4–7 hormone-free days (either no use or placebo pills). These methods are sometimes used for an extended period with infrequent or no hormone-free days. Combined hormonal contraceptives do not protect against STDs; consistent and correct use of male latex condoms reduces the risk for STDs, including HIV.

Initiation of Combined Hormonal Contraceptives

Timing

- Combined hormonal contraceptives can be initiated at any time if it is reasonably certain that the woman is not pregnant (Box 2).

Need for Back-Up Contraception

- If combined hormonal contraceptives are started within the first 5 days since menstrual bleeding started, no additional contraceptive protection is needed.
- If combined hormonal contraceptives are started >5 days since menstrual bleeding started, the woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.

Special Considerations

Amenorrhea (Not Postpartum)

- **Timing:** Combined hormonal contraceptives can be started at any time if it is reasonably certain that the woman is not pregnant (Box 2).
- **Need for back-up contraception:** The woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.

Postpartum (Breastfeeding)

- **Timing:** Combined hormonal contraceptives can be started when the woman is medically eligible to use the method (*5*) and if it is reasonably certain that she is not pregnant. (Box 2).
- Postpartum women who are breastfeeding should not use combined hormonal contraceptives during the first 3 weeks after delivery (U.S. MEC 4) because of concerns about increased risk for venous thromboembolism and generally should not use combined hormonal contraceptives during the fourth week postpartum (U.S. MEC 3) because of concerns about potential effects on breastfeeding performance. Postpartum breastfeeding women with other risk factors for venous thromboembolism generally should not use combined hormonal contraceptives 4–6 weeks after delivery (U.S. MEC 3).
- **Need for back-up contraception:** If the woman is <6 months postpartum, amenorrheic, and fully or nearly fully breastfeeding (exclusively breastfeeding or the vast majority [≥85%] of feeds are breastfeeds) (*27*), no additional contraceptive protection is needed. Otherwise, a woman who is ≥21 days postpartum and has not experienced return of her menstrual cycle needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days. If her menstrual cycles have returned and it has been >5 days since menstrual bleeding started, she needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.

Postpartum (Not Breastfeeding)

- **Timing:** Combined hormonal contraceptives can be started when the woman is medically eligible to use the method (*5*) and if it is reasonably certain that the she is not pregnant (Box 2).
- Postpartum women should not use combined hormonal contraceptives during the first 3 weeks after delivery (U.S. MEC 4) because of concerns about increased risk for venous thromboembolism. Postpartum women with other risk factors for venous thromboembolism generally should not use combined hormonal contraceptives 3–6 weeks after delivery (U.S. MEC 3).
- **Need for back-up contraception:** If a woman is <21 days postpartum, no additional contraceptive protection is needed. A woman who is ≥21 days postpartum and whose menstrual cycles have not returned needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days. If her menstrual cycles have returned and it has been >5 days since menstrual bleeding started, she needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.

Postabortion (Spontaneous or Induced)

- **Timing:** Combined hormonal contraceptives can be started within the first 7 days following first-trimester or second-trimester abortion, including immediately postabortion (U.S. MEC 1).
- **Need for back-up contraception:** The woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days unless combined hormonal contraceptives are started at the time of a surgical abortion.

Switching from Another Contraceptive Method

- **Timing:** Combined hormonal contraceptives can be started immediately if it is reasonably certain that the

woman is not pregnant (Box 2). Waiting for her next menstrual cycle is unnecessary.

- **Need for back-up contraception:** If it has been >5 days since menstrual bleeding started, she needs to abstain from sexual intercourse or use additional contraceptive protection for the next 7 days.
- **Switching from an IUD:** If the woman has had sexual intercourse since the start of her current menstrual cycle and it has been >5 days since menstrual bleeding started, theoretically, residual sperm might be in the genital tract, which could lead to fertilization if ovulation occurs. A health care provider may consider any of the following options:
  - Advise the women to retain the IUD for at least 7 days after combined hormonal contraceptives are initiated and return for IUD removal.
  - Advise the woman to abstain from sexual intercourse or use barrier contraception for 7 days before removing the IUD and switching to the new method.
  - If the woman cannot return for IUD removal and has not abstained from sexual intercourse or used barrier contraception for 7 days, advise the woman to use ECPs at the time of IUD removal. Combined hormonal contraceptives can be started immediately after use of ECPs (with the exception of UPA). Combined hormonal contraceptives can be started no sooner than 5 days after use of UPA.

Comments and Evidence Summary. In situations in which the health care provider is uncertain whether the woman might be pregnant, the benefits of starting combined hormonal contraceptives likely exceed any risk; therefore, starting combined hormonal contraceptives should be considered at any time, with a follow-up pregnancy test in 2–4 weeks. If a woman needs to use additional contraceptive protection when switching to combined hormonal contraceptives from another contraceptive method, consider continuing her previous method for 7 days after starting combined hormonal contraceptives.

A systematic review of 18 studies examined the effects of starting combined hormonal contraceptives on different days of the menstrual cycle (*202*). Overall, the evidence suggested that pregnancy rates did not differ by the timing of combined hormonal contraceptive initiation (*169*,*203–205*) (Level of evidence: I to II-3, fair, indirect). The more follicular activity that occurred before starting COCs, the more likely ovulation was to occur; however, no ovulations occurred when COCs were started at a follicle diameter of 10 mm (mean cycle day 7.6) or when the ring was started at 13 mm (median cycle day 11) (*206–215*) (Level of evidence: I to II-3, fair, indirect). Bleeding patterns and other side effects did not vary with the timing of combined hormonal contraceptive initiation (*204*,*205*,*216–220*) (Level of evidence: I to II-2, good to poor, direct). Although continuation rates of combined hormonal contraceptives were initially improved by the "quick start" approach (i.e., starting on the day of the visit), the advantage disappeared over time (*203*,*204*,*216–221*) (Level of evidence: I to II-2, good to poor, direct).

Examinations and Test Needed Before Initiation of Combined Hormonal Contraceptives

Among healthy women, few examinations or tests are needed before initiation of combined hormonal contraceptives (Table 4). Blood pressure should be measured before initiation of combined hormonal contraceptives. Baseline weight and BMI measurements might be useful for monitoring combined hormonal contraceptive users over time. Women with known medical problems or other special conditions might need additional examinations or tests before being determined to be appropriate candidates for a particular method of contraception. U.S. MEC might be useful in such circumstances (*5*).

Comments and Evidence Summary. Blood pressure: Women who have more severe hypertension (systolic pressure of ≥160 mmHg or diastolic pressure of ≥100 mm Hg) or vascular disease should not use combined hormonal contraceptives (U.S. MEC 4), and women who have less severe hypertension (systolic pressure of 140–159 mm Hg or diastolic pressure of 90–99 mm Hg) or adequately controlled hypertension generally should not use combined hormonal contraceptives (U.S. MEC 3) (*5*). Therefore, blood pressure should be evaluated before initiating combined hormonal contraceptives. In instances in which blood pressure cannot be measured by a provider, blood pressure measured in other settings can be reported by the woman to her provider. Evidence suggests that cardiovascular outcomes are worse among women who did not have their blood pressure measured before initiating COCs. A systematic review identified six articles from three studies that reported cardiovascular outcomes among women who had blood pressure measurements and women who did not have blood pressure measurements before initiating COCs (*170*). Three case-control studies showed that women who did not have blood pressure measurements before initiating COCs had a higher risk for acute myocardial infarction than women who did have blood pressure measurements (*222–224*). Two case-control studies showed that women who did not have blood pressure measurements before initiating COCs had a higher risk for ischemic stroke than women who did have blood pressure measurements (*225*,*226*). One case-control study showed no difference in the risk for hemorrhagic stroke among women who initiated COCs regardless of whether their

TABLE 4. Classification of examinations and tests needed before combined hormonal contraceptive initiation

Examination or test	Class*
Examination	
Blood pressure	A†
Weight (BMI) (weight [kg]/height [m]^2)	—§
Clinical breast examination	C
Bimanual examination and cervical inspection	C
Laboratory test	
Glucose	C
Lipids	C
Liver enzymes	C
Hemoglobin	C
Thrombogenic mutations	C
Cervical cytology (Papanicolaou smear)	C
STD screening with laboratory tests	C
HIV screening with laboratory tests	C

Abbreviations: BMI = body mass index; HIV = human immunodeficiency virus; STD = sexually transmitted disease; U.S. MEC = *U.S. Medical Eligibility Criteria for Contraceptive Use.*

* **Class A:** essential and mandatory in all circumstances for safe and effective use of the contraceptive method. **Class B:** contributes substantially to safe and effective use, but implementation may be considered within the public health and/or service context; the risk of not performing an examination or test should be balanced against the benefits of making the contraceptive method available. **Class C:** does not contribute substantially to safe and effective use of the contraceptive method.

† In instances in which blood pressure cannot be measured by a provider, blood pressure measured in other settings can be reported by the woman to her provider.

§ Weight (BMI) measurement is not needed to determine medical eligibility for any methods of contraception because all methods can be used (U.S. MEC 1) or generally can be used (U.S. MEC 2) among obese women (Box 1). However, measuring weight and calculating BMI at baseline might be helpful for monitoring any changes and counseling women who might be concerned about weight change perceived to be associated with their contraceptive method.

blood pressure was measured (*227*). Studies that examined hormonal contraceptive methods other than COCs were not identified (Level of evidence: II-2, fair, direct).

Weight (BMI): Obese women generally can use combined hormonal contraceptives (U.S. MEC 2) (*5*); therefore, screening for obesity is not necessary for the safe initiation of combined hormonal contraceptives. However, measuring weight and calculating BMI at baseline might be helpful for monitoring any changes and counseling women who might be concerned about weight change perceived to be associated with their contraceptive method.

Bimanual examination and cervical inspection: Pelvic examination is not necessary before initiation of combined hormonal contraceptives because it does not facilitate detection of conditions for which hormonal contraceptives would be unsafe. Women with certain conditions such as current breast cancer, severe hypertension or vascular disease, heart disease, migraine headaches with aura, and certain liver diseases, as well as women aged ≥35 years and who smoke ≥15 cigarettes per day, should not use (U.S. MEC 4) or generally should not use (U.S. MEC 3) combined hormonal contraceptives (*5*); however, none of these conditions are likely to be detected by pelvic examination (*145*). A systematic review identified two case-control studies that compared delayed and immediate pelvic examination before initiation of hormonal contraceptives, specifically oral contraceptives or DMPA (*95*). No differences in risk factors for cervical neoplasia, incidence of STDs, incidence of abnormal Papanicolaou smears, or incidence of abnormal wet mounts were found (Level of evidence: Level II-2 fair, direct).

Glucose: Although women with complicated diabetes should not use (U.S. MEC 4) or generally should not use (U.S. MEC 3) combined hormonal contraceptives, depending on the severity of the condition (*5*), screening for diabetes before initiation of hormonal contraceptives is not necessary because of the low prevalence of undiagnosed diabetes and the high likelihood that women with complicated diabetes already would have had the condition diagnosed. A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened with glucose measurement before initiation of hormonal contraceptives (*57*). The prevalence of diabetes among women of reproductive age is low. During 2009–2012 among women aged 20–44 years in the United States, the prevalence of diabetes was 3.3% (*84*). During 1999–2008, the percentage of women aged 20–44 years with undiagnosed diabetes was 0.5% (*85*). Although hormonal contraceptives can have some adverse effects on glucose metabolism in healthy and diabetic women, the overall clinical effect is minimal (*171–177*).

Lipids: Screening for dyslipidemias is not necessary for the safe initiation of combined hormonal contraceptives because of the low prevalence of undiagnosed disease in women of reproductive age and the low likelihood of clinically significant changes with use of hormonal contraceptives. A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened with lipid measurement before initiation of hormonal contraceptives (*57*). During 2009–2012 among women aged 20–44 years in the United States, 7.6% had high cholesterol, defined as total serum cholesterol ≥240 mg/dL (*84*). During 1999–2008, the prevalence of undiagnosed hypercholesterolemia among women aged 20–44 years was approximately 2% (*85*). A systematic review identified few studies, all of poor quality, that suggest that women with known dyslipidemias using combined hormonal contraceptives might be at increased risk for myocardial infarction, cerebrovascular accident, or venous thromboembolism compared with women without dyslipidemias; no studies were identified that examined risk for pancreatitis among women with known dyslipidemias using combined hormonal contraceptives (*89*). Studies have shown mixed results regarding the effects of hormonal contraceptives on lipid levels among both healthy women and women with

baseline lipid abnormalities, and the clinical significance of these changes is unclear (*86–89*).

Liver enzymes: Although women with certain liver diseases should not use (U.S. MEC 4) or generally should not use (U.S. MEC 3) combined hormonal contraceptives (*5*), screening for liver disease before initiation of combined hormonal contraceptives is not necessary because of the low prevalence of these conditions and the high likelihood that women with liver disease already would have had the condition diagnosed. A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened with liver enzyme tests before initiation of hormonal contraceptives (*57*). In 2012, among U.S. women, the percentage with liver disease (not further specified) was 1.3% (*90*). In 2013, the incidence of acute hepatitis A, B, or C was ≤1 per 100,000 U.S. population (*91*). During 2002–2011, the incidence of liver carcinoma among U.S. women was approximately 3.7 per 100,000 population (*92*). Because estrogen and progestins are metabolized in the liver, the use of hormonal contraceptives among women with liver disease might, theoretically, be a concern. The use of hormonal contraceptives, specifically COCs and POPs, does not affect disease progression or severity in women with hepatitis, cirrhosis, or benign focal nodular hyperplasia (*93*,*94*), although evidence is limited; no evidence exists for other types of combined hormonal contraceptives.

Thrombogenic mutations: Women with thrombogenic mutations should not use combined hormonal contraceptives (U.S. MEC 4) (*5*) because of the increased risk for venous thromboembolism (*228*). However, studies have shown that universal screening for thrombogenic mutations before initiating COCs is not cost-effective because of the rarity of the conditions and the high cost of screening (*229–231*).

Clinical breast examination: Although women with current breast cancer should not use combined hormonal contraceptives (U.S. MEC 4) (*5*), screening asymptomatic women with a clinical breast examination before initiating combined hormonal contraceptives is not necessary because of the low prevalence of breast cancer among women of reproductive age. A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened with a breast examination before initiation of hormonal contraceptives (*95*). The incidence of breast cancer among women of reproductive age in the United States is low. In 2012, the incidence of breast cancer among women aged 20–49 years was approximately 70.7 per 100,000 women (*96*).

Other screening: Women with anemia, cervical intraepithelial neoplasia, cervical cancer, HIV infection, or other STDs can use (U.S. MEC 1) or generally can use (U.S. MEC 2) combined hormonal contraceptives (*5*); therefore, screening for these conditions is not necessary for the safe initiation of combined hormonal contraceptives.

Number of Pill Packs that Should Be Provided at Initial and Return Visits

- At the initial and return visits, provide or prescribe up to a 1-year supply of COCs (e.g., 13 28-day pill packs), depending on the woman's preferences and anticipated use.
- A woman should be able to obtain COCs easily in the amount and at the time she needs them.

Comments and Evidence Summary. The more pill packs given up to 13 cycles, the higher the continuation rates. Restricting the number of pill packs distributed or prescribed can result in unwanted discontinuation of the method and increased risk for pregnancy.

A systematic review of the evidence suggested that providing a greater number of pill packs was associated with increased continuation (*232*). Studies that compared provision of one versus 12 packs, one versus 12 or 13 packs, or three versus seven packs found increased continuation of pill use among women provided with more pill packs (*233–235*). However, one study found no difference in continuation when patients were provided one and then three packs versus four packs all at once (*236*). In addition to continuation, a greater number of pills packs provided was associated with fewer pregnancy tests, fewer pregnancies, and lower cost per client. However, a greater number of pill packs (i.e., 13 packs versus three packs) also was associated with increased pill wastage in one study (*234*) (Level of evidence: I to II-2, fair, direct).

Routine Follow-Up After Combined Hormonal Contraceptive Initiation

These recommendations address when routine follow-up is recommended for safe and effective continued use of contraception for healthy women. The recommendations refer to general situations and might vary for different users and different situations. Specific populations who might benefit from more frequent follow-up visits include adolescents, those with certain medical conditions or characteristics, and those with multiple medical conditions.

- Advise the woman to return at any time to discuss side effects or other problems or if she wants to change the method being used. No routine follow-up visit is required.
- At other routine visits, health care providers seeing combined hormonal contraceptive users should do the following:

- Assess the woman's satisfaction with her contraceptive method and whether she has any concerns about method use.
- Assess any changes in health status, including medications, that would change the appropriateness of combined hormonal contraceptives for safe and effective continued use based on U.S. MEC (e.g., category 3 and 4 conditions and characteristics).
- Assess blood pressure.
- Consider assessing weight changes and counseling women who are concerned about weight change perceived to be associated with their contraceptive method.

Comments and Evidence Summary. No evidence exists regarding whether a routine follow-up visit after initiating combined hormonal contraceptives improves correct or continued use. Monitoring blood pressure is important for combined hormonal contraceptive users. Health care providers might consider recommending women obtain blood pressure measurements in other settings.

A systematic review identified five studies that examined the incidence of hypertension among women who began using a COC versus those who started a nonhormonal method of contraception or a placebo (*123*). Few women developed hypertension after initiating COCs, and studies examining increases in blood pressure after COC initiation found mixed results. No studies were identified that examined changes in blood pressure among patch or vaginal ring users (Level of evidence: I, fair, to II-2, fair, indirect).

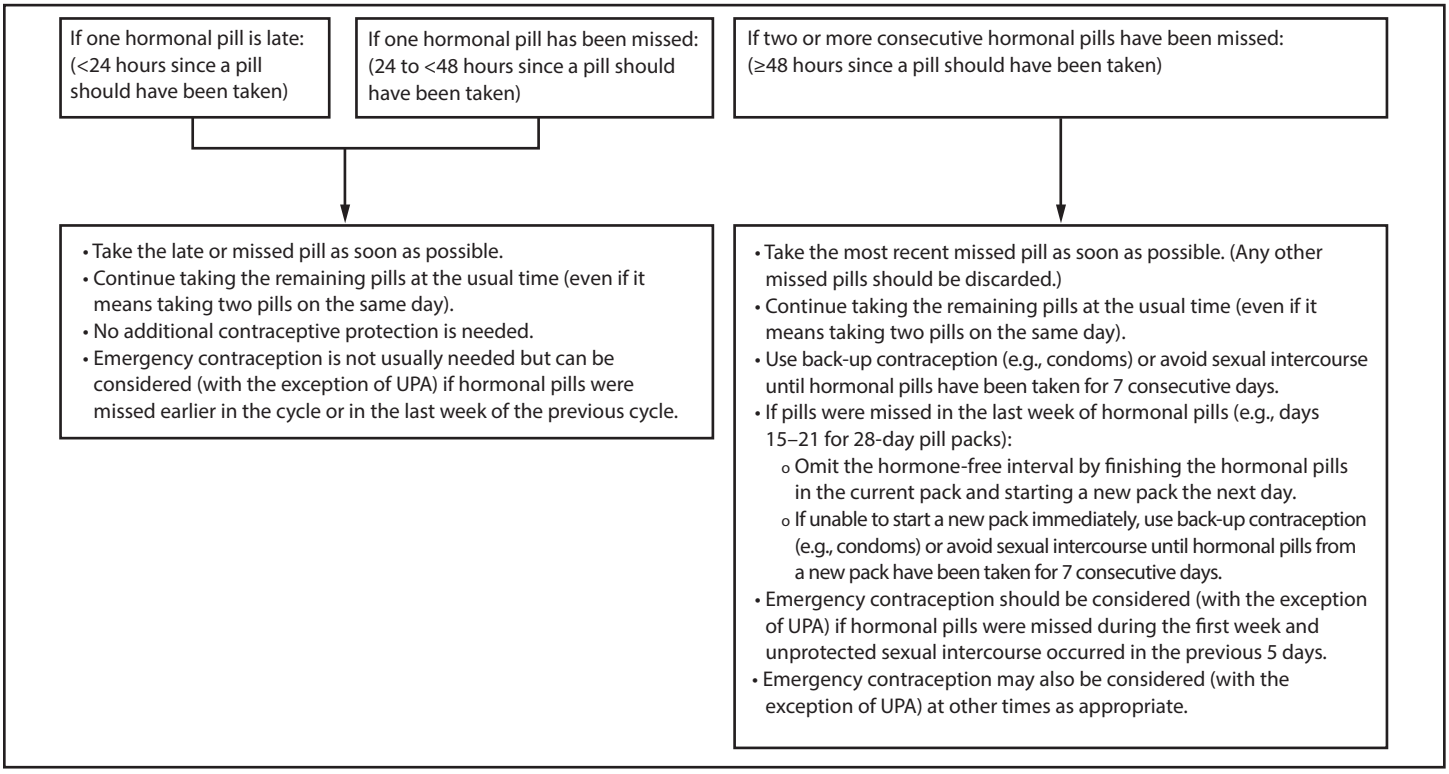
Late or Missed Doses and Side Effects from Combined Hormonal Contraceptive Use

For the following recommendations, a dose is considered late when <24 hours have elapsed since the dose should have been taken. A dose is considered missed if ≥24 hours have elapsed since the dose should have been taken. For example, if a COC pill was supposed to have been taken on Monday at 9:00 a.m. and is taken at 11:00 a.m., the pill is late; however, by Tuesday morning at 11:00 a.m., Monday's 9:00 a.m. pill has been missed and Tuesday's 9:00 a.m. pill is late. For COCs, the recommendations only apply to late or missed hormonally active pills and not to placebo pills. Recommendations are provided for late or missed pills (Figure 2), the patch (Figure 3), and the ring (Figure 4).

Comments and Evidence Summary. Inconsistent or incorrect use of combined hormonal contraceptives is a major cause of combined hormonal contraceptive failure. Extending the hormone-free interval is considered to be a particularly risky time to miss combined hormonal contraceptives. Seven days of continuous combined hormonal contraceptive use is deemed necessary to reliably prevent ovulation. The recommendations reflect a balance between simplicity and precision of science. Women who frequently miss COCs or experience other usage errors with combined hormonal patch or combined vaginal ring should consider an alternative contraceptive method that is less dependent on the user to be effective (e.g., IUD, implant, or injectable).

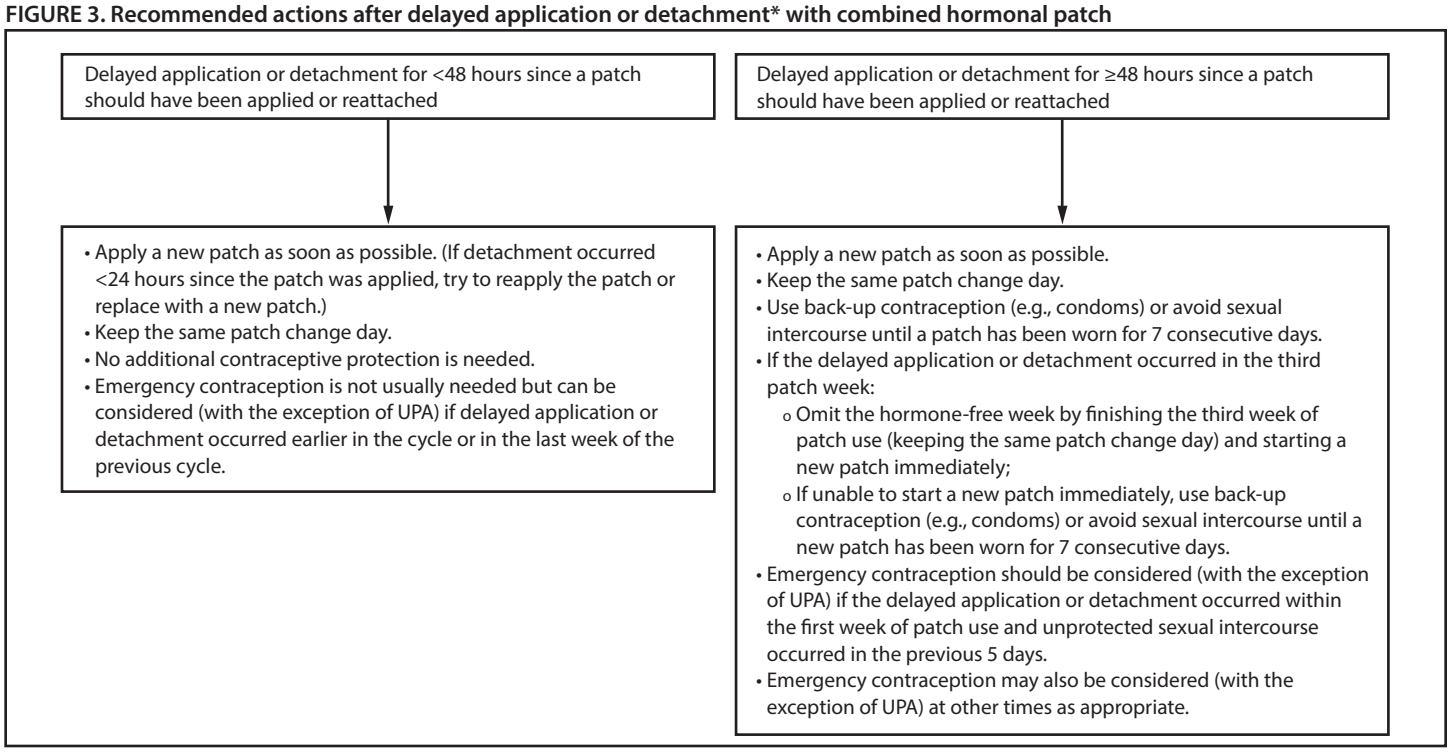
A systematic review identified 36 studies that examined measures of contraceptive effectiveness of combined hormonal contraceptives during cycles with extended hormone-free intervals, shortened hormone-free intervals, or deliberate nonadherence on days not adjacent to the hormone-free interval (*237*). Most of the studies examined COCs *(215*,*238–265)*, two examined the combined hormonal patch (*259*,*266*), and six examined the combined vaginal ring (*211*,*267–271*). No direct evidence on the effect of missed pills on the risk for pregnancy was found. Studies of women deliberately extending the hormone-free interval up to 14 days found wide variability in the amount of follicular development and occurrence of ovulation (*241*,*244*,*246*,*247*,*249*,*250*,*252–255*); in general, the risk for ovulation was low, and among women who did ovulate, cycles were usually abnormal. In studies of women who deliberately missed pills on various days during the cycle not adjacent to the hormone-free interval, ovulation occurred infrequently (*239*,*245–247*,*255*,*256*,*258*,*259*). Studies comparing 7-day hormone-free intervals with shorter hormone-free intervals found lower rates of pregnancy (*238*,*242*,*251*,*257*) and significantly greater suppression of ovulation (*240*,*250*,*261–263*,*265*) among women with shorter intervals in all but one study (*260*), which found no difference. Two studies that compared 30-μg ethinyl estradiol pills with 20-μg ethinyl estradiol pills showed more follicular activity when 20-μg ethinyl estradiol pills were missed (*241*,*244*). In studies examining the combined vaginal ring, three studies found that nondeliberate extension of the hormone-free interval for 24 to <48 hours from the scheduled period did not increase the risk for pregnancy (*267*,*268*,*270*); one study found that ring insertion after a deliberately extended hormone-free interval that allowed a 13-mm follicle to develop interrupted ovarian function and further follicular growth (*211*); and one study found that inhibition of ovulation was maintained after deliberately forgetting to remove the ring for up to 2 weeks after normal ring use (*271*). In studies examining the combined hormonal patch, one study found that missing 1–3 consecutive days before patch replacement (either wearing one patch 3 days longer before replacement or going 3 days without a patch before replacing the next patch) on days not adjacent to the patch-free interval resulted in little follicular activity and low risk for ovulation (*259*), and one pharmacokinetic study found that serum levels of

FIGURE 2. Recommended actions after late or missed combined oral contraceptives



Abbreviation: UPA = ulipristal acetate.

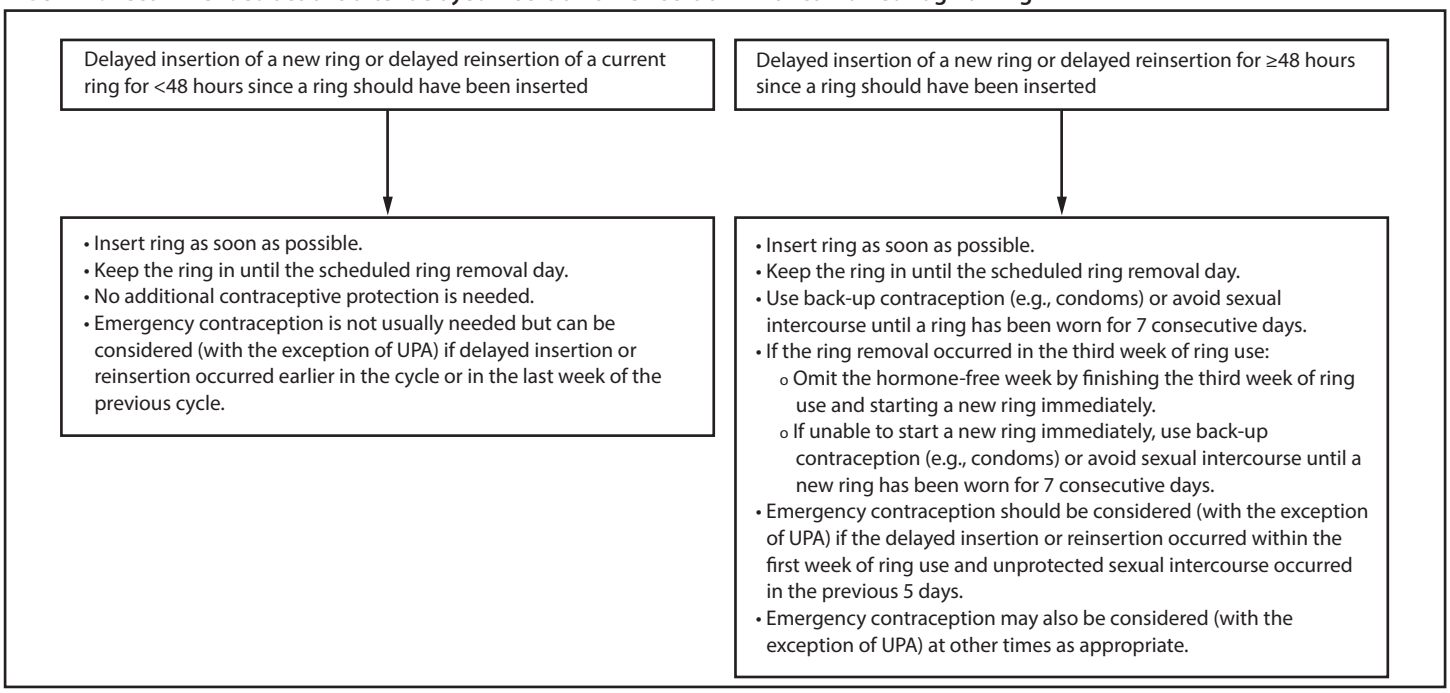
FIGURE 3. Recommended actions after delayed application or detachment* with combined hormonal patch



Abbreviation: UPA = ulipristal acetate.

* If detachment takes place but the woman is unsure when the detachment occurred, consider the patch to have been detached for ≥48 hours since a patch should have been applied or reattached.

FIGURE 4. Recommended actions after delayed insertion or reinsertion* with combined vaginal ring



Abbreviation: UPA = ulipristal acetate.
* If removal takes place but the woman is unsure of how long the ring has been removed, consider the ring to have been removed for ≥48 hours since a ring should have been inserted or reinserted.

ethinyl estradiol and progestin norelgestromin remained within reference ranges after extending patch wear for 3 days (*266*). No studies were found on extending the patch-free interval. In studies that provide indirect evidence on the effects of missed combined hormonal contraception on surrogate measures of pregnancy, how differences in surrogate measures correspond to pregnancy risk is unclear (Level of evidence: I, good, indirect to II-3, poor, direct).

Vomiting or Severe Diarrhea While Using COCs

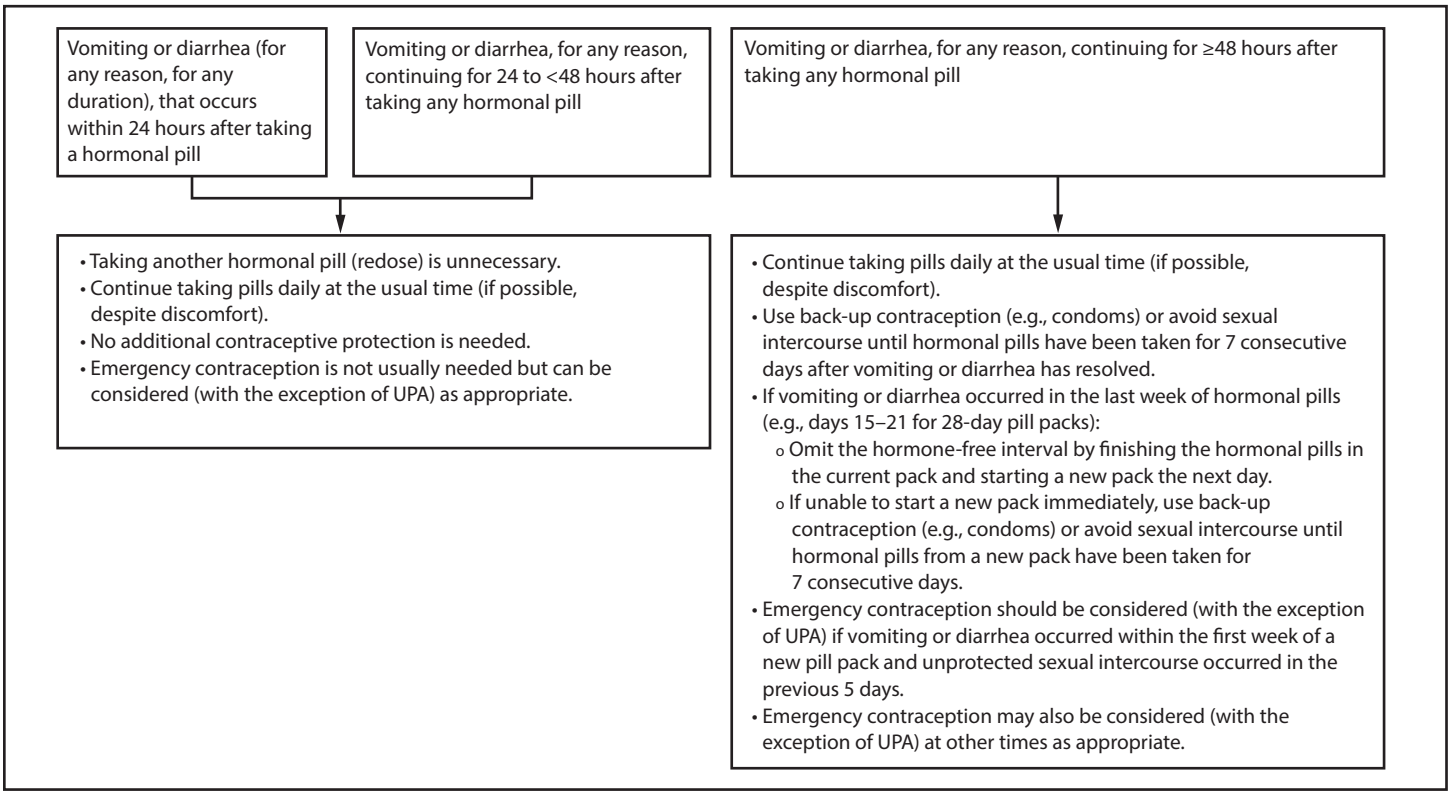
Certain steps should be taken by women who experience vomiting or severe diarrhea while using COCs (Figure 5).

Comments and Evidence Summary. Theoretically, the contraceptive effectiveness of COCs might be decreased because of vomiting or severe diarrhea. Because of the lack of evidence that addresses vomiting or severe diarrhea while using COCs, these recommendations are based on the recommendations for missed COCs. No evidence was found on the effects of vomiting or diarrhea on measures of contraceptive effectiveness including pregnancy, follicular development, hormone levels, or cervical mucus quality.

Unscheduled Bleeding with Extended or Continuous Use of Combined Hormonal Contraceptives

- Before initiation of combined hormonal contraceptives, provide counseling about potential changes in bleeding patterns during extended or continuous combined hormonal contraceptive use. (Extended contraceptive use is defined as a planned hormone-free interval after at least two contiguous cycles. Continuous contraceptive use is defined as uninterrupted use of hormonal contraception without a hormone-free interval) (*272*).
- Unscheduled spotting or bleeding is common during the first 3–6 months of extended or continuous combined hormonal contraceptive use. It is generally not harmful and decreases with continued combined hormonal contraceptive use.
- If clinically indicated, consider an underlying gynecological problem, such as inconsistent use, interactions with other medications, cigarette smoking, an STD, pregnancy, or new pathologic uterine conditions (e.g., polyps or fibroids). If an underlying gynecological problem is found, treat the condition or refer for care.
- If an underlying gynecological problem is not found and the woman wants treatment, the following treatment option can be considered:

FIGURE 5. Recommended actions after vomiting or diarrhea while using combined oral contraceptives



Abbreviation: UPA = ulipristal acetate.

  - Advise the woman to discontinue combined hormonal contraceptive use (i.e., a hormone-free interval) for 3–4 consecutive days; a hormone-free interval is not recommended during the first 21 days of using the continuous or extended combined hormonal contraceptive method. A hormone-free interval also is not recommended more than once per month because contraceptive effectiveness might be reduced.
- If unscheduled spotting or bleeding persists and the woman finds it unacceptable, counsel her on alternative contraceptive methods, and offer another method if it is desired.

Comments and Evidence Summary. During contraceptive counseling and before initiating extended or continuous combined hormonal contraceptives, information about common side effects such as unscheduled spotting or bleeding, especially during the first 3–6 months of use, should be discussed (*273*). These bleeding irregularities are generally not harmful and usually improve with persistent use of the hormonal method. To avoid unscheduled spotting or bleeding, counseling should emphasize the importance of correct use and timing; for users of contraceptive pills, emphasize consistent pill use. Enhanced counseling about expected bleeding patterns and reassurance that bleeding irregularities are generally not harmful has been shown to reduce method discontinuation in clinical trials with DMPA (*124*,*125*,*274*).

A systematic review identified three studies with small study populations that addressed treatments for unscheduled bleeding among women using extended or continuous combined hormonal contraceptives (*275*). In two separate randomized clinical trials in which women were taking either contraceptive pills or using the contraceptive ring continuously for 168 days, women assigned to a hormone-free interval of 3 or 4 days reported improved bleeding. Although they noted an initial increase in flow, this was followed by an abrupt decrease 7–8 days later with eventual cessation of flow 11–12 days later. These findings were compared with women who continued to use their method without a hormone-free interval, in which a greater proportion reported either treatment failure or fewer days of amenorrhea (*276*,*277*). In another randomized trial of 66 women with unscheduled bleeding among women using 84 days of hormonally active contraceptive pills, oral doxycycline (100 mg twice daily) initiated the first day of bleeding and taken for 5 days did not result in any improvement in bleeding compared with placebo (*278*) (Level of evidence: I, fair, direct).

Progestin-Only Pills

POPs contain only a progestin and no estrogen and are available in the United States. Approximately 9 out of 100 women become pregnant in the first year of use with POPs with typical use (*14*). POPs are reversible and can be used by women of all ages. POPs do not protect against STDs; consistent and correct use of male latex condoms reduces the risk for STDs, including HIV.

Initiation of POPs

Timing

- POPs can be started at any time if it is reasonably certain that the woman is not pregnant (Box 2).

Need for Back-Up Contraception

- If POPs are started within the first 5 days since menstrual bleeding started, no additional contraceptive protection is needed.
- If POPs are started >5 days since menstrual bleeding started, the woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 2 days.

Special Considerations

Amenorrhea (Not Postpartum)

- **Timing:** POPs can be started at any time if it is reasonably certain that the woman is not pregnant (Box 2).
- **Need for back-up contraception:** The woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 2 days.

Postpartum (Breastfeeding)

- **Timing:** POPs can be started at any time, including immediately postpartum (U.S. MEC 2 if <1 month postpartum; U.S. MEC 1 if ≥1 month postpartum) if it is reasonably certain that the woman is not pregnant (Box 2).
- **Need for back-up contraception:** If the woman is <6 months postpartum, amenorrheic, and fully or nearly fully breastfeeding (exclusively breastfeeding or the vast majority [≥85%] of feeds are breastfeeds) (*27*), no additional contraceptive protection is needed. Otherwise, a woman who is ≥21 days postpartum and has not experienced return of her menstrual cycles, she needs to abstain from sexual intercourse or use additional contraceptive protection for the next 2 days. If her menstrual cycles have returned and it has been >5 days since menstrual bleeding started, she needs to abstain from sexual intercourse or use additional contraceptive protection for the next 2 days.

Postpartum (Not Breastfeeding)

- **Timing:** POPs can be started at any time, including immediately postpartum (U.S. MEC 1), if it is reasonably certain that the woman is not pregnant (Box 2).
- **Need for back-up contraception:** If a woman is <21 days postpartum, no additional contraceptive protection is needed. Women who are ≥21 days postpartum and whose menstrual cycles have not returned need to abstain from sexual intercourse or use additional contraceptive protection for the next 2 days. If her menstrual cycles have returned and it has been >5 days since menstrual bleeding started, she needs to abstain from sexual intercourse or use additional contraceptive protection for the next 2 days.

Postabortion (Spontaneous or Induced)

- **Timing:** POPs can be started within the first 7 days, including immediately postabortion (U.S. MEC 1).
- **Need for back-up contraception:** The woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 2 days unless POPs are started at the time of a surgical abortion.

Switching from Another Contraceptive Method

- **Timing:** POPs can be started immediately if it is reasonably certain that the woman is not pregnant (Box 2). Waiting for her next menstrual cycle is unnecessary.
- **Need for back-up contraception:** If it has been >5 days since menstrual bleeding started, the woman needs to abstain from sexual intercourse or use additional contraceptive protection for the next 2 days.
- **Switching from an IUD:** If the woman has had sexual intercourse since the start of her current menstrual cycle and it has been >5 days since menstrual bleeding started, theoretically, residual sperm might be in the genital tract, which could lead to fertilization if ovulation occurs. A health care provider may consider any of the following options:
  - Advise the women to retain the IUD for at least 2 days after POPs are initiated and return for IUD removal.
  - Advise the woman to abstain from sexual intercourse or use barrier contraception for 7 days before removing the IUD and switching to the new method.
  - If the woman cannot return for IUD removal and has not abstained from sexual intercourse or used barrier contraception for 7 days, advise the woman to use ECPs at the time of IUD removal. POPs can be started immediately after use of ECPs (with the exception of UPA). POPs can be started no sooner than 5 days after use of UPA.

Comments and Evidence Summary. In situations in which the health care provider is uncertain whether the woman might

be pregnant, the benefits of starting POPs likely exceed any risk; therefore, starting POPs should be considered at any time, with a follow-up pregnancy test in 2–4 weeks.

Unlike COCs, POPs inhibit ovulation in about half of cycles, although the rates vary widely by individual (*279*). Peak serum steroid levels are reached about 2 hours after administration, followed by rapid distribution and elimination, such that by 24 hours after administration, serum steroid levels are near baseline (*279*). Therefore, taking POPs at approximately the same time each day is important. An estimated 48 hours of POP use has been deemed necessary to achieve the contraceptive effects on cervical mucus (*279*). If a woman needs to use additional contraceptive protection when switching to POPs from another contraceptive method, consider continuing her previous method for 2 days after starting POPs. No direct evidence was found regarding the effects of starting POPs at different times of the cycle.

Examinations and Tests Needed Before Initiation of POPs

Among healthy women, no examinations or tests are needed before initiation of POPs, although a baseline weight and BMI measurement might be useful for monitoring POP users over time (Table 5). Women with known medical problems or other special conditions might need additional examinations or tests before being determined to be appropriate candidates for a particular method of contraception. The U.S. MEC might be useful in such circumstances (*5*).

Comments and Evidence Summary. Weight (BMI): Obese women can use POPs (U.S. MEC 1) (*5*); therefore, screening for obesity is not necessary for the safe initiation of POPs. However, measuring weight and calculating BMI at baseline might be helpful for monitoring any changes and counseling women who might be concerned about weight change perceived to be associated with their contraceptive method.

Bimanual examination and cervical inspection: Pelvic examination is not necessary before initiation of POPs because it does not facilitate detection of conditions for which POPs would be unsafe. Women with current breast cancer should not use POPs (U.S. MEC 4), and women with certain liver diseases generally should not use POPs (U.S. MEC 3) (*5*); however, neither of these conditions are likely to be detected by pelvic examination (*145*). A systematic review identified two case-control studies that compared delayed versus immediate pelvic examination before initiation of hormonal contraceptives, specifically oral contraceptives or DMPA (*95*). No differences in risk factors for cervical neoplasia, incidence of STDs, incidence of abnormal Papanicolaou smears, or incidence of abnormal findings from wet mounts were observed (Level of evidence: II-2 fair, direct).

TABLE 5. Classification of examinations and tests needed before progestin-only pill initiation

Examination or test	Class*
Examination	
Blood pressure	C
Weight (BMI) (weight [kg]/height [m]^2)	—†
Clinical breast examination	C
Bimanual examination and cervical inspection	C
Laboratory test	
Glucose	C
Lipids	C
Liver enzymes	C
Hemoglobin	C
Thrombogenic mutations	C
Cervical cytology (Papanicolaou smear)	C
STD screening with laboratory tests	C
HIV screening with laboratory tests	C

Abbreviations: BMI = body mass index; HIV = human immunodeficiency virus; STD = sexually transmitted disease; U.S. MEC = *U.S. Medical Eligibility Criteria for Contraceptive Use*.

* **Class A:** essential and mandatory in all circumstances for safe and effective use of the contraceptive method. **Class B:** contributes substantially to safe and effective use, but implementation may be considered within the public health and/or service context; the risk of not performing an examination or test should be balanced against the benefits of making the contraceptive method available. **Class C:** does not contribute substantially to safe and effective use of the contraceptive method.

† Weight (BMI) measurement is not needed to determine medical eligibility for any methods of contraception because all methods can be used (U.S. MEC 1) or generally can be used (U.S. MEC 2) among obese women (Box 1). However, measuring weight and calculating BMI at baseline might be helpful for monitoring any changes and counseling women who might be concerned about weight change perceived to be associated with their contraceptive method.

Lipids: Screening for dyslipidemias is not necessary for the safe initiation of POPs because of the low prevalence of undiagnosed disease in women of reproductive age and the low likelihood of clinically significant changes with use of hormonal contraceptives. A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened with lipid measurement before initiation of hormonal contraceptives (*57*). During 2009–2012 among women aged 20–44 years in the United States, 7.6% had high cholesterol, defined as total serum cholesterol ≥240 mg/dL (*84*). During 1999–2008, the prevalence of undiagnosed hypercholesterolemia among women aged 20–44 years was approximately 2% (*85*). Studies have shown mixed results about the effects of hormonal methods on lipid levels among both healthy women and women with baseline lipid abnormalities, and the clinical significance of these changes is unclear (*86–89*).

Liver enzymes: Although women with certain liver diseases generally should not use POPs (U.S. MEC 3) (*5*), screening for liver disease before initiation of POPs is not necessary because of the low prevalence of these conditions and the high likelihood that women with liver disease already would

have had the condition diagnosed. A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened with liver enzyme tests before initiation of hormonal contraceptives (*57*). In 2012, among U.S. women, the percentage with liver disease (not further specified) was 1.3% (*90*). In 2013, the incidence of acute hepatitis A, B, or C was ≤1 per 100,000 U.S. population (*91*). During 2002–2011, the incidence of liver carcinoma among U.S. women was approximately 3.7 per 100,000 population (*92*). Because estrogen and progestins are metabolized in the liver, the use of hormonal contraceptives among women with liver disease might, theoretically, be a concern. The use of hormonal contraceptives, specifically COCs and POPs, does not affect disease progression or severity in women with hepatitis, cirrhosis, or benign focal nodular hyperplasia (*93*,*94*).

Clinical breast examination: Although women with current breast cancer should not use POPs (U.S. MEC 4) (*5*), screening asymptomatic women with a clinical breast examination before initiating POPs is not necessary because of the low prevalence of breast cancer among women of reproductive age. A systematic review did not identify any evidence regarding outcomes among women who were screened versus not screened with a clinical breast examination before initiation of hormonal contraceptives (*95*). The incidence of breast cancer among women of reproductive age in the United States is low. In 2012, the incidence of breast cancer among women aged 20–49 years was approximately 70.7 per 100,000 women (*96*).

Other screening: Women with hypertension, diabetes, anemia, thrombogenic mutations, cervical intraepithelial neoplasia, cervical cancer, STDs, or HIV infection can use (U.S. MEC 1) or generally can use (U.S. MEC 2) POPs (*5*); therefore, screening for these conditions is not necessary for the safe initiation of POPs.

Number of Pill Packs that Should Be Provided at Initial and Return Visits

- At the initial and return visit, provide or prescribe up to a 1-year supply of POPs (e.g., 13 28-day pill packs), depending on the woman's preferences and anticipated use.
- A woman should be able to obtain POPs easily in the amount and at the time she needs them.

Comments and Evidence Summary. The more pill packs given up to 13 cycles, the higher the continuation rates. Restricting the number of pill packs distributed or prescribed can result in unwanted discontinuation of the method and increased risk for pregnancy.

A systematic review of the evidence suggested that providing a greater number of pill packs was associated with increased continuation (*232*). Studies that compared provision of one versus 12 packs, one versus 12 or 13 packs, or three versus seven packs found increased continuation of pill use among women provided with more pill packs (*233*–*235*). However, one study found no difference in continuation when patients were provided one and then three packs versus four packs all at once (*236*). In addition to continuation, a greater number of pill packs provided was associated with fewer pregnancy tests, fewer pregnancies, and lower cost per client. However, a greater number of pill packs (13 packs versus three packs) also was associated with increased pill wastage in one study (*234*) (Level of evidence: I to II-2, fair, direct).

Routine Follow-Up After POP Initiation

These recommendations address when routine follow-up is recommended for safe and effective continued use of contraception for healthy women. The recommendations refer to general situations and might vary for different users and different situations. Specific populations who might benefit from more frequent follow-up visits include adolescents, those with certain medical conditions or characteristics, and those with multiple medical conditions.

- Advise the woman to return at any time to discuss side effects or other problems or if she wants to change the method being used. No routine follow-up visit is required.
- At other routine visits, health care providers seeing POP users should do the following:
  - Assess the woman's satisfaction with her contraceptive method and whether she has any concerns about method use.
  - Assess any changes in health status, including medications, that would change the appropriateness of POPs for safe and effective continued use based on U.S. MEC (e.g., category 3 and 4 conditions and characteristics).
  - Consider assessing weight changes and counseling women who are concerned about weight change perceived to be associated with their contraceptive method.

Comments and Evidence Summary. No evidence was found regarding whether a routine follow-up visit after initiating POPs improves correct and continued use.

Missed POPs

For the following recommendations, a dose is considered missed if it has been >3 hours since it should have been taken.

- Take one pill as soon as possible.
- Continue taking pills daily, one each day, at the same time each day, even if it means taking two pills on the same day.
- Use back-up contraception (e.g., condoms) or avoid sexual intercourse until pills have been taken correctly, on time, for 2 consecutive days.

- Emergency contraception should be considered (with the exception of UPA) if the woman has had unprotected sexual intercourse.

Comments and Evidence Summary. Inconsistent or incorrect use of oral contraceptive pills is a major reason for oral contraceptive failure. Unlike COCs, POPs inhibit ovulation in about half of cycles, although this rate varies widely by individual (*279*). Peak serum steroid levels are reached about 2 hours after administration, followed by rapid distribution and elimination, such that by 24 hours after administration, serum steroid levels are near baseline (*279*). Therefore, taking POPs at approximately the same time each day is important. An estimated 48 hours of POP use was deemed necessary to achieve the contraceptive effects on cervical mucus (*279*). Women who frequently miss POPs should consider an alternative contraceptive method that is less dependent on the user to be effective (e.g., IUD, implant, or injectable). No evidence was found regarding the effects of missed POPs available in the United States on measures of contraceptive effectiveness including pregnancy, follicular development, hormone levels, or cervical mucus quality.

Vomiting or Diarrhea (for any Reason or Duration) that Occurs Within 3 Hours After Taking a Pill

- Take another pill as soon as possible (if possible, despite discomfort).
- Continue taking pills daily, one each day, at the same time each day.
- Use back-up contraception (e.g., condoms) or avoid sexual intercourse until 2 days after vomiting or diarrhea has resolved.
- Emergency contraception should be considered (with the exception of UPA) if the woman has had unprotected sexual intercourse.

Comments and Evidence Summary. Theoretically, the contraceptive effectiveness of POPs might be decreased because of vomiting or severe diarrhea. Because of the lack of evidence to address this question, these recommendations are based on the recommendations for missed POPs. No evidence was found regarding the effects of vomiting or diarrhea on measures of contraceptive effectiveness, including pregnancy, follicular development, hormone levels, or cervical mucus quality.

Standard Days Method

SDM is a method based on fertility awareness; users must avoid unprotected sexual intercourse on days 8–19 of the menstrual cycle (*280*). Approximately 5 out of 100 women become pregnant in the first year of use with perfect (i.e., correct and consistent) use of SDM (*280*); effectiveness based on typical use is not available for this method but is expected to be lower than that for perfect use. SDM is reversible and can be used by women of all ages. SDM does not protect against STDs; consistent and correct use of male latex condoms reduces the risk for STDs, including HIV.

Use of SDM Among Women with Various Durations of the Menstrual Cycle

Menstrual Cycles of 26–32 Days

- The woman may use the method.
- Provide a barrier method of contraception for protection on days 8–19 if she wants one.
- If she has unprotected sexual intercourse during days 8–19, consider the use of emergency contraception if appropriate.

Two or More Cycles of <26 or >32 Days Within Any 1 Year of SDM Use

- Advise the woman that the method might not be appropriate for her because of a higher risk for pregnancy. Help her consider another method.

Comments and Evidence Summary. The probability of pregnancy is increased when the menstrual cycle is outside the range of 26–32 days, even if unprotected sexual intercourse is avoided on days 8–19. A study of 7,600 menstrual cycles, including information on cycle length and signs of ovulation, concluded that the theoretical effectiveness of SDM is greatest for women with cycles of 26–32 days, that the method is still effective for women who occasionally have a cycle outside this range, and that it is less effective for women who consistently have cycles outside this range. Information from daily hormonal measurements shows that the timing of the 6-day fertile window varies greatly, even among women with regular cycles (*21*,*281*,*282*).

Emergency Contraception

Emergency contraception consists of methods that can be used by women after sexual intercourse to prevent pregnancy. Emergency contraception methods have varying ranges of effectiveness depending on the method and timing of administration. Four options are available in the United States: the Cu-IUD and three types of ECPs.

Types of Emergency Contraception

Intrauterine Device

- Cu-IUD

ECPs

- UPA in a single dose (30 mg)
- Levonorgestrel in a single dose (1.5 mg) or as a split dose (1 dose of 0.75 mg of levonorgestrel followed by a second dose of 0.75 mg of levonorgestrel 12 hours later)
- Combined estrogen and progestin in 2 doses (Yuzpe regimen: 1 dose of 100 μg of ethinyl estradiol plus 0.50 mg of levonorgestrel followed by a second dose of 100 μg of ethinyl estradiol plus 0.50 mg of levonorgestrel 12 hours later)

Initiation of Emergency Contraception

Timing

Cu-IUD

- The Cu-IUD can be inserted within 5 days of the first act of unprotected sexual intercourse as an emergency contraceptive.
- In addition, when the day of ovulation can be estimated, the Cu-IUD can be inserted beyond 5 days after sexual intercourse, as long as insertion does not occur >5 days after ovulation.

ECPs

- ECPs should be taken as soon as possible within 5 days of unprotected sexual intercourse.

Comments and Evidence Summary. Cu-IUDs are highly effective as emergency contraception (*283*) and can be continued as regular contraception. UPA and levonorgestrel ECPs have similar effectiveness when taken within 3 days after unprotected sexual intercourse; however, UPA has been shown to be more effective than the levonorgestrel formulation 3–5 days after unprotected sexual intercourse (*284*). The combined estrogen and progestin regimen is less effective than UPA or levonorgestrel and also is associated with more frequent occurrence of side effects (nausea and vomiting) (*285*). The levonorgestrel formulation might be less effective than UPA among obese women (*286*).

Two studies of UPA use found consistent decreases in pregnancy rates when administered within 120 hours of unprotected sexual intercourse (*284*,*287*). Five studies found that the levonorgestrel and combined regimens decreased risk for pregnancy through the fifth day after unprotected sexual intercourse; however, rates of pregnancy were slightly higher when ECPs were taken after 3 days (*288*–*292*). A meta-analysis of levonorgestrel ECPs found that pregnancy rates were low when administered within 4 days after unprotected sexual intercourse but increased at 4–5 days (*293*) (Level of evidence: I to II-2, good to poor, direct).

Advance Provision of ECPs

- An advance supply of ECPs may be provided so that ECPs will be available when needed and can be taken as soon as possible after unprotected sexual intercourse.

Comments and Evidence Summary. A systematic review identified 17 studies that reported on safety or effectiveness of advance ECPs in adult or adolescent women (*294*). Any use of ECPs was two to seven times greater among women who received an advance supply of ECPs. However, a summary estimate (relative risk = 0.97; 95% confidence interval = 0.77–1.22) of five randomized controlled trials did not indicate a significant reduction in unintended pregnancies at 12 months with advance provision of ECPs. In the majority of studies among adults or adolescents, patterns of regular contraceptive use, pregnancy rates, and incidence of STDs did not vary between those who received advance ECPs and those who did not. Although available evidence supports the safety of advance provision of ECPs, effectiveness of advance provision of ECPs in reducing pregnancy rates at the population level has not been demonstrated (Level of evidence: I to II-3, good to poor, direct).

Initiation of Regular Contraception After ECPs

UPA

- Advise the woman to start or resume hormonal contraception no sooner than 5 days after use of UPA, and provide or prescribe the regular contraceptive method as needed. For methods requiring a visit to a health care provider, such as DMPA, implants, and IUDs, starting the method at the time of UPA use may be considered; the risk that the regular contraceptive method might decrease the effectiveness of UPA must be weighed against the risk of not starting a regular hormonal contraceptive method.
- The woman needs to abstain from sexual intercourse or use barrier contraception for the next 7 days after starting or resuming regular contraception or until her next menses, whichever comes first.
- Any nonhormonal contraceptive method can be started immediately after the use of UPA.
- Advise the woman to have a pregnancy test if she does not have a withdrawal bleed within 3 weeks.

Levonorgestrel and Combined Estrogen and Progestin ECPs

- Any regular contraceptive method can be started immediately after the use of levonorgestrel or combined estrogen and progestin ECPs.
- The woman needs to abstain from sexual intercourse or use barrier contraception for 7 days.
- Advise the woman to have a pregnancy test if she does not have a withdrawal bleed within 3 weeks.

Comments and Evidence Summary. The resumption or initiation of regular hormonal contraception after ECP use involves consideration of the risk for pregnancy if ECPs fail and the risks for unintended pregnancy if contraception initiation is delayed until the subsequent menstrual cycle. A health care provider may provide or prescribe pills, the patch, or the ring for a woman to start no sooner than 5 days after use of UPA. For methods requiring a visit to a health care provider, such as DMPA, implants, and IUDs, starting the method at the time of UPA use may be considered; the risk that the regular contraceptive method might decrease the effectiveness of UPA must be weighed against the risk of not starting a regular hormonal contraceptive method.

Data on when a woman can start regular contraception after ECPs are limited to pharmacodynamic data and expert opinion (*295*–*297*). In one pharmacodynamic study of women who were randomly assigned to either UPA or placebo groups mid-cycle followed by a 21-day course of combined hormonal contraception found no difference between UPA and placebo groups in the time for women's ovaries to reach quiescence by ultrasound and serum estradiol (*296*); this finding suggests that UPA did not have an effect on the combined hormonal contraception. In another pharmacodynamic study with a crossover design, women were randomly assigned to one of three groups: 1) UPA followed by desogestrel for 20 days started 1 day later; 2) UPA plus placebo; or 3) placebo plus desogestrel for 20 days (*295*). Among women taking UPA followed by desogestrel, a higher incidence of ovulation in the first 5 days was found compared with UPA alone (45% versus 3%, respectively), suggesting desogestrel might decrease the effectiveness of UPA. No concern exists that administering combined estrogen and progestin or levonorgestrel formulations of ECPs concurrently with systemic hormonal contraception decreases the effectiveness of either emergency or regular contraceptive methods because these formulations do not have antiprogestin properties like UPA. If a woman is planning to initiate contraception after the next menstrual bleeding after ECP use, the cycle in which ECPs are used might be shortened, prolonged, or involve unscheduled bleeding.

Prevention and Management of Nausea and Vomiting with ECP Use

Nausea and Vomiting

- Levonorgestrel and UPA ECPs cause less nausea and vomiting than combined estrogen and progestin ECPs.
- Routine use of antiemetics before taking ECPs is not recommended. Pretreatment with antiemetics may be considered depending on availability and clinical judgment.

Vomiting Within 3 Hours of Taking ECPs

- Another dose of ECP should be taken as soon as possible. Use of an antiemetic should be considered.

Comments and Evidence Summary. Many women do not experience nausea or vomiting when taking ECPs, and predicting which women will experience nausea or vomiting is difficult. Although routine use of antiemetics before taking ECPs is not recommended, antiemetics are effective in some women and can be offered when appropriate. Health care providers who are deciding whether to offer antiemetics to women taking ECPs should consider the following: 1) women taking combined estrogen and progestin ECPs are more likely to experience nausea and vomiting than those who take levonorgestrel or UPA ECPs; 2) evidence indicates that antiemetics reduce the occurrence of nausea and vomiting in women taking combined estrogen and progestin ECPs; and 3) women who take antiemetics might experience other side effects from the antiemetics.

A systematic review examined incidence of nausea and vomiting with different ECP regimens and effectiveness of antinausea drugs in reducing nausea and vomiting with ECP use (*298*). The levonorgestrel regimen was associated with significantly less nausea than a nonstandard dose of UPA (50 mg) and the standard combined estrogen and progestin regimen (*299*–*301*). Use of the split-dose levonorgestrel showed no differences in nausea and vomiting compared with the single-dose levonorgestrel (*288*,*290*,*292*,*302*) (Level of evidence: I, good-fair, indirect). Two trials of antinausea drugs, meclizine and metoclopramide, taken before combined estrogen and progestin ECPs, reduced the severity of nausea (*303*,*304*). Significantly less vomiting occurred with meclizine but not metoclopramide (Level of evidence: I, good-fair, direct). No direct evidence was found regarding the effects of vomiting after taking ECPs.

Female Sterilization

Laparoscopic, abdominal, and hysteroscopic methods of female sterilization are available in the United States, and

some of these procedures can be performed in an outpatient procedure or office setting. Fewer than 1 out of 100 women become pregnant in the first year after female sterilization (*14*). Because these methods are intended to be irreversible, all women should be appropriately counseled about the permanency of sterilization and the availability of highly effective, long-acting, reversible methods of contraception. Female sterilization does not protect against STDs; consistent and correct use of male latex condoms reduces the risk for STDs, including HIV.

When Hysteroscopic Sterilization is Reliable for Contraception

- Before a woman can rely on hysteroscopic sterilization for contraception, a hysterosalpingogram (HSG) must be performed 3 months after the sterilization procedure to confirm bilateral tubal occlusion.
- The woman should be advised that she needs to abstain from sexual intercourse or use additional contraceptive protection until she has confirmed bilateral tubal occlusion.

When Laparoscopic and Abdominal Approaches are Reliable for Contraception

- A woman can rely on sterilization for contraception immediately after laparoscopic and abdominal approaches. No additional contraceptive protection is needed.

Comments and Evidence Summary. HSG confirmation is necessary to confirm bilateral tubal occlusion after hysteroscopic sterilization. The inserts for the hysteroscopic sterilization system available in the United States are placed bilaterally into the fallopian tubes and require 3 months for adequate fibrosis and scarring leading to bilateral tubal occlusion. After hysteroscopic sterilization, advise the woman to correctly and consistently use an effective method of contraception while awaiting confirmation. If compliance with another method might be a problem, a woman and her health care provider may consider DMPA injection at the time of sterilization to ensure adequate contraception for 3 months. Unlike laparoscopic and abdominal sterilizations, pregnancy risk beyond 7 years of follow-up has not been studied among women who received hysteroscopic sterilization.

Pregnancy risk with at least 10 years of follow-up has been studied among women who received laparoscopic and abdominal sterilizations (*305*,*306*). Although these methods are highly effective, pregnancies can occur many years after the procedure, and the risk for pregnancy is higher among younger women (*306*,*307*).

A systematic review was conducted to identify studies that reported whether pregnancies occurred after hysteroscopic sterilization (*308*). Twenty-four studies were identified that reported whether pregnancies occurred after hysteroscopic sterilization and found that very few pregnancies occurred among women with confirmed bilateral tubal occlusion; however, few studies include long-term follow-up, and none with follow up for >7 years. Among women who had successful bilateral placement, most pregnancies that occurred after hysteroscopic sterilization were in women who did not have confirmed bilateral tubal occlusion at 3 months, either because of lack of follow up or misinterpretation of HSG results (*309–311*). Some pregnancies occurred within 3 months of placement, including among women who were already pregnant at the time of the procedure, women who did not use alternative contraception, or women who had failures of alternative contraception (*310–315*). Although these studies generally demonstrated high rates of bilateral placement, some pregnancies occurred as a result of lack of bilateral placement identified on later imaging (*310*,*311*,*313–316*). Most pregnancies occurred after deviations from FDA directions, which include placement in the early follicular phase of the menstrual cycle, imaging at 3 months to document proper placement, and use of effective alternative contraception until documented occlusion (Level of evidence: II-3, fair, direct).

Male Sterilization

Male sterilization, or vasectomy, is one of the few contraceptive methods available to men and can be performed in an outpatient procedure or office setting. Fewer than 1 woman out of 100 becomes pregnant in the first year after her male partner undergoes sterilization (*14*). Because male sterilization is intended to be irreversible, all men should be appropriately counseled about the permanency of sterilization and the availability of highly effective, long-acting, reversible methods of contraception for women. Male sterilization does not protect against STDs; consistent and correct use of male latex condoms reduces the risk for STDs, including HIV.

When Vasectomy is Reliable for Contraception

- A semen analysis should be performed 8–16 weeks after a vasectomy to ensure the procedure was successful.
- The man should be advised that he should use additional contraceptive protection or abstain from sexual intercourse until he has confirmation of vasectomy success by postvasectomy semen analysis.

Other Postprocedure Recommendations

- The man should refrain from ejaculation for approximately 1 week after the vasectomy to allow for healing of surgical sites and, after certain methods of vasectomy, occlusion of the vas.

Comments and Evidence Summary. The Vasectomy Guideline Panel of the American Urological Association performed a systematic review of key issues concerning the practice of vasectomy (*317*). All English-language publications on vasectomy published during 1949–2011 were reviewed. For more information, see the American Urological Association *Vasectomy Guidelines* (https://www.auanet.org/common/pdf/education/clinical-guidance/Vasectomy.pdf).

Motile sperm disappear within a few weeks after vasectomy (*318–321*). The time to azoospermia varies widely in different studies; however, by 12 weeks after the vasectomy, 80% of men have azoospermia, and almost all others have rare nonmotile sperm (defined as ≤100,000 nonmotile sperm per milliliter) (*317*). The number of ejaculations after vasectomy is not a reliable indicator of when azoospermia or rare nonmotile sperm will be achieved (*317*). Once azoospermia or rare nonmotile sperm has been achieved, patients can rely on the vasectomy for contraception, although not with 100% certainty. The risk for pregnancy after a man has achieved postvasectomy azoospermia is approximately one in 2,000 (*322–326*).

A median of 78% (range 33%–100%) of men return for a single postvasectomy semen analysis (*317*). In the largest cohorts that appear typical of North American vasectomy practice, approximately two thirds of men (55%–71%) return for at least one postvasectomy semen analysis (*322,327–331*). Assigning men an appointment after their vasectomy might improve compliance with follow-up (*332*).

When Women Can Stop Using Contraceptives

- Contraceptive protection is still needed for women aged >44 years if the woman wants to avoid pregnancy.

Comments and Evidence Summary. The age at which a woman is no longer at risk for pregnancy is not known. Although uncommon, spontaneous pregnancies occur among women aged >44 years. Both the American College of Obstetricians and Gynecologists and the North American Menopause Society recommend that women continue contraceptive use until menopause or age 50–55 years (*333,334*). The median age of menopause is approximately 51 years in North America (*333*) but can vary from ages 40–60 years (*335*). The median age of definitive loss of natural fertility is 41 years but can range up to age 51 years (*336,337*). No reliable laboratory tests are available to confirm definitive loss of fertility in a woman. The assessment of follicle-stimulating hormone levels to determine when a woman is no longer fertile might not be accurate (*333*).

Health care providers should consider the risks for becoming pregnant in a woman of advanced reproductive age, as well as any risks of continuing contraception until menopause. Pregnancies among women of advanced reproductive age are at higher risk for maternal complications, such as hemorrhage, venous thromboembolism, and death, and fetal complications, such as spontaneous abortion, stillbirth, and congenital anomalies (*338–340*). Risks associated with continuing contraception, in particular risks for acute cardiovascular events (venous thromboembolism, myocardial infarction, or stroke) or breast cancer, also are important to consider. U.S. MEC states that on the basis of age alone, women aged >45 years can use POPs, implants, the LNG-IUD, or the Cu- IUD (U.S. MEC 1) (*5*). Women aged >45 years generally can use combined hormonal contraceptives and DMPA (U.S. MEC 2) (*5*). However, women in this age group might have chronic conditions or other risk factors that might render use of hormonal contraceptive methods unsafe; U.S. MEC might be helpful in guiding the safe use of contraceptives in these women.

In two studies, the incidence of venous thromboembolism was higher among oral contraceptive users aged ≥45 years compared with younger oral contraceptive users (*341–343*); however, an interaction between hormonal contraception and increased age compared with baseline risk was not demonstrated (*341,342*) or was not examined (*343*). The relative risk for myocardial infarction was higher among all oral contraceptive users than in nonusers, although a trend of increased relative risk with increasing age was not demonstrated (*344,345*). No studies were found regarding the risk for stroke in COC users aged ≥45 years (Level of evidence: II-2, good to poor, direct).

A pooled analysis by the Collaborative Group on Hormonal Factors and Breast Cancer in 1996 (*346*) found small increased relative risks for breast cancer among women aged ≥45 years whose last use of combined hormonal contraceptives was <5 years previously and for those whose last use was 5–9 years previously. Seven more recent studies suggested small but nonsignificant increased relative risks for breast carcinoma in situ or breast cancer among women who had used oral contraceptives or DMPA when they were aged ≥40 years compared with those who had never used either method (*347–353*) (Level of evidence: II-2, fair, direct).

Conclusion

Most women can start most contraceptive methods at any time, and few examinations or tests, if any, are needed before starting a contraceptive method. Routine follow-up for most women includes assessment of her satisfaction with the contraceptive method, concerns about method use, and changes in health status or medications that could affect medical eligibility for continued use of the method. Because changes in bleeding patterns are one of the major reasons for discontinuation of contraception, recommendations are provided for the management of bleeding irregularities with various contraceptive methods. In addition, because women and health care providers can be confused about the procedures for missed pills and dosing errors with the contraceptive patch and ring, the instructions are streamlined for easier use. ECPs and emergency use of the Cu-IUD are important options for women, and recommendations on using these methods, as well as starting regular contraception after use of emergency contraception, are provided. Male and female sterilization are highly effective methods of contraception for men, women, and couples who have completed childbearing; for men undergoing vasectomy and women undergoing a hysteroscopic sterilization procedure, additional contraceptive protection is needed until the success of the procedure can be confirmed.

CDC is committed to working with partners at the federal, national, and local levels to disseminate, implement, and evaluate U.S. SPR recommendations so that the information reaches health care providers. Strategies for dissemination and implementation include collaborating with other federal agencies and professional and service organizations to widely distribute the recommendations through presentations, electronic distribution, newsletters, and other publications; development of provider tools and job aids to assist providers in implementing the new recommendations; and training activities for students, as well as for continuing education. CDC conducts surveys of family planning health care providers to assess attitudes and practices related to contraceptive use. Results from these surveys will assist CDC in evaluating the impact of these recommendations on the provision of contraceptives in the United States. Finally, CDC will continually monitor new scientific evidence and will update these recommendations as warranted by new evidence. Updates to the recommendations, as well as provider tools and other resources, are available on the CDC U.S. SPR website: http://www.cdc.gov/reproductivehealth/UnintendedPregnancy/USSPR.htm.

Acknowledgments

This report is based, in part, on the work of the Promoting Family Planning Team, Department of Reproductive Health and Research, World Health Organization, and its development of the *Selected Practice Recommendations for Contraceptive Use, 3rd edition*.

U.S. Selected Practice Recommendations for Contraceptive Use Participants

CDC Guideline Development Group for *U.S. Medical Eligibility Criteria for Contraceptive Use* and *U.S. Selected Practice Recommendations for Contraceptive Use*

Kathryn M. Curtis, PhD (Chair), Erin Berry-Bibee, MD, Suzanne G. Folger, PhD, Leah G. Horton, MSPH, Denise J. Jamieson, MD, Tara C. Jatlaoui, MD, Polly A. Marchbanks, PhD, H. Pamela Pagano, MPH, Halley E.M. Riley, MPH, Mirelys Rodriguez, Katharine B. Simmons, MD, Naomi K. Tepper, MD, Maura K. Whiteman, PhD, Lauren B. Zapata, PhD, CDC, Atlanta, Georgia.

Invited Meeting Participants
August 27–28, 2014, Atlanta, Georgia

Herbert Peterson, MD, University of North Carolina, Chapel Hill, North Carolina (Chair); Gale Burstein, MD, Erie County Department of Health, Buffalo, New York; Alison Edelman, MD, Oregon Health and Science University, Portland, Oregon; Eve Espey, MD, University of New Mexico, Albuquerque, New Mexico; Emily Godfrey, MD, University of Washington, Seattle, WA; Andrew Kaunitz, MD, University of Florida, Jacksonville, Florida; Susan Moskosky, MS, U.S. Department of Health and Human Services, Rockville, Maryland; Kavita Nanda, MD, FHI360 and American College of Obstetricians and Gynecologists, Durham, NC; Deborah Nucatola, MD, Planned Parenthood Federation of America, New York, New York; Michael Policar, MD, University of California, San Francisco, California; Carolyn Westhoff, MD, Columbia University, New York, New York.

Systematic Review Presenters and Authors Meeting, August 26–28, 2015, Atlanta, Georgia

Kathryn M. Curtis, PhD, Tara C. Jatlaoui, MD, Polly A. Marchbanks, PhD, Lauren B. Zapata, PhD, CDC, Atlanta, Georgia; Nathalie Kapp, MD, HRA Pharma, Paris, France.

Invited Meeting Participants
August 26–28, 2015, Atlanta, Georgia

Herbert Peterson, MD, University of North Carolina, Chapel Hill, North Carolina (Chair); Rebecca Allen, MD, American Society for Reproductive Medicine and Women and Infants Hospital, Providence, Rhode Island; Abbey Berenson, MD, University of Texas Medical Branch, Galveston, Texas; Amanda Black, MD, University of Ottawa, Ontario, Canada; Cora Collette Breuner, MD, American Academy of Pediatrics and Seattle Children's Hospital, Seattle, Washington; Gale Burstein, MD, Erie County Department of Health, Buffalo, New York; Alicia Christy, MD, National Institutes of Health, Rockville, Maryland; Mitchell D. Creinin, MD, University of California, Davis, California; Linda Dominguez, Southwest Women's Health, Albuquerque, New Mexico; Alison Edelman, MD, Oregon Health and Science University, Portland, Oregon; Eve Espey, MD, University of New Mexico, Albuquerque, New Mexico; Emily Godfrey, MD, University of Washington, Seattle, WA; Marji Gold, MD, Albert Einstein College of Medicine, Bronx, New York; Robert Hatcher, MD, Emory University, Atlanta, Georgia; Mark Hathaway, MD, JHPIEGO and Unity Healthcare, Washington, DC; Stephen Heartwell, MD, Susan Thompson Buffett Foundation, Omaha, Nebraska; Paula Hillard, MD, Stanford University, Palo Alto, California; Andrew Kaunitz, MD, University of Florida, Jacksonville, Florida; Reagan McDonald-Mosley, MD, Planned Parenthood Federation of America, New York, New York; Susan Moskosky, MS, U.S. Department of Health and Human Services, Rockville, Maryland; Kavita Nanda, MD, FHI360, Durham, North Carolina; Jeffrey Peipert, MD, Washington University, St. Louis, Missouri; Michael Policar, MD, University of California, San Francisco, California; Sarah Prager, MD, University of Washington, Seattle Washington; David Soper, MD, Medical University of South Carolina, Charleston, South Carolina; Lisa Soule, MD, Food and Drug Administration, Rockville, Maryland; James Trussell, PhD, Princeton University, Princeton, New Jersey; Carolyn Westhoff, MD, Columbia University, New York, New York; Christopher Zahn, MD, American College of Obstetricians and Gynecologists, Washington, DC.

CDC Attendees

Wanda D. Barfield, MD, Peter Briss, MD, W. Craig Hooper, PhD, Jill Huppert, MD, Caroline King, PhD, Michele Mandel, Titilope Oduyebo, MD, Cria Perrine, MD, Sam Posner, PhD.

External Reviewers

Claude Burnett, MD, Northeast Health District, Georgia Department of Public Health, Athens, Georgia; Carrie Cwiak, MD, Emory University, Atlanta, Georgia; David Klein, MD, Uniformed Services University, Bethesda, Maryland; Seema Menon, MD, Medical College of Wisconsin, Milwaukee, Wisconsin.

Conflicts of Interest for Invited Meeting Participants
August 26–28, 2015, Atlanta, Georgia

Rebecca Allen, Nexplanon trainer for Merck and Liletta trainer for Actavis, consultant, advisory board, and education grant from Bayer; Mitchell D. Creinin, Nexplanon trainer for Merck, litigation consultant for Bayer, advisory board for Merck and Teva Pharmaceutical Industries Ltd., consultant for Lemonaid — PolkaDoc app, research support to University of California, Davis from Medicines360, Contramed, Merck, Eunice Kennedy Shriver National Institute of Child Health and Human Development, and Society of Family Planning; Linda Dominguez, speaker for Bayer, Merck, and Actavis; Alison Edelman, royalties from Up to Date, Inc., consultant for Genzyme, grant support from National Institutes of Health and Gates Foundation, travel funds from World Health Organization, grant support and honorarium from Society of Family Planning, honorarium and travel funds from Contemporary Forum, trainer for Merck, consultant for Gynuity Health Projects, honorarium from CDC, Projects In Knowledge, and American Congress of Obstetricians and Gynecologists, advisory board for Agile Therapeutics; Eve Espey, travel funds from the American Congress of Obstetricians and Gynecologists, Society for Family Planning, and U.S. Food and Drug Administration, Reproductive and Drug Advisory Committee for U.S. Food and Drug Administration, travel funds and honoraria from Wayne State University, Telluride Conference, New Mexico Department of Health Clinician Conference, Planned Parenthood National Medical Conference and Society of Family Planning, British Columbia Contraception Access Research Team Conference, and American Congress of Obstetricians and Gynecologists annual meeting; Emily Godfrey, research funding from Bayer Women's Health, Prima-Temp, and Teva Pharmaceutical Industries Ltd., trainer for Merck and Upstream USA, grant reviewer for Fellowship of Family Planning and Society of Family Planning Research Fund; Mark Hathaway, Liletta trainer and speaker for Actavis and Medicines360, Nexplanon trainer for Merck, advisory board for Contramed LLC and Afaxys Pharmaceuticals; Paula Hillard consultant for American Civil Liberties Union, Advanced Health Media, CMEology, National Sleep Foundation, and Planned Parenthood Federation of America, honoraria from National Sleep Foundation, Dignity Health, CMEology, Advance Health Media, and Medscape, editorial board for Advanstar — Contemporary OB/GYN, board examiner for the American Board of Obstetrics and Gynecology, contract reviewer for the Department of Health and Human Services, editorial board for EBSCO — PEMSoft, Nexplanon trainer for Merck, scientific advisor to Proctor and Gamble, publication royalties from Wiley Blackwell Publishing; Nathalie Kapp, employee of HRA Pharma; Andrew Kaunitz, advisory board participant of Allergan, Bayer, Merck, and Pfizer, clinical trial funding to University of Florida from Agile Therapeutics, Bayer, Merck; Jeffrey Peipert, research funding from Bayer and Teva Pharmaceutical Industries Ltd., advisory board for Perrigo; Michael Policar, litigation consultant for Bayer; James Trussell, advisory board for Merck and Teva Pharmaceutical Industries Ltd., consultant for Bayer; Carolyn Westhoff, data and safety monitoring board for Merck and Bayer, advisory board for Agile Therapeutics, MicroChips Biotech, and Actavis, research support to Columbia University from Medicines360, León Farma, and ContraMed.

Handling Conflict of Interest

To promote transparency, all participants were asked to disclose any potential conflicts of interest to CDC prior to the expert meeting and to report any potential conflicts of interest during the introductory portion of the expert meeting. All potential conflicts of interest are listed above. No participants were excluded from discussion based on potential conflicts of interest. One presenter was an employee of a pharmaceutical company and participated by teleconference; after the presentation and questions related to the presentation, the presenter was excused from the discussion. CDC staff who ultimately decided and developed these recommendations have no financial interests or other relationships with the manufacturers of commercial products, suppliers of commercial services, or commercial supporters relevant to these recommendations.

References

1. Finer LB, Zolna MR. Declines in unintended pregnancy in the United States, 2008–2011. N Engl J Med 2016;374:843–52. http://dx.doi.org/10.1056/NEJMsa1506575
2. Gipson JD, Koenig MA, Hindin MJ. The effects of unintended pregnancy on infant, child, and parental health: a review of the literature. Stud Fam Plann 2008;39:18–38. http://dx.doi.org/10.1111/j.1728-4465.2008.00148.x
3. Sonfield A, Kost K. Public costs from unintended pregnancies and the role of public insurance programs in paying for pregnancy-related care: national and state estimates for 2010. New York, NY: The Guttmacher Institute; 2015.
4. Finer LB, Henshaw SK. Disparities in rates of unintended pregnancy in the United States, 1994 and 2001. Perspect Sex Reprod Health 2006;38:90–6. http://dx.doi.org/10.1363/3809006
5. CDC. U.S. medical eligibility criteria for contraceptive use, 2016. MMWR Recomm Rep (No. RR-3);2016.
6. CDC. U.S. selected practice recommendations for contraceptive use, 2013: adapted from the World Health Organization selected practice recommendations for contraceptive use, 2nd edition. MMWR Recomm Rep 2013;62(No. RR-5).
7. Mohllajee AP, Curtis KM, Flanagan RG, Rinehart W, Gaffield ML, Peterson HB. Keeping up with evidence: a new system for WHO's evidence-based family planning guidance. Am J Prev Med 2005;28:483–90. http://dx.doi.org/10.1016/j.amepre.2005.02.008
8. Liberati A, Altman DG, Tetzlaff J, et al. The PRISMA statement for reporting systematic reviews and meta-analyses of studies that evaluate health care interventions: explanation and elaboration. J Clin Epidemiol 2009;62:e1–34. http://dx.doi.org/10.1016/j.jclinepi.2009.06.006
9. Moher D, Liberati A, Tetzlaff J, Altman DG; PRISMA Group. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. BMJ 2009;339:b2535.
10. Harris RP, Helfand M, Woolf SH, et al; Methods Work Group, Third US Preventive Services Task Force. Current methods of the U.S. Preventive Services Task Force: a review of the process. Am J Prev Med 2001;20(Suppl):21–35. http://dx.doi.org/10.1016/S0749-3797(01)00261-6
11. Horton LG, Folger SG, Berry-Bibee E, Jatlaoui TC, Tepper NK, Curtis KM. Research gaps from evidence-based contraceptives guidance: the U.S. Medical Eligibility Criteria for Contraceptive Use, 2016, and the U.S. Selected Practice Recommendations for Contraceptive Use, 2016. Contraception. In press 2016.
12. Gavin L, Moskosky S, Carter M, et al. Providing quality family planning services: Recommendations of CDC and the U.S. Office of Population Affairs. MMWR Recomm Rep 2014;63(No. RR-4).
13. World Health Organization. Selected practice recommendations for contraceptive use. 2nd ed. Geneva, Switzerland: WHO Press; 2004.
14. Trussell J. Contraceptive failure in the United States. Contraception 2011;83:397–404. http://dx.doi.org/10.1016/j.contraception.2011.01.021
15. Workowski KA, Bolan GA. Sexually transmitted diseases treatment guidelines, 2015. MMWR Recomm Rep 2015;64(No. RR-3).
16. Stanback J, Nakintu N, Qureshi Z, Nasution M. Does assessment of signs and symptoms add to the predictive value of an algorithm to rule out pregnancy? J Fam Plann Reprod Health Care 2006;32:27–9. http://dx.doi.org/10.1783/147118906775275370
17. Stanback J, Nanda K, Ramirez Y, Rountree W, Cameron SB. Validation of a job aid to rule out pregnancy among family planning clients in Nicaragua. Rev Panam Salud Publica 2008;23:116–8. http://dx.doi.org/10.1590/S1020-49892008000200007
18. Stanback J, Qureshi Z, Sekadde-Kigondu C, Gonzalez B, Nutley T. Checklist for ruling out pregnancy among family-planning clients in primary care. Lancet 1999;354:566. http://dx.doi.org/10.1016/S0140-6736(99)01578-0
19. Torpey K, Mwenda L, Kabaso M, et al. Excluding pregnancy among women initiating antiretroviral therapy: efficacy of a family planning job aid. BMC Public Health 2010;10:249. http://dx.doi.org/10.1186/1471-2458-10-249
20. Cole LA, Ladner DG, Byrn FW. The normal variabilities of the menstrual cycle. Fertil Steril 2009;91:522–7. http://dx.doi.org/10.1016/j.fertnstert.2007.11.073
21. Wilcox AJ, Dunson D, Baird DD. The timing of the "fertile window" in the menstrual cycle: day specific estimates from a prospective study. BMJ 2000;321:1259–62. http://dx.doi.org/10.1136/bmj.321.7271.1259
22. Donnet ML, Howie PW, Marnie M, Cooper W, Lewis M. Return of ovarian function following spontaneous abortion. Clin Endocrinol (Oxf) 1990;33:13–20. http://dx.doi.org/10.1111/j.1365-2265.1990.tb00460.x
23. Lähteenmäki P. Postabortal contraception. Ann Med 1993;25:185–9. http://dx.doi.org/10.3109/07853899309164166
24. Stoddard A, Eisenberg DL. Controversies in family planning: timing of ovulation after abortion and the conundrum of postabortion intrauterine device insertion. Contraception 2011;84:119–21. http://dx.doi.org/10.1016/j.contraception.2010.12.010
25. Jackson E, Glasier A. Return of ovulation and menses in postpartum nonlactating women: a systematic review. Obstet Gynecol 2011;117:657–62. http://dx.doi.org/10.1097/AOG.0b013e31820ce18c
26. Kennedy KI, Rivera R, McNeilly AS. Consensus statement on the use of breastfeeding as a family planning method. Contraception 1989;39:477–96. http://dx.doi.org/10.1016/0010-7824(89)90103-0
27. Labbok MH, Perez A, Valdes V, et al. The lactational amenorrhea method (LAM): a postpartum introductory family planning method with policy and program implications. Adv Contracept 1994;10:93–109. http://dx.doi.org/10.1007/BF01978103
28. US Food and Drug Administration. 510(k) premarket notification. Silver Spring, MD; 2015. https://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfpmn/pmn.cfm
29. Cervinski MA, Gronowski AM. Reproductive-endocrine point-of-care testing: current status and limitations. Clin Chem Lab Med 2010;48:935–42. http://dx.doi.org/10.1515/CCLM.2010.183
30. Cole LA. Human chorionic gonadotropin tests. Expert Rev Mol Diagn 2009;9:721–47. http://dx.doi.org/10.1586/erm.09.51
31. Eichner SF, Timpe EM. Urinary-based ovulation and pregnancy: point-of-care testing. Ann Pharmacother 2004;38:325–31. http://dx.doi.org/10.1345/aph.1D210
32. Wilcox AJ, Baird DD, Dunson D, McChesney R, Weinberg CR. Natural limits of pregnancy testing in relation to the expected menstrual period. JAMA 2001;286:1759–61. http://dx.doi.org/10.1001/jama.286.14.1759
33. Korhonen J, Alfthan H, Ylöstalo P, Veldhuis J, Stenman UH. Disappearance of human chorionic gonadotropin and its alpha- and beta-subunits after term pregnancy. Clin Chem 1997;43:2155–63.

34. Reyes FI, Winter JS, Faiman C. Postpartum disappearance of chorionic gonadotropin from the maternal and neonatal circulations. Am J Obstet Gynecol 1985;153:486–9. http://dx.doi.org/10.1016/0002-9378(85)90458-2
35. Steier JA, Bergsjø P, Myking OL. Human chorionic gonadotropin in maternal plasma after induced abortion, spontaneous abortion, and removed ectopic pregnancy. Obstet Gynecol 1984;64:391–4.
36. Bracken MB. Oral contraception and congenital malformations in offspring: a review and meta-analysis of the prospective studies. Obstet Gynecol 1990;76:552–7.
37. Gray RH, Pardthaisong T. In utero exposure to steroid contraceptives and survival during infancy. Am J Epidemiol 1991;134:804–11.
38. Pardthaisong T, Gray RH. In utero exposure to steroid contraceptives and outcome of pregnancy. Am J Epidemiol 1991;134:795–803.
39. Jaffe B, Harlap S, Baras M, et al. Long-term effects of MPA on human progeny: intellectual development. Contraception 1988;37:607–19. http://dx.doi.org/10.1016/0010-7824(88)90007-8
40. Pardthaisong T, Yenchit C, Gray R. The long-term growth and development of children exposed to Depo-Provera during pregnancy or lactation. Contraception 1992;45:313–24. http://dx.doi.org/10.1016/0010-7824(92)90053-V
41. Brahmi D, Steenland MW, Renner RM, Gaffield ME, Curtis KM. Pregnancy outcomes with an IUD in situ: a systematic review. Contraception 2012;85:131–9. http://dx.doi.org/10.1016/j.contraception.2011.06.010
42. Tepper NK, Marchbanks PA, Curtis KM. Use of a checklist to rule out pregnancy: a systematic review. Contraception 2013;87:661–5. http://dx.doi.org/10.1016/j.contraception.2012.08.007
43. Whiteman MK, Tyler CP, Folger SG, Gaffield ME, Curtis KM. When can a woman have an intrauterine device inserted? A systematic review. Contraception 2013;87:666–73. http://dx.doi.org/10.1016/j.contraception.2012.08.015
44. Teva Women's Health Inc. ParaGard T 380a intrauterine copper contraceptive [Prescribing information]. Sellersville, PA; 2013. http://paragard.com/Pdf/ParaGard-PI.pdf
45. Bayer HealthCare Pharmaceuticals Inc. Mirena (levonorgestrel-releasing intrauterine system) [Prescribing information]. Whippany, NJ; 2014. http://labeling.bayerhealthcare.com/html/products/pi/Mirena_PI. pdf
46. Jatlaoui TC, Simmons KB, Curtis KM. The safety of intrauterine contraception initiation among women with current asymptomatic cervical infections or at increased risk of sexually transmitted infections. Contraception 2016. Epub June 1, 2016. http://dx.doi.org/10.1016/j.contraception.2016.05.013
47. Avonts D, Sercu M, Heyerick P, Vandermeeren I, Meheus A, Piot P. Incidence of uncomplicated genital infections in women using oral contraception or an intrauterine device: a prospective study. Sex Transm Dis 1990;17:23–9. http://dx.doi.org/10.1097/00007435-199017010-00006
48. Birgisson NE, Zhao Q, Secura GM, Madden T, Peipert JF. Positive testing for Neisseria gonorrhoeae and Chlamydia trachomatis and the risk of pelvic inflammatory disease in IUD users. J Womens Health (Larchmt) 2015;24:354–9. http://dx.doi.org/10.1089/jwh.2015.5190
49. Campbell SJ, Cropsey KL, Matthews CA. Intrauterine device use in a high-risk population: experience from an urban university clinic. Am J Obstet Gynecol 2007;197: 193.e1–6; discussion 193.e6–7.
50. Cropsey KL, Matthews C, Campbel S, Ivey S, Adawadkar S. Long-term, reversible contraception use among high-risk women treated in a university-based gynecology clinic: comparison between IUD and Depo-Provera. J Womens Health (Larchmt) 2010;19:349–53. http://dx.doi.org/10.1089/jwh.2009.1518
51. Farley TMM, Rosenberg MJ, Rowe PJ, Chen JH, Meirik O. Intrauterine devices and pelvic inflammatory disease: an international perspective. Lancet 1992;339:785–8. http://dx.doi.org/10.1016/0140-6736(92)91904-M
52. Grimes DA, Schulz KF. Antibiotic prophylaxis for intrauterine contraceptive device insertion. Cochrane Database Syst Rev 2001;(2):CD001327.
53. LeFevre ML; US Preventive Services Task Force. Screening for chlamydia and gonorrhea: U.S. Preventive Services Task Force recommendation statement. Ann Intern Med 2014;161:902–10. http://dx.doi.org/10.7326/M14-1981
54. Mohllajee AP, Curtis KM, Peterson HB. Does insertion and use of an intrauterine device increase the risk of pelvic inflammatory disease among women with sexually transmitted infection? A systematic review. Contraception 2006;73:145–53. http://dx.doi.org/10.1016/j.contraception.2005.08.007
55. Morrison CS, Turner AN, Jones LB. Highly effective contraception and acquisition of HIV and other sexually transmitted infections. Best Pract Res Clin Obstet Gynaecol 2009;23:263–84. http://dx.doi.org/10.1016/j.bpobgyn.2008.11.004
56. Sufrin CB, Postlethwaite D, Armstrong MA, Merchant M, Wendt JM, Steinauer JE. Neisseria gonorrhea and Chlamydia trachomatis screening at intrauterine device insertion and pelvic inflammatory disease. Obstet Gynecol 2012;120:1314–21.
57. Tepper NK, Steenland MW, Marchbanks PA, Curtis KM. Laboratory screening prior to initiating contraception: a systematic review. Contraception 2013;87:645–9. http://dx.doi.org/10.1016/j.contraception.2012.08.009
58. Tepper NK, Steenland MW, Marchbanks PA, Curtis KM. Hemoglobin measurement prior to initiating copper intrauterine devices: a systematic review. Contraception 2013;87:639–44. http://dx.doi.org/10.1016/j.contraception.2012.08.008
59. Rivera R, Almonte H, Arreola M, et al. The effects of three different regimens of oral contraceptives and three different intrauterine devices on the levels of hemoglobin, serum iron and iron binding capacity in anemic women. Contraception 1983;27:311–27. http://dx.doi.org/10.1016/0010-7824(83)90009-4
60. Ursin G, Ross RK, Sullivan-Halley J, Hanisch R, Henderson B, Bernstein L. Use of oral contraceptives and risk of breast cancer in young women. Breast Cancer Res Treat 1998;50:175–84. http://dx.doi.org/10.1023/A:1006037823178
61. Hassan EO, el-Husseini M, el-Nahal N. The effect of 1-year use of the CuT 380A and oral contraceptive pills on hemoglobin and ferritin levels. Contraception 1999;60:101–5. http://dx.doi.org/10.1016/S0010-7824(99)00065-7
62. Calzolari E, Guglielmo R, Viola F, Migliore L. Hematological parameters and iron therapy in women with IUDs. Experimental study [Italian]. Minerva Ginecol 1981;33:355–62.
63. Andersson K, Odlind V, Rybo G. Levonorgestrel-releasing and copper-releasing (Nova T) IUDs during five years of use: a randomized comparative trial. Contraception 1994;49:56–72. http://dx.doi.org/10.1016/0010-7824(94)90109-0
64. Andrade AT, Pizarro E, Shaw STJ Jr, Souza JP, Belsey EM, Rowe PJ; World Health Organization. Consequences of uterine blood loss caused by various intrauterine contraceptive devices in South American women. Contraception 1988;38:1–18. http://dx.doi.org/10.1016/0010-7824(88)90091-1
65. Blum M, Ariel J, Zacharowitch D. Ferritin, a faithful reflection of iron deficiency in IUD wearers with mild vaginal spotting. Adv Contracept 1991;7:39–42. http://dx.doi.org/10.1007/BF01850717
66. El-sheikha Z, Hamza A, Mahmoud M. Menstrual blood loss of TCu-380 A and TCu-200 B IUDs. Popul Sci 1990;July:55–62.
67. Gallegost AJ, Aznar R, Merino G, Guizer E. Intrauterine devices and menstrual blood loss. A comparative study of eight devices during the first six months of use. Contraception 1978;17:153–61. http://dx.doi.org/10.1016/0010-7824(78)90071-9
68. Gao J, Zeng S, Sun BL, et al. Menstrual blood loss, haemoglobin and ferritin concentration of Beijing women wearing steel ring, VCu 200, and TCu 220c IUDs. Contraception 1986;34:559–71. http://dx.doi.org/10.1016/S0010-7824(86)80012-9

69. Goh TH, Hariharan M. Effect of laparoscopic sterilization and insertion of Multiload Cu 250 and Progestasert IUDs on serum ferritin levels. Contraception 1983;28:329–36. http://dx.doi.org/10.1016/0010-7824(83)90034-3
70. Goh TH, Hariharan M, Tan CH. A longitudinal study of serum iron indices and haemoglobin concentration following copper-IUD insertion. Contraception 1980;22:389–95. http://dx.doi.org/10.1016/0010-7824(80)90024-4
71. Guillebaud J, Bonnar J, Morehead J, Matthews A. Menstrual blood-loss with intrauterine devices. Lancet 1976;1:387–90. http://dx.doi.org/10.1016/S0140-6736(76)90216-6
72. Haugan T, Skjeldestad FE, Halvorsen LE, Kahn H. A randomized trial on the clinical performance of Nova T380 and Gyne T380 Slimline copper IUDs. Contraception 2007;75:171–6. http://dx.doi.org/10.1016/j.contraception.2006.09.005
73. Kivijärvi A, Timonen H, Rajamäki A, Grönroos M. Iron deficiency in women using modern copper intrauterine devices. Obstet Gynecol 1986;67:95–8.
74. Larsson B, Hamberger L, Rybo G. Influence of copper intrauterine contraceptive devices (Cu-7-IUD) on the menstrual blood-loss. Acta Obstet Gynecol Scand 1975;54:315–8. http://dx.doi.org/10.3109/00016347509156760
75. Larsson G, Milsom I, Jonasson K, Lindstedt G, Rybo G. The long-term effects of copper surface area on menstrual blood loss and iron status in women fitted with an IUD. Contraception 1993;48:471–80. http://dx.doi.org/10.1016/0010-7824(93)90136-U
76. Malmqvist R, Petersohn L, Bengtsson LP. Menstrual bleeding with copper-covered intrauterine contraceptive devices. Contraception 1974;9:627–33. http://dx.doi.org/10.1016/0010-7824(74)90048-1
77. Milsom I, Rybo G, Lindstedt G. The influence of copper surface area on menstrual blood loss and iron status in women fitted with an IUD. Contraception 1990;41:271–81. http://dx.doi.org/10.1016/0010-7824(90)90068-7
78. Piedras J, Córdova MS, Pérez-Toral MC, Lince E, Garza-Flores J. Predictive value of serum ferritin in anemia development after insertion of T Cu 220 intrauterine device. Contraception 1983;27:289–97. http://dx.doi.org/10.1016/0010-7824(83)90007-0
79. Sivin I, Alvarez F, Diaz J, et al. Intrauterine contraception with copper and with levonorgestrel: a randomized study of the TCu 380Ag and levonorgestrel 20 mcg/day devices. Contraception 1984;30:443–56. http://dx.doi.org/10.1016/0010-7824(84)90036-2
80. Sivin I, Stern J, Diaz J, et al. Two years of intrauterine contraception with levonorgestrel and with copper: a randomized comparison of the TCu 380Ag and levonorgestrel 20 mcg/day devices. Contraception 1987;35:245–55. http://dx.doi.org/10.1016/0010-7824(87)90026-6
81. Tchai BS, Kim SW, Han JH, Im MW. Menstrual blood loss, iron nutriture, and the effects of Alza-T IPCS 52, T-Cu 220C and Lippes Loop D in Korean women. Seoul J Med 1987;28:51–9.
82. Wright EA, Kapu MM, Isichei UP. Zinc depletion and menorrhagia in Nigerians using copper T-200 intrauterine device. Trace Elem Med 1989;6:147–9.
83. Milsom I, Andersson K, Jonasson K, Lindstedt G, Rybo G. The influence of the Gyne-T 380S IUD on menstrual blood loss and iron status. Contraception 1995;52:175–9. http://dx.doi.org/10.1016/0010-7824(95)00163-5
84. CDC. Safe motherhood at a glance: Hyattsville, MD: CDC; 2015. http://www.cdc.gov/chronicdisease/resources/publications/aag/pdf/2015/safe-motherhood-aag-2015.pdf
85. Cox S, Dietz P, Bowman B, Posner S. Prevalence of chronic conditions among women of reproductive age. Third National Summit on Preconception Health and Health Care: Tampa, FL; 2011.
86. Berenson AB, Rahman M, Wilkinson G. Effect of injectable and oral contraceptives on serum lipids. Obstet Gynecol 2009;114:786–94. http://dx.doi.org/10.1097/AOG.0b013e3181b76bea
87. Dilbaz B, Ozdegirmenci O, Caliskan E, Dilbaz S, Haberal A. Effect of etonogestrel implant on serum lipids, liver function tests and hemoglobin levels. Contraception 2010;81:510–4. http://dx.doi.org/10.1016/j.contraception.2010.01.014
88. Nelson AL, Cwiak C. Combined oral contraceptives. In: Hatcher RA, Trussel J, Nelson A, Cates W Jr, Kowal D, Policar M, editors. Contraceptive technology. 20th ed. New York, NY: Ardent Media; 2011:249–342.
89. Dragoman M, Curtis KM, Gaffield ME. Combined hormonal contraceptive use among women with known dyslipidemias: a systematic review of critical safety outcomes. Contraception 2015;S0010-7824(15)00509-0.
90. US Department of Health and Human Services. Summary health statistics for U.S. adults: National Health Interview Survey, 2012. Report No. 0083–1972. Hyattsville, MD: US Department of Health and Human Services; 2014.
91. CDC. Surveillance for viral hepatitis—United States, 2013. Atlanta, GA: CDC; 2013. http://www.cdc.gov/hepatitis/statistics/2013surveillance/index.htm
92. Kohler BA, Sherman RL, Howlader N, et al. Annual report to the nation on the status of cancer, 1975-2011, featuring incidence of breast cancer subtypes by race/ethnicity, poverty, and state. J Natl Cancer Inst 2015;107:djv048. http://dx.doi.org/10.1093/jnci/djv048
93. Kapp N, Tilley IB, Curtis KM. The effects of hormonal contraceptive use among women with viral hepatitis or cirrhosis of the liver: a systematic review. Contraception 2009;80:381–6. http://dx.doi.org/10.1016/j.contraception.2009.04.007
94. Kapp N, Curtis KM. Hormonal contraceptive use among women with liver tumors: a systematic review. Contraception 2009;80:387–90. http://dx.doi.org/10.1016/j.contraception.2009.01.021
95. Tepper NK, Curtis KM, Steenland MW, Marchbanks PA. Physical examination prior to initiating hormonal contraception: a systematic review. Contraception 2013;87:650–4. http://dx.doi.org/10.1016/j.contraception.2012.08.010
96. National Cancer Institute. SEER Fast Stats. Washington DC: Surveillance Research Program, National Cancer Institute; 2015. http://seer.cancer.gov/faststats
97. Watson M, Saraiya M, Benard V, et al. Burden of cervical cancer in the United States, 1998–2003. Cancer 2008;113(Suppl):2855–64. http://dx.doi.org/10.1002/cncr.23756
98. National Center for Health Statistics, CDC. Health, United States, 2014: with special feature on adults. Hyattsville, MD: CDC; 2015.
99. European Study Group on Heterosexual Transmission of HIV. Comparison of female to male and male to female transmission of HIV in 563 stable couples. BMJ 1992;304:809–13. http://dx.doi.org/10.1136/bmj.304.6830.809
100. Coleman JS, Mwachari C, Balkus J, et al. Effect of the levonorgestrel intrauterine device on genital HIV-1 RNA shedding among HIV-1-infected women not taking antiretroviral therapy in Nairobi, Kenya. J Acquir Immune Defic Syndr 2013;63:245–8. http://dx.doi.org/10.1097/QAI.0b013e31828decf8
101. Haddad LB, Cwiak C, Jamieson DJ, et al. Contraceptive adherence among HIV-infected women in Malawi: a randomized controlled trial of the copper intrauterine device and depot medroxyprogesterone acetate. Contraception 2013;88:737–43. http://dx.doi.org/10.1016/j.contraception.2013.08.006

102. Heikinheimo O, Lehtovirta P, Aho I, Ristola M, Paavonen J. The levonorgestrel-releasing intrauterine system in human immunodeficiency virus-infected women: a 5-year follow-up study. Am J Obstet Gynecol 2011;204:126 e1–4.
103. Heikinheimo O, Lehtovirta P, Suni J, Paavonen J. The levonorgestrel-releasing intrauterine system (LNG-IUS) in HIV-infected women—effects on bleeding patterns, ovarian function and genital shedding of HIV. Hum Reprod 2006;21:2857–61. http://dx.doi.org/10.1093/humrep/del264
104. Kakaire O, Byamugisha JK, Tumwesigye NM, Gemzell-Danielsson K. Clinical versus laboratory screening for sexually transmitted infections prior to insertion of intrauterine contraception among women living with HIV/AIDS: a randomized controlled trial. Hum Reprod 2015;30:1573–9. http://dx.doi.org/10.1093/humrep/dev109
105. Kovacs A, Wasserman SS, Burns D, et al; DATRI Study Group; WIHS Study Group. Determinants of HIV-1 shedding in the genital tract of women. Lancet 2001;358:1593–601. http://dx.doi.org/10.1016/S0140-6736(01)06653-3
106. Landolt NK, Phanuphak N, Teeratakulpisarn N, et al. Uptake and continuous use of copper intrauterine device in a cohort of HIV-positive women. AIDS Care 2013;25:710–4. http://dx.doi.org/10.1080/09540121.2012.752786
107. Lehtovirta P, Paavonen J, Heikinheimo O. Experience with the levonorgestrel-releasing intrauterine system among HIV-infected women. Contraception 2007;75:37–9. http://dx.doi.org/10.1016/j.contraception.2006.09.006
108. Morrison CS, Sekadde-Kigondu C, Sinei SK, Weiner DH, Kwok C, Kokonya D. Is the intrauterine device appropriate contraception for HIV-1-infected women? BJOG 2001;108:784–90. http://dx.doi.org/10.1111/j.1471-0528.2001.00204.x
109. Mostad SB, Overbaugh J, DeVange DM, et al. Hormonal contraception, vitamin A deficiency, and other risk factors for shedding of HIV-1 infected cells from the cervix and vagina. Lancet 1997;350:922–7. http://dx.doi.org/10.1016/S0140-6736(97)04240-2
110. Richardson BA, Morrison CS, Sekadde-Kigondu C, et al. Effect of intrauterine device use on cervical shedding of HIV-1 DNA. AIDS 1999;13:2091–7. http://dx.doi.org/10.1097/00002030-199910220-00012
111. Sinei SK, Morrison CS, Sekadde-Kigondu C, Allen M, Kokonya D. Complications of use of intrauterine devices among HIV-1-infected women. Lancet 1998;351:1238–41. http://dx.doi.org/10.1016/S0140-6736(97)10319-1
112. Stringer EM, Kaseba C, Levy J, Sinkala M, Goldenberg RL, Chi BH, et al. A randomized trial of the intrauterine contraceptive device vs hormonal contraception in women who are infected with the human immunodeficiency virus. Am J Obstet Gynecol 2007;197:144 e1–8.
113. Stringer EM, Levy J, Sinkala M, et al. HIV disease progression by hormonal contraceptive method: secondary analysis of a randomized trial. AIDS 2009;23:1377–82. http://dx.doi.org/10.1097/QAD.0b013e32832cbca8
114. Tepper NK, Curtis KM, Nanda K, Jamieson DJ. Safety of intrauterine devices among women with HIV: a systematic review. Contraception 2016. Epub June 22, 2016. http://dx.doi.org/10.1016/j.contraception.2016.06.011
115. Lopez LM, Bernholc A, Zeng Y, et al. Interventions for pain with intrauterine device insertion. Cochrane Database Syst Rev 2015;7:CD007373.
116. Zapata LB, Jatlaoui TC, Marchbanks PA, Curtis KM. Medications to ease intrauterine device insertion: a systematic review. Contraception 2016. Epub June 29, 2016. http://dx.doi.org/10.1016/j.contraception.2016.06.014
117. Bahamondes MV, Espejo-Arce X, Bahamondes L. Effect of vaginal administration of misoprostol before intrauterine contraceptive insertion following previous insertion failure: a double blind RCT. Hum Reprod 2015;30:1861–6. http://dx.doi.org/10.1093/humrep/dev137
118. Cirik D, Taskin E, Tuglu A, Ortac A, Dai O. Paracervical block with 1% lidocaine for pain control during intrauterine device insertion: a prospective, single-blinded, controlled study. Int J Reprod Contracept Obstet Gynecol 2013;2:263–7.
119. Mody SK, Kiley J, Rademaker A, Gawron L, Stika C, Hammond C. Pain control for intrauterine device insertion: a randomized trial of 1% lidocaine paracervical block. Contraception 2012;86:704–9. http://dx.doi.org/10.1016/j.contraception.2012.06.004
120. Grimes DA, Schulz KF. Prophylactic antibiotics for intrauterine device insertion: a metaanalysis of the randomized controlled trials. Contraception 1999;60:57–63. http://dx.doi.org/10.1016/S0010-7824(99)00071-2
121. Wilson W, Taubert KA, Gewitz M, et al; American Heart Association Rheumatic Fever, Endocarditis, and Kawasaki Disease Committee; American Heart Association Council on Cardiovascular Disease in the Young; American Heart Association Council on Clinical Cardiology; American Heart Association Council on Cardiovascular Surgery and Anesthesia; Quality of Care and Outcomes Research Interdisciplinary Working Group. Prevention of infective endocarditis: guidelines from the American Heart Association: a guideline from the American Heart Association Rheumatic Fever, Endocarditis, and Kawasaki Disease Committee, Council on Cardiovascular Disease in the Young, and the Council on Clinical Cardiology, Council on Cardiovascular Surgery and Anesthesia, and the Quality of Care and Outcomes Research Interdisciplinary Working Group. Circulation 2007;116:1736–54. http://dx.doi.org/10.1161/CIRCULATIONAHA.106.183095
122. Steenland MW, Zapata LB, Brahmi D, Marchbanks PA, Curtis KM. The effect of follow-up visits or contacts after contraceptive initiation on method continuation and correct use. Contraception 2013;87:625–30. http://dx.doi.org/10.1016/j.contraception.2012.09.018
123. Steenland MW, Zapata LB, Brahmi D, Marchbanks PA, Curtis KM. Appropriate follow up to detect potential adverse events after initiation of select contraceptive methods: a systematic review. Contraception 2013;87:611–24. http://dx.doi.org/10.1016/j.contraception.2012.09.017
124. Canto De Cetina TE, Canto P, Ordoñez Luna M. Effect of counseling to improve compliance in Mexican women receiving depot-medroxyprogesterone acetate. Contraception 2001;63:143–6. http://dx.doi.org/10.1016/S0010-7824(01)00181-0
125. Lei ZW, Wu SC, Garceau RJ, et al. Effect of pretreatment counseling on discontinuation rates in Chinese women given depo-medroxyprogesterone acetate for contraception. Contraception 1996;53:357–61. http://dx.doi.org/10.1016/0010-7824(96)00085-6
126. Godfrey EM, Folger SG, Jeng G, Jamieson DJ, Curtis KM. Treatment of bleeding irregularities in women with copper-containing IUDs: a systematic review. Contraception 2013;87:549–66. http://dx.doi.org/10.1016/j.contraception.2012.09.006
127. Toppozada M, Anwar M, Abdel Rahman H, Gaweesh S. Control of IUD-induced bleeding by three non-steroidal anti-inflammatory drugs. Contracept Deliv Syst 1982;3:117–25.
128. Toppozada M, El-Attar A, El-Ayyat MA, Khamis Y. Management of uterine bleeding by PGs or their synthesis inhibitors. Adv Prostaglandin Thromboxane Res 1980;8:1459–63.

129. Wu S, Wang C, Cheng W, et al. Randomized multi-center study of baofuxin for treatment of bleeding side-effect induced by IUD. Reprod Contracept 2000;11:152–7.
130. Mercorio F, De Simone R, Di Carlo C, et al. Effectiveness and mechanism of action of desmopressin in the treatment of copper intrauterine device-related menorrhagia: a pilot study. Hum Reprod 2003;18:2319–22. http://dx.doi.org/10.1093/humrep/deg449
131. Pedron N, Lozano M, Aznar R. Treatment of hypermenorrhea with mefenamic acid in women using IUDs. Contracept Deliv Syst 1982;3:135–9.
132. Pizarro E, Mehech G, Hidalgo M, Muñoz G, Romero C. Effect of meclofenamic acid on menstruation in hypermenorrheic women using intrauterine devices [Spanish]. Rev Chil Obstet Ginecol 1988;53:43–56.
133. Chinese National IUD Research Working Group. Prevention and treatment of IUD-induced menorrhagia with antifibrinolytic and antiprostaglandin drugs [Chinese]. Zhonghua Fu Chan Ke Za Zhi 1987;22:291–4, 312.
134. Di Lieto A, Catalano D, Miranda L, Paladini A. Action of a prostaglandin synthetase inhibitor on IUD associated uterine bleeding. Clin Exp Obstet Gynecol 1987;14:41–4.
135. Ylikorkala O, Viinikka L. Comparison between antifibrinolytic and antiprostaglandin treatment in the reduction of increased menstrual blood loss in women with intrauterine contraceptive devices. Br J Obstet Gynaecol 1983;90:78–83. http://dx.doi.org/10.1111/j.1471-0528.1983.tb06751.x
136. US Food and Drug Administration. Cyklokapron: tranexamic acid injection [Prescribing information]. New York, NY; 2011.
137. US Food and Drug Administration. Lysteda: tranexamic acid tablets [Prescribing information]. Parsippany; NJ. 2011.
138. Pedrón N, Lozano M, Gallegos AJ. The effect of acetylsalicylic acid on menstrual blood loss in women with IUDs. Contraception 1987;36:295–303. http://dx.doi.org/10.1016/0010-7824(87)90099-0
139. Hidalgo M, Bahamondes L, Perrotti M, Diaz J, Dantas-Monteiro C, Petta C. Bleeding patterns and clinical performance of the levonorgestrel-releasing intrauterine system (Mirena) up to two years. Contraception 2002;65:129–32. http://dx.doi.org/10.1016/S0010-7824(01)00302-X
140. Tepper NK, Steenland MW, Gaffield ME, Marchbanks PA, Curtis KM. Retention of intrauterine devices in women who acquire pelvic inflammatory disease: a systematic review. Contraception 2013;87:655–60. http://dx.doi.org/10.1016/j.contraception.2012.08.011
141. Larsson B, Wennergren M. Investigation of a copper-intrauterine device (Cu-IUD) for possible effect on frequency and healing of pelvic inflammatory disease. Contraception 1977;15:143–9. http://dx.doi.org/10.1016/0010-7824(77)90012-9
142. Söderberg G, Lindgren S. Influence of an intrauterine device on the course of an acute salpingitis. Contraception 1981;24:137–43. http://dx.doi.org/10.1016/0010-7824(81)90086-X
143. Teisala K. Removal of an intrauterine device and the treatment of acute pelvic inflammatory disease. Ann Med 1989;21:63–5. http://dx.doi.org/10.3109/07853898909149184
144. Altunyurt S, Demir N, Posaci C. A randomized controlled trial of coil removal prior to treatment of pelvic inflammatory disease. Eur J Obstet Gynecol Reprod Biol 2003;107:81–4. http://dx.doi.org/10.1016/S0301-2115(02)00342-1
145. Stewart FH, Harper CC, Ellertson CE, Grimes DA, Sawaya GF, Trussell J. Clinical breast and pelvic examination requirements for hormonal contraception: current practice vs evidence. JAMA 2001;285:2232–9. http://dx.doi.org/10.1001/jama.285.17.2232
146. Mansour D, Korver T, Marintcheva-Petrova M, Fraser IS. The effects of Implanon on menstrual bleeding patterns. Eur J Contracept Reprod Health Care 2008;13(Suppl 1):13–28. http://dx.doi.org/10.1080/13625180801959931
147. Abdel-Aleem H, d'Arcangues C, Vogelsong K, Gülmezoglu AM. Treatment of vaginal bleeding irregularities induced by progestin only contraceptives. Cochrane Database Syst Rev 2007;(2):CD003449.
148. Archer DF, Philput CB, Levine AS, et al. Effects of ethinyl estradiol and ibuprofen compared to placebo on endometrial bleeding, cervical mucus and the postcoital test in levonorgestrel subcutaneous implant users. Contraception 2008;78:106–12. http://dx.doi.org/10.1016/j.contraception.2008.04.003
149. Buasang K, Taneepanichskul S. Efficacy of celecoxib on controlling irregular uterine bleeding secondary to Jadelle use. J Med Assoc Thai 2009;92:301–7.
150. Phaliwong P, Taneepanichskul S. The effect of mefenamic acid on controlling irregular uterine bleeding second to Implanon use. J Med Assoc Thai 2004;87(Suppl 3):S64–8.
151. Weisberg E, Hickey M, Palmer D, et al. A randomized controlled trial of treatment options for troublesome uterine bleeding in Implanon users. Hum Reprod 2009;24:1852–61. http://dx.doi.org/10.1093/humrep/dep081
152. Weisberg E, Hickey M, Palmer D, et al. A pilot study to assess the effect of three short-term treatments on frequent and/or prolonged bleeding compared to placebo in women using Implanon. Hum Reprod 2006;21:295–302. http://dx.doi.org/10.1093/humrep/dei273
153. Cheng L, Zhu H, Wang A, Ren F, Chen J, Glasier A. Once a month administration of mifepristone improves bleeding patterns in women using subdermal contraceptive implants releasing levonorgestrel. Hum Reprod 2000;15:1969–72. http://dx.doi.org/10.1093/humrep/15.9.1969
154. Alvarez-Sanchez F, Brache V, Thevenin F, Cochon L, Faundes A. Hormonal treatment for bleeding irregularities in Norplant implant users. Am J Obstet Gynecol 1996;174:919–22. http://dx.doi.org/10.1016/S0002-9378(96)70326-5
155. Díaz S, Croxatto HB, Pavez M, Belhadj H, Stern J, Sivin I. Clinical assessment of treatments for prolonged bleeding in users of Norplant implants. Contraception 1990;42:97–109. http://dx.doi.org/10.1016/0010-7824(90)90094-C
156. Witjaksono J, Lau TM, Affandi B, Rogers PA. Oestrogen treatment for increased bleeding in Norplant users: preliminary results. Hum Reprod 1996;11(Suppl 2):109–14. http://dx.doi.org/10.1093/humrep/11.suppl_2.109
157. Abdel-Aleem H, Shaaban OM, Amin AF, Abdel-Aleem AM. Tamoxifen treatment of bleeding irregularities associated with Norplant use. Contraception 2005;72:432–7. http://dx.doi.org/10.1016/j.contraception.2005.05.015
158. Phupong V, Sophonsritsuk A, Taneepanichskul S. The effect of tranexamic acid for treatment of irregular uterine bleeding secondary to Norplant use. Contraception 2006;73:253–6. http://dx.doi.org/10.1016/j.contraception.2005.09.012
159. d'Arcangues C, Piaggio G, Brache V, et al; Study Group on Progestogen-induced Vaginal Bleeding Disturbances. Effectiveness and acceptability of vitamin E and low-dose aspirin, alone or in combination, on Norplant-induced prolonged bleeding. Contraception 2004;70:451–62. http://dx.doi.org/10.1016/j.contraception.2004.05.012

160. Subakir SB, Setiadi E, Affandi B, Pringgoutomo S, Freisleben HJ. Benefits of vitamin E supplementation to Norplant users—in vitro and in vivo studies. Toxicology 2000;148:173–8. http://dx.doi.org/10.1016/S0300-483X(00)00208-0
161. Kapp N, Gaffield ME. Initiation of progestogen-only injectables on different days of the menstrual cycle and its effect on contraceptive effectiveness and compliance: a systematic review. Contraception 2013;87:576–82. http://dx.doi.org/10.1016/j.contraception.2012.08.017
162. Petta CA, Faúndes A, Dunson TR, et al. Timing of onset of contraceptive effectiveness in Depo-Provera users. II. Effects on ovarian function. Fertil Steril 1998;70:817–20. http://dx.doi.org/10.1016/S0015-0282(98)00309-4
163. Petta CA, Faundes A, Dunson TR, et al. Timing of onset of contraceptive effectiveness in Depo-Provera users: Part I. Changes in cervical mucus. Fertil Steril 1998;69:252–7. http://dx.doi.org/10.1016/S0015-0282(97)00477-9
164. Siriwongse T, Snidvongs W, Tantayaporn P, Leepipatpaiboon S. Effect of depo-medroxyprogesterone acetate on serum progesterone levels when administered on various cycle days. Contraception 1982;26:487–93. http://dx.doi.org/10.1016/0010-7824(82)90147-0
165. Balkus J, Miller L. Same-day administration of depot-medroxyprogesterone acetate injection: a retrospective chart review. Contraception 2005;71:395–8. http://dx.doi.org/10.1016/j.contraception.2004.10.014
166. Morroni C, Grams M, Tiezzi L, Westhoff C. Immediate monthly combination contraception to facilitate initiation of the depot medroxyprogesterone acetate contraceptive injection. Contraception 2004;70:19–23. http://dx.doi.org/10.1016/j.contraception.2004.02.007
167. Nelson AL, Katz T. Initiation and continuation rates seen in 2-year experience with Same Day injections of DMPA. Contraception 2007;75:84–7. http://dx.doi.org/10.1016/j.contraception.2006.09.007
168. Rickert VI, Tiezzi L, Lipshutz J, León J, Vaughan RD, Westhoff C. Depo Now: preventing unintended pregnancies among adolescents and young adults. J Adolesc Health 2007;40:22–8. http://dx.doi.org/10.1016/j.jadohealth.2006.10.018
169. Sneed R, Westhoff C, Morroni C, Tiezzi L. A prospective study of immediate initiation of depo medroxyprogesterone acetate contraceptive injection. Contraception 2005;71:99–103. http://dx.doi.org/10.1016/j.contraception.2004.08.014
170. Tepper NK, Curtis KM, Steenland MW, Marchbanks PA. Blood pressure measurement prior to initiating hormonal contraception: a systematic review. Contraception 2013;87:631–8. http://dx.doi.org/10.1016/j.contraception.2012.08.025
171. Diab KM, Zaki MM. Contraception in diabetic women: comparative metabolic study of Norplant, depot medroxyprogesterone acetate, low dose oral contraceptive pill and CuT380A. J Obstet Gynaecol Res 2000;26:17–26. http://dx.doi.org/10.1111/j.1447-0756.2000.tb01195.x
172. Garg SK, Chase HP, Marshall G, Hoops SL, Holmes DL, Jackson WE. Oral contraceptives and renal and retinal complications in young women with insulin-dependent diabetes mellitus. JAMA 1994;271:1099–102. http://dx.doi.org/10.1001/jama.1994.03510380055037
173. Grigoryan OR, Grodnitskaya EE, Andreeva EN, Chebotnikova TV, Melnichenko GA. Use of the NuvaRing hormone-releasing system in late reproductive-age women with type 1 diabetes mellitus. Gynecol Endocrinol 2008;24:99–104. http://dx.doi.org/10.1080/09513590701708795
174. Kahn HS, Curtis KM, Marchbanks PA. Effects of injectable or implantable progestin-only contraceptives on insulin-glucose metabolism and diabetes risk. Diabetes Care 2003;26:216–25. http://dx.doi.org/10.2337/diacare.26.1.216
175. Lopez LM, Grimes DA, Schulz KF. Steroidal contraceptives: effect on carbohydrate metabolism in women without diabetes mellitus. Cochrane Database Syst Rev 2009; (4):CD006133.
176. Rogovskaya S, Rivera R, Grimes DA, et al. Effect of a levonorgestrel intrauterine system on women with type 1 diabetes: a randomized trial. Obstet Gynecol 2005;105:811–5. http://dx.doi.org/10.1097/01.AOG.0000156301.11939.56
177. Troisi RJ, Cowie CC, Harris MI. Oral contraceptive use and glucose metabolism in a national sample of women in the United States. Am J Obstet Gynecol 2000;183:389–95. http://dx.doi.org/10.1067/mob.2000.105909
178. Bonny AE, Secic M, Cromer B. Early weight gain related to later weight gain in adolescents on depot medroxyprogesterone acetate. Obstet Gynecol 2011;117:793–7. http://dx.doi.org/10.1097/AOG.0b013e31820f387c
179. Le YC, Rahman M, Berenson AB. Early weight gain predicting later weight gain among depot medroxyprogesterone acetate users. Obstet Gynecol 2009;114:279–84. http://dx.doi.org/10.1097/AOG.0b013e3181af68b2
180. Risser WL, Gefter LR, Barratt MS, Risser JM. Weight change in adolescents who used hormonal contraception. J Adolesc Health 1999;24:433–6. http://dx.doi.org/10.1016/S1054-139X(98)00151-7
181. Paulen ME, Curtis KM. When can a woman have repeat progestogen-only injectables—depot medroxyprogesterone acetate or norethisterone enantate? Contraception 2009;80:391–408. http://dx.doi.org/10.1016/j.contraception.2009.03.023
182. Pardthaisong T. Return of fertility after use of the injectable contraceptive Depo Provera: up-dated data analysis. J Biosoc Sci 1984;16:23–34. http://dx.doi.org/10.1017/S0021932000014760
183. Schwallie PC, Assenzo JR. The effect of depo-medroxyprogesterone acetate on pituitary and ovarian function, and the return of fertility following its discontinuation: a review. Contraception 1974;10:181–202. http://dx.doi.org/10.1016/0010-7824(74)90073-0
184. Steiner MJ, Kwok C, Stanback J, et al. Injectable contraception: what should the longest interval be for reinjections? Contraception 2008;77:410–4. http://dx.doi.org/10.1016/j.contraception.2008.01.017
185. Bassol S, Garza-Flores J, Cravioto MC, et al. Ovarian function following a single administration of depo-medroxyprogesterone acetate (DMPA) at different doses. Fertil Steril 1984;42:216–22.
186. Fotherby K, Koetsawang S, Mathrubutham M. Pharmacokinetic study of different doses of Depo Provera. Contraception 1980;22:527–36. http://dx.doi.org/10.1016/0010-7824(80)90105-5
187. Fotherby K, Saxena BN, Shrimanker K, et al. A preliminary pharmacokinetic and pharmacodynamic evaluation of depot-medroxyprogesterone acetate and norethisterone oenanthate. Fertil Steril 1980;34:131–9.
188. Garza-Flores J, Cardenas S, Rodríguez V, Cravioto MC, Diaz-Sanchez V, Perez-Palacios G. Return to ovulation following the use of long-acting injectable contraceptives: a comparative study. Contraception 1985;31:361–6. http://dx.doi.org/10.1016/0010-7824(85)90004-6
189. Jain J, Dutton C, Nicosia A, Wajszczuk C, Bode FR, Mishell DR Jr. Pharmacokinetics, ovulation suppression and return to ovulation following a lower dose subcutaneous formulation of Depo-Provera. Contraception 2004;70:11–8. http://dx.doi.org/10.1016/j.contraception.2004.01.011
190. Lan PT, Aedo AR, Landgren BM, Johannisson E, Diczfalusy E. Return of ovulation following a single injection of depo-medroxyprogesterone acetate: a pharmacokinetic and pharmacodynamic study. Contraception 1984;29:1–18. http://dx.doi.org/10.1016/0010-7824(84)90054-4

191. Ortiz A, Hirol M, Stanczyk FZ, Goebelsmann U, Mishell DR Jr. Serum medroxyprogesterone acetate (MPA) concentrations and ovarian function following intramuscular injection of depo-MPA. J Clin Endocrinol Metab 1977;44:32–8. http://dx.doi.org/10.1210/jcem-44-1-32
192. Saxena BN, Dusitsin N, Tankeyoon M, Chaudhury RR. Return of ovulation after the cessation of depot-medroxy progesterone acetate treatment in Thai women. J Med Assoc Thai 1980;63:66–9.
193. Toh YC, Jain J, Rahnny MH, Bode FR, Ross D. Suppression of ovulation by a new subcutaneous depot medroxyprogesterone acetate (104 mg/0. 65 mL) contraceptive formulation in Asian women. Clin Ther 2004;26:1845–54. http://dx.doi.org/10.1016/j.clinthera.2004.11.013
194. Arias RD, Jain JK, Brucker C, Ross D, Ray A. Changes in bleeding patterns with depot medroxyprogesterone acetate subcutaneous injection 104 mg. Contraception 2006;74:234–8. http://dx.doi.org/10.1016/j.contraception.2006.03.008
195. Dempsey A, Roca C, Westhoff C. Vaginal estrogen supplementation during Depo-Provera initiation: a randomized controlled trial. Contraception 2010;82:250–5. http://dx.doi.org/10.1016/j.contraception.2010.04.003
196. Abdel-Aleem H, d'Arcangues C, Vogelsong KM, Gülmezoglu AM. Treatment of vaginal bleeding irregularities induced by progestin only contraceptives. Cochrane Database Syst Rev 2007; (4):CD003449.
197. Harel Z, Biro F, Kollar L, Riggs S, Flanagan P, Vaz R. Supplementation with vitamin C and/or vitamin B(6) in the prevention of Depo-Provera side effects in adolescents. J Pediatr Adolesc Gynecol 2002;15:153–8. http://dx.doi.org/10.1016/S1083-3188(02)00148-1
198. Nathirojanakun P, Taneepanichskul S, Sappakitkumjorn N. Efficacy of a selective COX-2 inhibitor for controlling irregular uterine bleeding in DMPA users. Contraception 2006;73:584–7. http://dx.doi.org/10.1016/j.contraception.2005.09.013
199. Tantiwattanakul P, Taneepanichskul S. Effect of mefenamic acid on controlling irregular uterine bleeding in DMPA users. Contraception 2004;70:277–9. http://dx.doi.org/10.1016/j.contraception.2004.04.003
200. Said S, Sadek W, Rocca M, et al. Clinical evaluation of the therapeutic effectiveness of ethinyl oestradiol and oestrone sulphate on prolonged bleeding in women using depot medroxyprogesterone acetate for contraception. World Health Organization, Special Programme of Research, Development and Research Training in Human Reproduction, Task Force on Long-acting Systemic Agents for Fertility Regulation. Hum Reprod 1996;11(Suppl 2):1–13. http://dx.doi.org/10.1093/humrep/11.suppl_2.1
201. Sadeghi-Bazargani H, Ehdaeivand F, Arshi S, Eftekhar H, Sezavar H, Amanati L. Low-dose oral contraceptive to re-induce menstrual bleeding in amenorrheic women on DMPA treatment: a randomized clinical trial. Med Sci Monit 2006;12:CR420–5.
202. Brahmi D, Curtis KM. When can a woman start combined hormonal contraceptives (CHCs)? A systematic review. Contraception 2013;87:524–38. http://dx.doi.org/10.1016/j.contraception.2012.09.010
203. Edwards SM, Zieman M, Jones K, Diaz A, Robilotto C, Westhoff C. Initiation of oral contraceptives—start now! J Adolesc Health 2008;43:432–6. http://dx.doi.org/10.1016/j.jadohealth.2008.06.008
204. Westhoff C, Heartwell S, Edwards S, et al. Initiation of oral contraceptives using a quick start compared with a conventional start: a randomized controlled trial. Obstet Gynecol 2007;109:1270–6. http://dx.doi.org/10.1097/01.AOG.0000264550.41242.f2
205. Westhoff C, Morroni C, Kerns J, Murphy PA. Bleeding patterns after immediate vs. conventional oral contraceptive initiation: a randomized, controlled trial. Fertil Steril 2003;79:322–9. http://dx.doi.org/10.1016/S0015-0282(02)04680-0
206. Baerwald AR, Olatunbosun OA, Pierson RA. Ovarian follicular development is initiated during the hormone-free interval of oral contraceptive use. Contraception 2004;70:371–7. http://dx.doi.org/10.1016/j.contraception.2004.05.006
207. Baerwald AR, Pierson RA. Ovarian follicular development during the use of oral contraception: a review. J Obstet Gynaecol Can 2004;26:19–24. http://dx.doi.org/10.1016/S1701-2163(16)30692-2
208. Duijkers IJM, Klipping C, Verhoeven CHJ, Dieben TOM. Ovarian function with the contraceptive vaginal ring or an oral contraceptive: a randomized study. Hum Reprod 2004;19:2668–73. http://dx.doi.org/10.1093/humrep/deh493
209. Killick S, Eyong E, Elstein M. Ovarian follicular development in oral contraceptive cycles. Fertil Steril 1987;48:409–13.
210. Molloy BG, Coulson KA, Lee JM, Watters JK. "Missed pill" conception: fact or fiction? Br Med J (Clin Res Ed) 1985;290:1474–5. http://dx.doi.org/10.1136/bmj.290.6480.1474-a
211. Mulders TM, Dieben TO, Bennink HJ. Ovarian function with a novel combined contraceptive vaginal ring. Hum Reprod 2002;17:2594–9. http://dx.doi.org/10.1093/humrep/17.10.2594
212. Schwartz JL, Creinin MD, Pymar HC, Reid L. Predicting risk of ovulation in new start oral contraceptive users. Obstet Gynecol 2002;99:177–82.
213. Sitavarin S, Jaisamrarn U, Taneepanichskul S. A randomized trial on the impact of starting day on ovarian follicular activity in very low dose oral contraceptive pills users. J Med Assoc Thai 2003;86:442–8.
214. Taylor DR, Anthony FW, Dennis KJ. Suppression of ovarian function by Microgynon 30 in day 1 and day 5 "starters." Contraception 1986;33:463–71. http://dx.doi.org/10.1016/S0010-7824(86)80005-1
215. Smith SK, Kirkman RJ, Arce BB, McNeilly AS, Loudon NB, Baird DT. The effect of deliberate omission of Trinordiol or Microgynon on the hypothalamo-pituitary-ovarian axis. Contraception 1986;34:513–22. http://dx.doi.org/10.1016/0010-7824(86)90060-0
216. Lara-Torre E, Schroeder B. Adolescent compliance and side effects with Quick Start initiation of oral contraceptive pills. Contraception 2002;66:81–5. http://dx.doi.org/10.1016/S0010-7824(02)00326-8
217. Murthy AS, Creinin MD, Harwood B, Schreiber CA. Same-day initiation of the transdermal hormonal delivery system (contraceptive patch) versus traditional initiation methods. Contraception 2005;72:333–6. http://dx.doi.org/10.1016/j.contraception.2005.05.009
218. Westhoff C, Osborne LM, Schafer JE, Morroni C. Bleeding patterns after immediate initiation of an oral compared with a vaginal hormonal contraceptive. Obstet Gynecol 2005;106:89–96. http://dx.doi.org/10.1097/01.AOG.0000164483.13326.59
219. Yeshaya A, Orvieto R, Kaplan B, et al. Flexible starting schedule for oral contraception: effect on the incidence of breakthrough bleeding and compliance. Eur J Contracept Reprod Health Care 1998;3:121–3. http://dx.doi.org/10.3109/13625189809051414
220. Yeshaya A, Orvieto R, Kauschansky A, et al. A delayed starting schedule of oral contraception: the effect on the incidence of breakthrough bleeding and compliance in women. Eur J Contracept Reprod Health Care 1996;1:263–5. http://dx.doi.org/10.3109/13625189609150668
221. Westhoff C, Kerns J, Morroni C, Cushman LF, Tiezzi L, Murphy PA. Quick start: novel oral contraceptive initiation method. Contraception 2002;66:141–5. http://dx.doi.org/10.1016/S0010-7824(02)00351-7
222. WHO Collaborative Study of Cardiovascular Disease and Steroid Hormone Contraception. Acute myocardial infarction and combined oral contraceptives: results of an international multicentre case-control study. Lancet 1997;349:1202–9. http://dx.doi.org/10.1016/S0140-6736(97)02358-1

223. Dunn N, Thorogood M, Faragher B, et al. Oral contraceptives and myocardial infarction: results of the MICA case-control study. BMJ 1999;318:1579–83. http://dx.doi.org/10.1136/bmj.318.7198.1579
224. Lewis MA, Heinemann LA, Spitzer WO, MacRae KD, Bruppacher R. The use of oral contraceptives and the occurrence of acute myocardial infarction in young women. Results from the Transnational Study on Oral Contraceptives and the Health of Young Women. Contraception 1997;56:129–40. http://dx.doi.org/10.1016/S0010-7824(97)00118-2
225. WHO Collaborative Study of Cardiovascular Disease and Steroid Hormone Contraception. Ischaemic stroke and combined oral contraceptives: results of an international, multicentre, case-control study. Lancet 1996;348:498–505. http://dx.doi.org/10.1016/S0140-6736(95)12393-8
226. Heinemann LA, Lewis MA, Spitzer WO, Thorogood M, Guggenmoos-Holzmann I, Bruppacher R; Transnational Research Group on Oral Contraceptives and the Health of Young Women. Thromboembolic stroke in young women. A European case-control study on oral contraceptives. Contraception 1998;57:29–37. http://dx.doi.org/10.1016/S0010-7824(97)00204-7
227. WHO Collaborative Study of Cardiovascular Disease and Steroid Hormone Contraception. Haemorrhagic stroke, overall stroke risk, and combined oral contraceptives: results of an international, multicentre, case-control study. Lancet 1996;348:505–10. http://dx.doi.org/10.1016/S0140-6736(95)12394-6
228. Mohllajee AP, Curtis KM, Martins SL, Peterson HB. Does use of hormonal contraceptives among women with thrombogenic mutations increase their risk of venous thromboembolism? A systematic review. Contraception 2006;73:166–78. http://dx.doi.org/10.1016/j.contraception.2005.08.011
229. Wu O, Robertson L, Twaddle S, et al. Screening for thrombophilia in high-risk situations: systematic review and cost-effectiveness analysis. The Thrombosis: Risk and Economic Assessment of Thrombophilia Screening (TREATS) study. Health Technol Assess 2006;10:1–110. http://dx.doi.org/10.3310/hta10110
230. Wu O, Greer IA. Is screening for thrombophilia cost-effective? Curr Opin Hematol 2007;14:500–3. http://dx.doi.org/10.1097/MOH.0b013e32825f5318
231. Blickstein D, Blickstein I. Oral contraception and thrombophilia. Curr Opin Obstet Gynecol 2007;19:370–6. http://dx.doi.org/10.1097/GCO.0b013e32821642e6
232. Steenland MW, Rodriguez MI, Marchbanks PA, Curtis KM. How does the number of oral contraceptive pill packs dispensed or prescribed affect continuation and other measures of consistent and correct use? A systematic review. Contraception 2013;87:605–10. http://dx.doi.org/10.1016/j.contraception.2012.08.004
233. White KO, Westhoff C. The effect of pack supply on oral contraceptive pill continuation: a randomized controlled trial. Obstet Gynecol 2011;118:615–22. http://dx.doi.org/10.1097/AOG.0b013e3182289eab
234. Foster DG, Parvataneni R, de Bocanegra HT, Lewis C, Bradsberry M, Darney P. Number of oral contraceptive pill packages dispensed, method continuation, and costs. Obstet Gynecol 2006;108:1107–14. http://dx.doi.org/10.1097/01.AOG.0000239122.98508.39
235. Foster DG, Hulett D, Bradsberry M, Darney P, Policar M. Number of oral contraceptive pill packages dispensed and subsequent unintended pregnancies. Obstet Gynecol 2011;117:566–72. http://dx.doi.org/10.1097/AOG.0b013e3182056309
236. Chin-Quee DS, Cuthbertson C, Janowitz B. Over-the-counter pill provision: evidence from Jamaica. Stud Fam Plann 2006;37:99–110. http://dx.doi.org/10.1111/j.1728-4465.2006.00089.x
237. Zapata LB, Steenland MW, Brahmi D, Marchbanks PA, Curtis KM. Effect of missed combined hormonal contraceptives on contraceptive effectiveness: a systematic review. Contraception 2013;87:685–700. http://dx.doi.org/10.1016/j.contraception.2012.08.035
238. Anttila L, Kunz M, Marr J. Bleeding pattern with drospirenone 3 mg+ethinyl estradiol 20 mcg 24/4 combined oral contraceptive compared with desogestrel 150 mcg+ethinyl estradiol 20 mcg 21/7 combined oral contraceptive. Contraception 2009;80:445–51. http://dx.doi.org/10.1016/j.contraception.2009.03.013
239. Chowdhury V, Joshi UM, Gopalkrishna K, Betrabet S, Mehta S, Saxena BN. 'Escape' ovulation in women due to the missing of low dose combination oral contraceptive pills. Contraception 1980;22:241–7. http://dx.doi.org/10.1016/S0010-7824(80)80003-5
240. Christin-Maitre S, Serfaty D, Chabbert-Buffet N, Ochsenbein E, Chassard D, Thomas JL. Comparison of a 24-day and a 21-day pill regimen for the novel combined oral contraceptive, nomegestrol acetate and 17*b*-estradiol (NOMAC/E2): a double-blind, randomized study. Hum Reprod 2011;26:1338–47. http://dx.doi.org/10.1093/humrep/der058
241. Creinin MD, Lippman JS, Eder SE, Godwin AJ, Olson W. The effect of extending the pill-free interval on follicular activity: triphasic norgestimate/35 μg ethinyl estradiol versus monophasic levonorgestrel/20 μg ethinyl estradiol. Contraception 2002;66:147–52. http://dx.doi.org/10.1016/S0010-7824(02)00344-X
242. Dinger J, Minh TD, Buttmann N, Bardenheuer K. Effectiveness of oral contraceptive pills in a large U.S. cohort comparing progestogen and regimen. Obstet Gynecol 2011;117:33–40. http://dx.doi.org/10.1097/AOG.0b013e31820095a2
243. Elomaa K, Lähteenmäki P. Ovulatory potential of preovulatory sized follicles during oral contraceptive treatment. Contraception 1999;60:275–9. http://dx.doi.org/10.1016/S0010-7824(99)00094-3
244. Elomaa K, Rolland R, Brosens I, et al. Omitting the first oral contraceptive pills of the cycle does not automatically lead to ovulation. Am J Obstet Gynecol 1998;179:41–6. http://dx.doi.org/10.1016/S0002-9378(98)70249-2
245. Endrikat J, Wessel J, Rosenbaum P, Düsterberg B. Plasma concentrations of endogenous hormones during one regular treatment cycle with a low-dose oral contraceptive and during two cycles with deliberate omission of two tablets. Gynecol Endocrinol 2004;18:318–26. http://dx.doi.org/10.1080/09513590420001998869
246. Hamilton CJ, Hoogland HJ. Longitudinal ultrasonographic study of the ovarian suppressive activity of a low-dose triphasic oral contraceptive during correct and incorrect pill intake. Am J Obstet Gynecol 1989;161:1159–62. http://dx.doi.org/10.1016/0002-9378(89)90655-8
247. Hedon B, Cristol P, Plauchut A, et al. Ovarian consequences of the transient interruption of combined oral contraceptives. Int J Fertil 1992;37:270–6.
248. Killick SR. Ovarian follicles during oral contraceptive cycles: their potential for ovulation. Fertil Steril 1989;52:580–2.
249. Killick SR, Bancroft K, Oelbaum S, Morris J, Elstein M. Extending the duration of the pill-free interval during combined oral contraception. Adv Contracept 1990;6:33–40. http://dx.doi.org/10.1007/BF01849485
250. Klipping C, Duijkers I, Trummer D, Marr J. Suppression of ovarian activity with a drospirenone-containing oral contraceptive in a 24/4 regimen. Contraception 2008;78:16–25. http://dx.doi.org/10.1016/j.contraception.2008.02.019

251. Klipping C, Marr J. Effects of two combined oral contraceptives containing ethinyl estradiol 20 microg combined with either drospirenone or desogestrel on lipids, hemostatic parameters and carbohydrate metabolism. Contraception 2005;71:409–16. http://dx.doi.org/10.1016/j.contraception.2004.12.005
252. Landgren BM, Csemiczky G. The effect of follicular growth and luteal function of "missing the pill." A comparison between a monophasic and a triphasic combined oral contraceptive. Contraception 1991;43:149–59. http://dx.doi.org/10.1016/0010-7824(91)90042-E
253. Landgren BM, Diczfalusy E. Hormonal consequences of missing the pill during the first two days of three consecutive artificial cycles. Contraception 1984;29:437–46. http://dx.doi.org/10.1016/0010-7824(84)90017-9
254. Letterie GS. A regimen of oral contraceptives restricted to the periovulatory period may permit folliculogenesis but inhibit ovulation. Contraception 1998;57:39–44. http://dx.doi.org/10.1016/S0010-7824(97)00205-9
255. Letterie GS, Chow GE. Effect of "missed" pills on oral contraceptive effectiveness. Obstet Gynecol 1992;79:979–82.
256. Morris SE, Groom GV, Cameron ED, Buckingham MS, Everitt JM, Elstein M. Studies on low dose oral contraceptives: plasma hormone changes in relation to deliberate pill ('Microgynon 30') omission. Contraception 1979;20:61–9. http://dx.doi.org/10.1016/0010-7824(79)90045-3
257. Nakajima ST, Archer DF, Ellman H. Efficacy and safety of a new 24-day oral contraceptive regimen of norethindrone acetate 1 mg/ethinyl estradiol 20 μg (Loestrin 24 Fe). Contraception 2007;75:16–22. http://dx.doi.org/10.1016/j.contraception.2006.08.004
258. Nuttall ID, Elstein M, McCafferty E, Seth J, Cameron ED. The effect of ethinyl estradiol 20 mcg and levonorgestrel 250 mcg on the pituitary-ovarian function during normal tablet-taking and when tablets are missed. Contraception 1982;26:121–35. http://dx.doi.org/10.1016/0010-7824(82)90081-6
259. Pierson RA, Archer DF, Moreau M, Shangold GA, Fisher AC, Creasy GW. Ortho Evra/Evra versus oral contraceptives: follicular development and ovulation in normal cycles and after an intentional dosing error. Fertil Steril 2003;80:34–42. http://dx.doi.org/10.1016/S0015-0282(03)00556-9
260. Rible RD, Taylor D, Wilson ML, Stanczyk FZ, Mishell DR Jr. Follicular development in a 7-day versus 4-day hormone-free interval with an oral contraceptive containing 20 mcg ethinyl estradiol and 1 mg norethindrone acetate. Contraception 2009;79:182–8. http://dx.doi.org/10.1016/j.contraception.2008.10.005
261. Schlaff WD, Lynch AM, Hughes HD, Cedars MI, Smith DL. Manipulation of the pill-free interval in oral contraceptive pill users: the effect on follicular suppression. Am J Obstet Gynecol 2004;190:943–51. http://dx.doi.org/10.1016/j.ajog.2004.02.012
262. Spona J, Elstein M, Feichtinger W, et al. Shorter pill-free interval in combined oral contraceptives decreases follicular development. Contraception 1996;54:71–7. http://dx.doi.org/10.1016/0010-7824(96)00137-0
263. Sullivan H, Furniss H, Spona J, Elstein M. Effect of 21-day and 24-day oral contraceptive regimens containing gestodene (60 μg) and ethinyl estradiol (15 μg) on ovarian activity. Fertil Steril 1999;72:115–20. http://dx.doi.org/10.1016/S0015-0282(99)00205-8
264. Wang E, Shi S, Cekan SZ, Landgren BM, Diczfalusy E. Hormonal consequences of "missing the pill." Contraception 1982;26:545–66. http://dx.doi.org/10.1016/0010-7824(82)90131-7
265. Willis SA, Kuehl TJ, Spiekerman AM, Sulak PJ. Greater inhibition of the pituitary—ovarian axis in oral contraceptive regimens with a shortened hormone-free interval. Contraception 2006;74:100–3. http://dx.doi.org/10.1016/j.contraception.2006.02.006
266. Abrams LS, Skee DM, Natarajan J, et al. Pharmacokinetics of norelgestromin and ethinyl estradiol delivered by a contraceptive patch (Ortho Evra/Evra) under conditions of heat, humidity, and exercise. J Clin Pharmacol 2001;41:1301–9. http://dx.doi.org/10.1177/00912700122012887
267. Ahrendt HJ, Nisand I, Bastianelli C, et al. Efficacy, acceptability and tolerability of the combined contraceptive ring, NuvaRing, compared with an oral contraceptive containing 30 microg of ethinyl estradiol and 3 mg of drospirenone. Contraception 2006;74:451–7. http://dx.doi.org/10.1016/j.contraception.2006.07.004
268. Bjarnadóttir RI, Tuppurainen M, Killick SR. Comparison of cycle control with a combined contraceptive vaginal ring and oral levonorgestrel/ethinyl estradiol. Am J Obstet Gynecol 2002;186:389–95. http://dx.doi.org/10.1067/mob.2002.121103
269. Brucker C, Karck U, Merkle E. Cycle control, tolerability, efficacy and acceptability of the vaginal contraceptive ring, NuvaRing: results of clinical experience in Germany. Eur J Contracept Reprod Health Care 2008;13:31–8. http://dx.doi.org/10.1080/13625180701577122
270. Dieben TO, Roumen FJ, Apter D. Efficacy, cycle control, and user acceptability of a novel combined contraceptive vaginal ring. Obstet Gynecol 2002;100:585–93.
271. Mulders TM, Dieben TO. Use of the novel combined contraceptive vaginal ring NuvaRing for ovulation inhibition. Fertil Steril 2001;75:865–70. http://dx.doi.org/10.1016/S0015-0282(01)01689-2
272. Guilbert E, Boroditsky R, Black A, et al; Society of Obstetricians and Gynaecologists of Canada. Canadian Consensus Guideline on Continuous and Extended Hormonal Contraception, 2007. J Obstet Gynaecol Can 2007;29(Suppl 2):S1–32. http://dx.doi.org/10.1016/S1701-2163(16)32526-9
273. Wiegratz I, Stahlberg S, Manthey T, et al. Effect of extended-cycle regimen with an oral contraceptive containing 30 mcg ethinylestradiol and 2 mg dienogest on bleeding patterns, safety, acceptance and contraceptive efficacy. Contraception 2011;84:133–43. http://dx.doi.org/10.1016/j.contraception.2011.01.002
274. Hubacher D, Fortney J. Follow-up visits after IUD insertion. Are more better? J Reprod Med 1999;44:801–6.
275. Godfrey EM, Whiteman MK, Curtis KM. Treatment of unscheduled bleeding in women using extended- or continuous-use combined hormonal contraception: a systematic review. Contraception 2013;87:567–75. http://dx.doi.org/10.1016/j.contraception.2012.08.005
276. Sulak PJ, Kuehl TJ, Coffee A, Willis S. Prospective analysis of occurrence and management of breakthrough bleeding during an extended oral contraceptive regimen. Am J Obstet Gynecol 2006;195:935–41. http://dx.doi.org/10.1016/j.ajog.2006.02.048
277. Sulak PJ, Smith V, Coffee A, Witt I, Kuehl AL, Kuehl TJ. Frequency and management of breakthrough bleeding with continuous use of the transvaginal contraceptive ring: a randomized controlled trial. Obstet Gynecol 2008;112:563–71. http://dx.doi.org/10.1097/AOG.0b013e3181842071
278. Kaneshiro B, Edelman A, Carlson N, Morgan K, Nichols M, Jensen J. Treatment of unscheduled bleeding in continuous oral contraceptive users with doxycycline: a randomized controlled trial. Obstet Gynecol 2010;115:1141–9. http://dx.doi.org/10.1097/AOG.0b013e3181e0119c

279. McCann MF, Potter LS. Progestin-only oral contraception: a comprehensive review. Contraception 1994;50(Suppl 1):S1–195. http://dx.doi.org/10.1016/0010-7824(94)90124-4
280. Arévalo M, Jennings V, Sinai I. Efficacy of a new method of family planning: the Standard Days Method. Contraception 2002;65:333–8. http://dx.doi.org/10.1016/S0010-7824(02)00288-3
281. Arévalo M, Sinai I, Jennings V. A fixed formula to define the fertile window of the menstrual cycle as the basis of a simple method of natural family planning. Contraception 1999;60:357–60. http://dx.doi.org/10.1016/S0010-7824(99)00106-7
282. Wilcox AJ, Dunson DB, Weinberg CR, Trussell J, Baird DD. Likelihood of conception with a single act of intercourse: providing benchmark rates for assessment of post-coital contraceptives. Contraception 2001;63:211–5. http://dx.doi.org/10.1016/S0010-7824(01)00191-3
283. Cleland K, Zhu H, Goldstuck N, Cheng L, Trussell J. The efficacy of intrauterine devices for emergency contraception: a systematic review of 35 years of experience. Hum Reprod 2012;27:1994–2000. http://dx.doi.org/10.1093/humrep/des140
284. Glasier AF, Cameron ST, Fine PM, et al. Ulipristal acetate versus levonorgestrel for emergency contraception: a randomised non-inferiority trial and meta-analysis. Lancet 2010;375:555–62. http://dx.doi.org/10.1016/S0140-6736(10)60101-8
285. Raymond E, Taylor D, Trussell J, Steiner MJ. Minimum effectiveness of the levonorgestrel regimen of emergency contraception. Contraception 2004;69:79–81. http://dx.doi.org/10.1016/j.contraception.2003.09.013
286. Jatlaoui TC, Curtis KM. Safety and effectiveness data for emergency contraceptive pills among women with obesity: a systematic review. Contraception 2016. Epub May 24, 2016. http://dx.doi.org/10.1016/j.contraception.2016.05.002
287. Fine P, Mathé H, Ginde S, Cullins V, Morfesis J, Gainer E. Ulipristal acetate taken 48-120 hours after intercourse for emergency contraception. Obstet Gynecol 2010;115:257–63. http://dx.doi.org/10.1097/AOG.0b013e3181c8e2aa
288. Dada OA, Godfrey EM, Piaggio G, von Hertzen H; Nigerian Network for Reproductive Health Research and Training. A randomized, double-blind, noninferiority study to compare two regimens of levonorgestrel for emergency contraception in Nigeria. Contraception 2010;82:373–8. http://dx.doi.org/10.1016/j.contraception.2010.06.004
289. Ellertson C, Evans M, Ferden S, et al. Extending the time limit for starting the Yuzpe regimen of emergency contraception to 120 hours. Obstet Gynecol 2003;101:1168–71.
290. Ngai SW, Fan S, Li S, et al. A randomized trial to compare 24 h versus 12 h double dose regimen of levonorgestrel for emergency contraception. Hum Reprod 2005;20:307–11. http://dx.doi.org/10.1093/humrep/deh583
291. Rodrigues I, Grou F, Joly J. Effectiveness of emergency contraceptive pills between 72 and 120 hours after unprotected sexual intercourse. Am J Obstet Gynecol 2001;184:531–7. http://dx.doi.org/10.1067/mob.2001.111102
292. von Hertzen H, Piaggio G, Ding J, et al; WHO Research Group on Post-ovulatory Methods of Fertility Regulation. Low dose mifepristone and two regimens of levonorgestrel for emergency contraception: a WHO multicentre randomised trial. Lancet 2002;360:1803–10. http://dx.doi.org/10.1016/S0140-6736(02)11767-3
293. Piaggio G, Kapp N, von Hertzen H. Effect on pregnancy rates of the delay in the administration of levonorgestrel for emergency contraception: a combined analysis of four WHO trials. Contraception 2011;84:35–9. http://dx.doi.org/10.1016/j.contraception.2010.11.010
294. Rodriguez MI, Curtis KM, Gaffield ML, Jackson E, Kapp N. Advance supply of emergency contraception: a systematic review. Contraception 2013;87:590–601. http://dx.doi.org/10.1016/j.contraception.2012.09.011
295. Brache V, Cochon L, Duijkers IJ, et al. A prospective, randomized, pharmacodynamic study of quick-starting a desogestrel progestin-only pill following ulipristal acetate for emergency contraception. Hum Reprod 2015;30:2785–93.
296. Cameron ST, Berger C, Michie L, Klipping C, Gemzell-Danielsson K. The effects on ovarian activity of ulipristal acetate when 'quickstarting' a combined oral contraceptive pill: a prospective, randomized, double-blind parallel-arm, placebo-controlled study. Hum Reprod 2015;30:1566–72. http://dx.doi.org/10.1093/humrep/dev115
297. Salcedo J, Rodriguez MI, Curtis KM, Kapp N. When can a woman resume or initiate contraception after taking emergency contraceptive pills? A systematic review. Contraception 2013;87:602–4. http://dx.doi.org/10.1016/j.contraception.2012.08.013
298. Rodriguez MI, Godfrey EM, Warden M, Curtis KM. Prevention and management of nausea and vomiting with emergency contraception: a systematic review. Contraception 2013;87:583–9. http://dx.doi.org/10.1016/j.contraception.2012.09.031
299. Creinin MD, Schlaff W, Archer DF, et al. Progesterone receptor modulator for emergency contraception: a randomized controlled trial. Obstet Gynecol 2006;108:1089–97. http://dx.doi.org/10.1097/01.AOG.0000239440.02284.45
300. Farajkhoda T, Khoshbin A, Enjezab B, Bokaei M, Karimi Zarchi M. Assessment of two emergency contraceptive regimens in Iran: levonorgestrel versus the Yuzpe. Niger J Clin Pract 2009;12:450–2.
301. Ho PC, Kwan MSW. A prospective randomized comparison of levonorgestrel with the Yuzpe regimen in post-coital contraception. Hum Reprod 1993;8:389–92.
302. Arowojolu AO, Okewole IA, Adekunle AO. Comparative evaluation of the effectiveness and safety of two regimens of levonorgestrel for emergency contraception in Nigerians. Contraception 2002;66:269–73. http://dx.doi.org/10.1016/S0010-7824(02)00337-2
303. Ragan RE, Rock RW, Buck HW. Metoclopramide pretreatment attenuates emergency contraceptive-associated nausea. Am J Obstet Gynecol 2003;188:330–3. http://dx.doi.org/10.1067/mob.2003.90
304. Raymond EG, Creinin MD, Barnhart KT, Lovvorn AE, Rountree RW, Trussell J. Meclizine for prevention of nausea associated with use of emergency contraceptive pills: a randomized trial. Obstet Gynecol 2000;95:271–7.
305. Peterson HB. Sterilization. Obstet Gynecol 2008;111:189–203. http://dx.doi.org/10.1097/01.AOG.0000298621.98372.62
306. Peterson HB, Xia Z, Hughes JM, Wilcox LS, Tylor LR, Trussell J. The risk of pregnancy after tubal sterilization: findings from the U.S. Collaborative Review of Sterilization. Am J Obstet Gynecol 1996;174:1161–8, discussion 1168–70. http://dx.doi.org/10.1016/S0002-9378(96)70658-0
307. Lawrie TA, Nardin JM, Kulier R, Boulvain M. Techniques for the interruption of tubal patency for female sterilisation. Cochrane Database Syst Rev 2011; (2):CD003034.
308. Cleary TP, Tepper NK, Cwiak C, et al. Pregnancies after hysteroscopic sterilization: a systematic review. Contraception 2013;87:539–48. http://dx.doi.org/10.1016/j.contraception.2012.08.006
309. Legendre G, Levaillant JM, Faivre E, Deffieux X, Gervaise A, Fernandez H. 3D ultrasound to assess the position of tubal sterilization microinserts. Hum Reprod 2011;26:2683–9. http://dx.doi.org/10.1093/humrep/der242
310. Levy B, Levie MD, Childers ME. A summary of reported pregnancies after hysteroscopic sterilization. J Minim Invasive Gynecol 2007;14:271–4. http://dx.doi.org/10.1016/j.jmig.2006.11.007

311. Veersema S, Vleugels MP, Moolenaar LM, Janssen CA, Brölmann HA. Unintended pregnancies after Essure sterilization in the Netherlands. Fertil Steril 2010;93:35–8. http://dx.doi.org/10.1016/j.fertnstert.2008.10.005
312. Arjona JE, Miño M, Cordón J, Povedano B, Pelegrin B, Castelo-Branco C. Satisfaction and tolerance with office hysteroscopic tubal sterilization. Fertil Steril 2008;90:1182–6. http://dx.doi.org/10.1016/j.fertnstert.2007.08.007
313. Duffy S, Marsh F, Rogerson L, et al. Female sterilisation: a cohort controlled comparative study of ESSURE versus laparoscopic sterilisation. BJOG 2005;112:1522–8. http://dx.doi.org/10.1111/j.1471-0528.2005.00726.x
314. Grosdemouge I, Engrand JB, Dhainault C, et al. Essure implants for tubal sterilisation in France [French]. Gynecol Obstet Fertil 2009;37:389–95. http://dx.doi.org/10.1016/j.gyobfe.2009.03.024
315. Levie MD, Chudnoff SG. Prospective analysis of office-based hysteroscopic sterilization. J Minim Invasive Gynecol 2006;13:98–101. http://dx.doi.org/10.1016/j.jmig.2005.11.010
316. Shavell VI, Abdallah ME, Diamond MP, Berman JM. Placement of a permanent birth control device at a university medical center. J Reprod Med 2009;54:218–22.
317. Teisala K. Removal of an intrauterine device and the treatment of acute pelvic inflammatory disease. Ann Med 1989;21:63–5. http://dx.doi.org/10.3109/07853898909149184
318. Bedford JM, Zelikovsky G. Viability of spermatozoa in the human ejaculate after vasectomy. Fertil Steril 1979;32:460–3.
319. Edwards IS. Earlier testing after vasectomy, based on the absence of motile sperm. Fertil Steril 1993;59:431–6.
320. Jouannet P, David G. Evolution of the properties of semen immediately following vasectomy. Fertil Steril 1978;29:435–41.
321. Labrecque M, Hays M, Chen-Mok M, Barone MA, Sokal D. Frequency and patterns of early recanalization after vasectomy. BMC Urol 2006;6:25. http://dx.doi.org/10.1186/1471-2490-6-25
322. Alderman PM. The lurking sperm. A review of failures in 8879 vasectomies performed by one physician. JAMA 1988;259:3142–4. http://dx.doi.org/10.1001/jama.1988.03720210032024
323. Black T, Francome C. The evolution of the Marie Stopes electrocautery no-scalpel vasectomy procedure. J Fam Plann Reprod Health Care 2002;28:137–8. http://dx.doi.org/10.1783/147118902101196270
324. Davies AH, Sharp RJ, Cranston D, Mitchell RG. The long-term outcome following "special clearance" after vasectomy. Br J Urol 1990;66:211–2. http://dx.doi.org/10.1111/j.1464-410X.1990.tb14907.x
325. Philp T, Guillebaud J, Budd D. Late failure of vasectomy after two documented analyses showing azoospermic semen. Br Med J (Clin Res Ed) 1984;289:77–9. http://dx.doi.org/10.1136/bmj.289.6437.77
326. Philp T, Guillebaud J, Budd D. Complications of vasectomy: review of 16,000 patients. Br J Urol 1984;56:745–8. http://dx.doi.org/10.1111/j.1464-410X.1984.tb06161.x
327. Belker AM, Sexter MS, Sweitzer SJ, Raff MJ. The high rate of noncompliance for post-vasectomy semen examination: medical and legal considerations. J Urol 1990;144:284–6.
328. Chawla A, Bowles B, Zini A. Vasectomy follow-up: clinical significance of rare nonmotile sperm in postoperative semen analysis. Urology 2004;64:1212–5. http://dx.doi.org/10.1016/j.urology.2004.07.007
329. Labrecque M, Bédard L, Laperrière L. Efficacy and complications associated with vasectomies in two clinics in the Quebec region [French]. Can Fam Physician 1998;44:1860–6.
330. Labrecque M, Nazerali H, Mondor M, Fortin V, Nasution M. Effectiveness and complications associated with 2 vasectomy occlusion techniques. J Urol 2002;168:2495–8, discussion 2498. http://dx.doi.org/10.1016/S0022-5347(05)64176-6
331. Maatman TJ, Aldrin L, Carothers GG. Patient noncompliance after vasectomy. Fertil Steril 1997;68:552–5. http://dx.doi.org/10.1016/S0015-0282(97)00251-3
332. Dhar NB, Jones JS, Bhatt A, Babineau D. A prospective evaluation of the impact of scheduled follow-up appointments with compliance rates after vasectomy. BJU Int 2007;99:1094–7. http://dx.doi.org/10.1111/j.1464-410X.2006.06725.x
333. Hillard PJ, Berek JS, Barss VA, et al. Guidelines for women's health care: A resource manual. 3rd ed. Washington, DC: American College of Obstetricians and Gynecologists; 2007.
334. Shifren J, Gass M; NAMS Recommendations for Clinical Care of Midlife Women Working Group. The North American Menopause Society recommendation for clinical care of midlife women. Menopause 2014;21;1038–62.
335. te Velde ER, Pearson PL. The variability of female reproductive ageing. Hum Reprod Update 2002;8:141–54. http://dx.doi.org/10.1093/humupd/8.2.141
336. Broekmans FJ, Soules MR, Fauser BC. Ovarian aging: mechanisms and clinical consequences. Endocr Rev 2009;30:465–93. http://dx.doi.org/10.1210/er.2009-0006
337. Wood JW. Fecundity and natural fertility in humans. Oxf Rev Reprod Biol 1989;11:61–109.
338. Balasch J, Gratacós E. Delayed childbearing: effects on fertility and the outcome of pregnancy. Curr Opin Obstet Gynecol 2012;24:187–93. http://dx.doi.org/10.1097/GCO.0b013e3283517908
339. Bateman BT, Simpson LL. Higher rate of stillbirth at the extremes of reproductive age: a large nationwide sample of deliveries in the United States. Am J Obstet Gynecol 2006;194:840–5. http://dx.doi.org/10.1016/j.ajog.2005.08.038
340. Berg CJ, Callaghan WM, Syverson C, Henderson Z. Pregnancy-related mortality in the United States, 1998 to 2005. Obstet Gynecol 2010;116:1302–9. http://dx.doi.org/10.1097/AOG.0b013e3181fdfb11
341. Lidegaard Ø, Løkkegaard E, Svendsen AL, Agger C. Hormonal contraception and risk of venous thromboembolism: national follow-up study. BMJ 2009;339:b2890.
342. Lidegaard Ø, Nielsen LH, Skovlund CW, Skjeldestad FE, Løkkegaard E. Risk of venous thromboembolism from use of oral contraceptives containing different progestogens and oestrogen doses: Danish cohort study, 2001–9. BMJ 2011;343:d6423.
343. Nightingale AL, Lawrenson RA, Simpson EL, Williams TJ, MacRae KD, Farmer RD. The effects of age, body mass index, smoking and general health on the risk of venous thromboembolism in users of combined oral contraceptives. Eur J Contracept Reprod Health Care 2000;5:265–74. http://dx.doi.org/10.1080/13625180008500402
344. Slone D, Shapiro S, Kaufman DW, Rosenberg L, Miettinen OS, Stolley PD. Risk of myocardial infarction in relation to current and discontinued use of oral contraceptives. N Engl J Med 1981;305:420–4. http://dx.doi.org/10.1056/NEJM198108203050802
345. Tanis BC, van den Bosch MA, Kemmeren JM, et al. Oral contraceptives and the risk of myocardial infarction. N Engl J Med 2001;345:1787–93. http://dx.doi.org/10.1056/NEJMoa003216
346. Collaborative Group on Hormonal Factors in Breast Cancer. Breast cancer and hormonal contraceptives: collaborative reanalysis of individual data on 53 297 women with breast cancer and 100 239 women without breast cancer from 54 epidemiological studies. Lancet 1996;347:1713–27. http://dx.doi.org/10.1016/S0140-6736(96)90806-5
347. Gill JK, Press MF, Patel AV, Bernstein L. Oral contraceptive use and risk of breast carcinoma in situ (United States). Cancer Causes Control 2006;17:1155–62. http://dx.doi.org/10.1007/s10552-006-0056-0

348. Kumle M, Weiderpass E, Braaten T, Persson I, Adami HO, Lund E. Use of oral contraceptives and breast cancer risk: The Norwegian-Swedish Women's Lifestyle and Health Cohort Study. Cancer Epidemiol Biomarkers Prev 2002;11:1375–81.
349. Marchbanks PA, McDonald JA, Wilson HG, et al. Oral contraceptives and the risk of breast cancer. N Engl J Med 2002;346:2025–32. http://dx.doi.org/10.1056/NEJMoa013202
350. Newcomb PA, Longnecker MP, Storer BE, et al. Recent oral contraceptive use and risk of breast cancer (United States). Cancer Causes Control 1996;7:525–32. http://dx.doi.org/10.1007/BF00051885
351. Rosenberg L, Palmer JR, Rao RS, et al. Case-control study of oral contraceptive use and risk of breast cancer. Am J Epidemiol 1996;143:25–37. http://dx.doi.org/10.1093/oxfordjournals.aje.a008654
352. Rosenberg L, Zhang Y, Coogan PF, Strom BL, Palmer JR. A case-control study of oral contraceptive use and incident breast cancer. Am J Epidemiol 2009;169:473–9. http://dx.doi.org/10.1093/aje/kwn360
353. Shapiro S, Rosenberg L, Hoffman M, et al. Risk of breast cancer in relation to the use of injectable progestogen contraceptives and combined estrogen/progestogen contraceptives. Am J Epidemiol 2000;151:396–403. http://dx.doi.org/10.1093/oxfordjournals.aje.a010219

Appendix A

Summary Chart of *U.S. Medical Eligibility Criteria for Contraceptive Use, 2016*

Health-care providers can use the summary table as a quick reference guide to the classifications for hormonal contraceptive methods and intrauterine contraception to compare classifications across these methods (Box A1) (Table A1). For complete guidance, see the 2016 *U.S. Medical Eligibility Criteria for Contraceptive Use* (U.S. MEC) (Curtis KM, Tepper NK, Jatlaoui TC, et al. U.S. medical eligibility criteria for contraceptive use, 2016. MMWR Recomm Rep 2016;65[No. RR-3]) for clarifications to the numeric categories, as well as for summaries of the evidence and additional comments. Hormonal contraceptives and intrauterine devices do not protect against sexually transmitted diseases (STDs), including human immunodeficiency virus (HIV), and women using these methods should be counseled that consistent and correct use of the male latex condom reduces the risk for transmission of HIV and other STDs. Use of female condoms can provide protection from transmission of STDs, although data are limited.

BOX A1. Categories for classifying hormonal contraceptives and intrauterine devices

1 = A condition for which there is no restriction for the use of the contraceptive method.
2 = A condition for which the advantages of using the method generally outweigh the theoretical or proven risks.
3 = A condition for which the theoretical or proven risks usually outweigh the advantages of using the method.
4 = A condition that represents an unacceptable health risk if the contraceptive method is used.

TABLE A1. Summary of classifications for hormonal contraceptive methods and intrauterine devices

Condition	Cu-IUD	LNG-IUD	Implants	DMPA	POP	CHCs
Personal Characteristics and Reproductive History						
Pregnancy	4*	4*	NA*	NA*	NA*	NA*
Age	Menarche to <20 years: 2 ≥20 years: 1	Menarche to <20 years: 2 ≥20 years: 1	Menarche to <18 years: 1 18–45 years: 1 >45 years: 1	Menarche to <18 years: 2 18–45 years: 1 >45 years: 2	Menarche to <18 years: 1 18–45 years: 1 >45 years: 1	Menarche to <40 years: 1 ≥40 years: 2
Parity						
a. Nulliparous	2	2	1	1	1	1
b. Parous	1	1	1	1	1	1
Breastfeeding						
a. <21 days postpartum	—	—	2*	2*	2*	4*
b. 21 to <30 days postpartum						
i. With other risk factors for VTE (e.g., age ≥35 years, previous VTE, thrombophilia, immobility, transfusion at delivery, peripartum cardiomyopathy, BMI ≥30 kg/m^2, postpartum hemorrhage, postcesarean delivery, preeclampsia, or smoking)	—	—	2*	2*	2*	3*
ii. Without other risk factors for VTE	—	—	2*	2*	2*	3*
c. 30–42 days postpartum						
i. With other risk factors for VTE (e.g., age ≥35 years, previous VTE, thrombophilia, immobility, transfusion at delivery, peripartum cardiomyopathy, BMI ≥30 kg/m^2, postpartum hemorrhage, postcesarean delivery, preeclampsia, or smoking)	—	—	1*	1*	1*	3*

See table footnotes on page 61.

TABLE A1. (*Continued*) Summary of classifications for hormonal contraceptive methods and intrauterine devices

Condition	Cu-IUD	LNG-IUD	Implants	DMPA	POP	CHCs
ii. Without other risk factors for VTE	—	—	1*	1*	1*	2*
d. >42 days postpartum	—	—	1*	1*	1*	2*
Postpartum (nonbreastfeeding women)						
a. <21 days postpartum	—	—	1	1	1	4
b. 21–42 days postpartum						
i. With other risk factors for VTE (e.g., age ≥35 years, previous VTE, thrombophilia, immobility, transfusion at delivery, peripartum cardiomyopathy, BMI ≥30 kg/m^2, postpartum hemorrhage, postcesarean delivery, preeclampsia, or smoking)	—	—	1	1	1	3*
ii. Without other risk factors for VTE	—	—	1	1	1	2
c. >42 days postpartum	—	—	1	1	1	1
Postpartum (including cesarean delivery)						
a. <10 minutes after delivery of the placenta						
i. Breastfeeding	1*	2*	—	—	—	—
ii. Nonbreastfeeding	1*	1*	—	—	—	—
b. 10 minutes after delivery of the placenta to <4 weeks (breastfeeding or nonbreastfeeding)	2*	2*	—	—	—	—
c. ≥4 weeks (breastfeeding or nonbreastfeeding)	1*	1*	—	—	—	—
d. Postpartum sepsis	4	4	—	—	—	—
Postabortion						
a. First trimester	1*	1*	1*	1*	1*	1*
b. Second trimester	2*	2*	1*	1*	1*	1*
c. Immediate postseptic abortion	4	4	1*	1*	1*	1*
Past ectopic pregnancy	1	1	1	1	2	1
History of pelvic surgery (see Postpartum [Including Cesarean Delivery] section)	1	1	1	1	1	1
Smoking						
a. Age <35 years	1	1	1	1	1	2
b. Age ≥35 years						
i. <15 cigarettes/day	1	1	1	1	1	3
ii. ≥15 cigarettes/day	1	1	1	1	1	4
Obesity						
a. BMI ≥30 kg/m^2	1	1	1	1	1	2
b. Menarche to <18 years and BMI ≥30 kg/m^2	1	1	1	2	1	2
History of bariatric surgery This condition is associated with increased risk for adverse health events as a result of pregnancy.						
a. Restrictive procedures: decrease storage capacity of the stomach (vertical banded gastroplasty, laparoscopic adjustable gastric band, or laparoscopic sleeve gastrectomy)	1	1	1	1	1	1
b. Malabsorptive procedures: decrease absorption of nutrients and calories by shortening the functional length of the small intestine (Roux-en-Y gastric bypass or biliopancreatic diversion)	1	1	1	1	3	COCs: 3 Patch and ring: 1

See table footnotes on page 61.

TABLE A1. (*Continued*) Summary of classifications for hormonal contraceptive methods and intrauterine devices

Condition	Cu-IUD	LNG-IUD	Implants	DMPA	POP	CHCs
Cardiovascular Disease						
Multiple risk factors for atherosclerotic cardiovascular disease (e.g., older age, smoking, diabetes, hypertension, low HDL, high LDL, or high triglyceride levels)	1	2	2*	3*	2*	3/4*
Hypertension Systolic blood pressure ≥160 mm Hg or diastolic blood pressure ≥100 mm Hg are associated with increased risk for adverse health events as a result of pregnancy.						
a. Adequately controlled hypertension	1*	1*	1*	2*	1*	3*
b. Elevated blood pressure levels (properly taken measurements)						
i. Systolic 140–159 mm Hg or diastolic 90–99 mm Hg	1*	1*	1*	2*	1*	3*
ii. Systolic ≥160 mm Hg or diastolic ≥100 mm Hg	1*	2*	2*	3*	2*	4*
c. Vascular disease	1*	2*	2*	3*	2*	4*
History of high blood pressure during pregnancy (when current blood pressure is measurable and normal)	1	1	1	1	1	2
Deep venous thrombosis/ Pulmonary embolism						
a. History of DVT/PE, not receiving anticoagulant therapy						
i. Higher risk for recurrent DVT/PE (one or more risk factors) • History of estrogen-associated DVT/PE • Pregnancy-associated DVT/PE • Idiopathic DVT/PE • Known thrombophilia, including antiphospholipid syndrome • Active cancer (metastatic, receiving therapy, or within 6 months after clinical remission), excluding nonmelanoma skin cancer • History of recurrent DVT/PE	1	2	2	2	2	4
ii. Lower risk for recurrent DVT/PE (no risk factors)	1	2	2	2	2	3
b. Acute DVT/PE	2	2	2	2	2	4
c. DVT/PE and established receiving anticoagulant therapy for at least 3 months						
i. Higher risk for recurrent DVT/PE (one or more risk factors) • Known thrombophilia, including antiphospholipid syndrome • Active cancer (metastatic, receiving therapy, or within 6 months after clinical remission), excluding nonmelanoma skin cancer • History of recurrent DVT/PE	2	2	2	2	2	4*
ii. Lower risk for recurrent DVT/PE (no risk factors)	2	2	2	2	2	3*

See table footnotes on page 61.

TABLE A1. (*Continued*) Summary of classifications for hormonal contraceptive methods and intrauterine devices

Condition	Cu-IUD	LNG-IUD	Implants	DMPA	POP	CHCs
d. Family history (first-degree relatives)	1	1	1	1	1	2
e. Major surgery						
i. With prolonged immobilization	1	2	2	2	2	4
ii. Without prolonged immobilization	1	1	1	1	1	2
f. Minor surgery without immobilization	1	1	1	1	1	1
Known thrombogenic mutations (e.g., factor V Leiden; prothrombin mutation; and protein S, protein C, and antithrombin deficiencies) This condition is associated with increased risk for adverse health events as a result of pregnancy.	1*	2*	2*	2*	2*	4*
Superficial venous disorders						
a. Varicose veins	1	1	1	1	1	1
b. Superficial venous thrombosis (acute or history)	1	1	1	1	1	3*
Current and history of ischemic heart disease This condition is associated with increased risk for adverse health events as a result of pregnancy.	1	Initiation: 2 Continuation: 3	Initiation: 2 Continuation: 3	3	Initiation: 2 Continuation: 3	4
Stroke (history of cerebrovascular accident) This condition is associated with increased risk for adverse health events as a result of pregnancy.	1	2	Initiation: 2 Continuation: 3	3	Initiation: 2 Continuation: 3	4
Valvular heart disease Complicated valvular heart disease is associated with increased risk for adverse health events as a result of pregnancy.						
a. Uncomplicated	1	1	1	1	1	2
b. Complicated (pulmonary hypertension, risk for atrial fibrillation, or history of subacute bacterial endocarditis)	1	1	1	1	1	4
Peripartum cardiomyopathy This condition is associated with increased risk for adverse health events as a result of pregnancy.						
a. Normal or mildly impaired cardiac function (New York Heart Association Functional Class I or II: patients with no limitation of activities or patients with slight, mild limitation of activity) (*2*)						
i. <6 months	2	2	1	1	1	4
ii. ≥6 months	2	2	1	1	1	3
b. Moderately or severely impaired cardiac function (New York Heart Association Functional Class III or IV: patients with marked limitation of activity or patients who should be at complete rest) (*2*).	2	2	2	2	2	4
Rheumatic Diseases						
Systemic lupus erythematosus This condition is associated with increased risk for adverse health events as a result of pregnancy.	Initiation / Continuation			Initiation / Continuation		
a. Positive (or unknown) antiphospholipid antibodies	1* / 1*	3*	3*	3* / 3*	3*	4*

See table footnotes on page 61.

TABLE A1. (*Continued*) Summary of classifications for hormonal contraceptive methods and intrauterine devices

Condition	Cu-IUD		LNG-IUD		Implants	DMPA		POP	CHCs
b. Severe thrombocytopenia	3*	2*	2*		2*	3*	2*	2*	2*
c. Immunosuppressive therapy	2*	1*	2*		2*	2*	2*	2*	2*
d. None of the above	1*	1*	2*		2*	2*	2*	2*	2*
Rheumatoid arthritis	Initiation	Continuation	Initiation	Continuation					
a. Receiving immunosuppressive therapy	2	1	2	1	1	2/3*		1	2
b. Not receiving immunosuppressive therapy	1		1		1	2		1	2
Neurologic Conditions									
Headaches									
a. Nonmigraine (mild or severe)	1		1		1	1		1	1*
b. Migraine									
i. Without aura (This category of migraine includes menstrual migraine.)	1		1		1	1		1	2*
ii. With aura	1		1		1	1		1	4*
Epilepsy This condition is associated with increased risk for adverse health events as a result of pregnancy.	1		1		1*	1*		1*	1*
Multiple sclerosis									
a. With prolonged immobility	1		1		1	2		1	3
b. Without prolonged immobility	1		1		1	2		1	1
Depressive Disorders									
Depressive disorders	1*		1*		1*	1*		1*	1*
Reproductive Tract Infections and Disorders									
Vaginal bleeding patterns			Initiation	Continuation					
a. Irregular pattern without heavy bleeding	1		1	1	2	2		2	1
b. Heavy or prolonged bleeding (includes regular and irregular patterns)	2*		1*	2*	2*	2*		2*	1*
Unexplained vaginal bleeding (suspicious for serious condition) before evaluation	Initiation	Continuation	Initiation	Continuation					
	4*	2*	4*	2*	3*	3*		2*	2*
Endometriosis	2		1		1	1		1	1
Benign ovarian tumors (including cysts)	1		1		1	1		1	1
Severe dysmenorrhea	2		1		1	1		1	1
Gestational trophoblastic disease This condition is associated with increased risk for adverse health events as a result of pregnancy.									
a. Suspected gestational trophoblastic disease (immediate postevacuation)									
i. Uterine size first trimester	1*		1*		1*	1*		1*	1*
ii. Uterine size second trimester	2*		2*		1*	1*		1*	1*
b. Confirmed gestational trophoblastic disease (after initial evacuation and during monitoring)	Initiation	Continuation	Initiation	Continuation					
i. Undetectable/ nonpregnant β-hCG levels	1*	1*	1*	1*	1*	1*		1*	1*
ii. Decreasing β-hCG levels	2*	1*	2*	1*	1*	1*		1*	1*
iii. Persistently elevated β-hCG levels or malignant disease, with no evidence or suspicion of intrauterine disease	2*	1*	2*	1*	1*	1*		1*	1*

See table footnotes on page 61.

TABLE A1. (*Continued*) Summary of classifications for hormonal contraceptive methods and intrauterine devices

Condition	Cu-IUD		LNG-IUD		Implants	DMPA	POP	CHCs
iv. Persistently elevated β-hCG levels or malignant disease, with evidence or suspicion of intrauterine disease	4*	2*	4*	2*	1*	1*	1*	1*
Cervical ectropion	1		1		1	1	1	1
Cervical intraepithelial neoplasia	1		2		2	2	1	2
Cervical cancer (awaiting treatment)	Initiation 4	Continuation 2	Initiation 4	Continuation 2	2	2	1	2
Breast disease Breast cancer is associated with increased risk of adverse health events as a result of pregnancy.								
a. Undiagnosed mass	1		2		2*	2*	2*	2*
b. Benign breast disease	1		1		1	1	1	1
c. Family history of cancer	1		1		1	1	1	1
d. Breast cancer								
i. Current	1		4		4	4	4	4
ii. Past and no evidence of current disease for 5 years	1		3		3	3	3	3
Endometrial hyperplasia	1		1		1	1	1	1
Endometrial cancer This condition is associated with increased risk for adverse health events as a result of pregnancy.	Initiation 4	Continuation 2	Initiation 4	Continuation 2	1	1	1	1
Ovarian cancer This condition is associated with increased risk for adverse health events as a result of pregnancy.	1		1		1	1	1	1
Uterine fibroids	2		2		1	1	1	1
Anatomical abnormalities								
a. Distorted uterine cavity (any congenital or acquired uterine abnormality distorting the uterine cavity in a manner that is incompatible with IUD insertion)	4		4		—	—	—	—
b. Other abnormalities (including cervical stenosis or cervical lacerations) not distorting the uterine cavity or interfering with IUD insertion	2		2		—	—	—	—
Pelvic inflammatory disease								
a. Past PID (assuming no current risk factors for STDs)	Initiation	Continuation	Initiation	Continuation				
i. With subsequent pregnancy	1	1	1	1	1	1	1	1
ii. Without subsequent pregnancy	2	2	2	2	1	1	1	1
b. Current PID	4	2*	4	2*	1	1	1	1
Sexually transmitted diseases	Initiation	Continuation	Initiation	Continuation				
a. Current purulent cervicitis or chlamydial infection or gonococcal infection	4	2*	4	2*	1	1	1	1
b. Vaginitis (including *Trichomonas vaginalis* and bacterial vaginosis)	2	2	2	2	1	1	1	1
c. Other factors related to STDs	2*	2	2*	2	1	1	1	1
HIV								
	Initiation	Continuation	Initiation	Continuation				
High risk for HIV	2	2	2	2	1	1*	1	1
HIV infection For women with HIV infection who are not clinically well or not receiving ARV therapy, this condition is associated with increased risk for adverse health events as a result of pregnancy.	—	—	—	—	1*	1*	1*	1*
a. Clinically well receiving ARV therapy	1	1	1	1	—	—	—	—

See table footnotes on page 61.

TABLE A1. (*Continued*) Summary of classifications for hormonal contraceptive methods and intrauterine devices

Condition	Cu-IUD		LNG-IUD		Implants	DMPA	POP	CHCs	
b. Not clinically well or not receiving ARV therapy	2	1	2	1	—	—	—	—	
Other Infections									
Schistosomiasis Schistosomiasis with fibrosis of the liver is associated with increased risk for adverse health events as a result of pregnancy.									
a. Uncomplicated	1		1		1	1	1	1	
b. Fibrosis of the liver (if severe, see Cirrhosis)	1		1		1	1	1	1	
Tuberculosis This condition is associated with increased risk for adverse health events as a result of pregnancy.	Initiation	Continuation	Initiation	Continuation					
a. Nonpelvic	1	1	1	1	1*	1*	1*	1*	
b. Pelvic	4	3	4	3	1*	1*	1*	1*	
Malaria	1		1		1	1	1	1	
Endocrine Conditions									
Diabetes Insulin-dependent diabetes; diabetes with nephropathy, retinopathy, neuropathy, or diabetes with other vascular disease; or diabetes of >20 years' duration are associated with increased risk of adverse health events as a result of pregnancy.									
a. History of gestational disease	1		1		1	1	1	1	
b. Nonvascular disease									
i. Non-insulin dependent	1		2		2	2	2	2	
ii. Insulin dependent	1		2		2	2	2	2	
c. Nephropathy, retinopathy, or neuropathy	1		2		2	3	2	3/4*	
d. Other vascular disease or diabetes of >20 years' duration	1		2		2	3	2	3/4*	
Thyroid disorders									
a. Simple goiter	1		1		1	1	1	1	
b. Hyperthyroid	1		1		1	1	1	1	
c. Hypothyroid	1		1		1	1	1	1	
Gastrointestinal Conditions									
Inflammatory bowel disease (ulcerative colitis or Crohn's disease)	1		1		1	2	2	2/3*	
Gallbladder disease									
a. Symptomatic									
i. Treated by cholecystectomy	1		2		2	2	2	2	
ii. Medically treated	1		2		2	2	2	3	
iii. Current	1		2		2	2	2	3	
b. Asymptomatic	1		2		2	2	2	2	
History of cholestasis									
a. Pregnancy related	1		1		1	1	1	2	
b. Past COC related	1		2		2	2	2	3	
Viral hepatitis								Initiation	Continuation
a. Acute or flare	1		1		1	1	1	3/4*	2
b. Carrier	1		1		1	1	1	1	1
c. Chronic	1		1		1	1	1	1	1
Cirrhosis Severe cirrhosis is associated with increased risk for adverse health events as a result of pregnancy.									
a. Mild (compensated)	1		1		1	1	1	1	
b. Severe (decompensated)	1		3		3	3	3	4	
Liver tumors Hepatocellular adenoma and malignant liver tumors are associated with increased risk for adverse health events as a result of pregnancy.									
a. Benign									

See table footnotes on page 61.

TABLE A1. (*Continued*) Summary of classifications for hormonal contraceptive methods and intrauterine devices

Condition	Cu-IUD		LNG-IUD		Implants	DMPA	POP	CHCs
i. Focal nodular hyperplasia	1		2		2	2	2	2
ii. Hepatocellular adenoma	1		3		3	3	3	4
b. Malignant (hepatoma)	1		3		3	3	3	4
Respiratory Conditions								
Cystic fibrosis This condition is associated with increased risk for adverse health events as a result of pregnancy.	1*		1*		1*	2*	1*	1*
Anemias								
Thalassemia	2		1		1	1	1	1
Sickle cell disease This condition is associated with increased risk for adverse health events as a result of pregnancy.	2		1		1	1	1	2
Iron-deficiency anemia	2		1		1	1	1	1
Solid Organ Transplantation								
Solid organ transplantation This condition is associated with increased risk for adverse health events as a result of pregnancy.	Initiation	Continuation	Initiation	Continuation				
a. Complicated: graft failure (acute or chronic), rejection, or cardiac allograft vasculopathy	3	2	3	2	2	2	2	4
b. Uncomplicated	2		2		2	2	2	2*
Drug Interactions								
Antiretroviral therapy	Initiation	Continuation	Initiation	Continuation				
a. Nucleoside reverse transcriptase inhibitors (NRTIs)								
i. Abacavir (ABC)	1/2*	1*	1/2*	1*	1	1	1	1
ii. Tenofovir (TDF)	1/2*	1*	1/2*	1*	1	1	1	1
iii. Zidovudine (AZT)	1/2*	1*	1/2*	1*	1	1	1	1
iv. Lamivudine (3TC)	1/2*	1*	1/2*	1*	1	1	1	1
v. Didanosine (DDI)	1/2*	1*	1/2*	1*	1	1	1	1
vi. Emtricitabine (FTC)	1/2*	1*	1/2*	1*	1	1	1	1
vii. Stavudine (D4T)	1/2*	1*	1/2*	1*	1	1	1	1
b. Nonnucleoside reverse transcriptase inhibitors (NNRTIs)								
i. Efavirenz (EFV)	1/2*	1*	1/2*	1*	2*	1*	2*	2*
ii. Etravirine (ETR)	1/2*	1*	1/2*	1*	1	1	1	1
iii. Nevirapine (NVP)	1/2*	1*	1/2*	1*	1	1	1	1
iv. Rilpivirine (RPV)	1/2*	1*	1/2*	1*	1	1	1	1
c. Ritonavir-boosted protease inhibitors								
i. Ritonavir-boosted atazanavir (ATV/r)	1/2*	1*	1/2*	1*	2*	1*	2*	2*
ii. Ritonavir-boosted darunavir (DRV/r)	1/2*	1*	1/2*	1*	2*	1*	2*	2*
iii. Ritonavir-boosted fosemprenavir (FPV/r)	1/2*	1*	1/2*	1*	2*	1*	2*	2*
iv. Ritonavir-boosted lopinavir (LPV/r)	1/2*	1*	1/2*	1*	1	1	1	1
v. Ritonavir-boosted saquinavir (SQV/r)	1/2*	1*	1/2*	1*	2*	1*	2*	2*
vi. Ritonavir-boosted tipranavir (TPV/r)	1/2*	1*	1/2*	1*	2*	1*	2*	2*
d. Protease inhibitors without ritonavir								
i. Atazanavir (ATV)	1/2*	1*	1/2*	1*	1	1	1	2*
ii. Fosamprenavir (FPV)	1/2*	1*	1/2*	1*	2*	2*	2*	3*
iii. Indinavir (IDV)	1/2*	1*	1/2*	1*	1	1	1	1
iv. Nelfinavir (NFV)	1/2*	1*	1/2*	1*	2*	1*	2*	2*
e. CCR5 co-receptor antagonists								
i. Maraviroc (MVC)	1/2*	1*	1/2*	1*	1	1	1	1
f. HIV integrase strand transfer inhibitors								
i. Raltegravir (RAL)	1/2*	1*	1/2*	1*	1	1	1	1
ii. Dolutegravir (DTG)	1/2*	1*	1/2*	1*	1	1	1	1
iii. Elvitegravir (EVG)	1/2*	1*	1/2*	1*	1	1	1	1

See table footnotes on next page.

TABLE A1. (*Continued*) Summary of classifications for hormonal contraceptive methods and intrauterine devices

Condition	Cu-IUD		LNG-IUD		Implants	DMPA	POP	CHCs
g. Fusion inhibitors								
i. Enfuvirtide	1/2*	1*	1/2*	1*	1	1	1	1
Anticonvulsant therapy								
a. Certain anticonvulsants (phenytoin, carbamazepine, barbiturates, primidone, topiramate, and oxcarbazepine)	1		1		2*	1*	3*	3*
b. Lamotrigine	1		1		1	1	1	3*
Antimicrobial therapy								
a. Broad-spectrum antibiotics	1		1		1	1	1	1
b. Antifungals	1		1		1	1	1	1
c. Antiparasitics	1		1		1	1	1	1
d. Rifampin or rifabutin therapy	1		1		2*	1*	3*	3*
Psychotropic medications								
a. SSRIs	1		1		1	1	1	1
St. John's wort	1		1		2	1	2	2

Abbreviations: BMI = body mass index; COC = combined oral contraceptive; Cu-IUD = copper-containing IUD; DMPA = depot medroxyprogesterone acetate; DVT = deep venous thrombosis; hCG = human chorionic gonadotropin; HDL = high-density lipoprotein; HIV = human immunodeficiency virus.; IUD = intrauterine device; LDL = low-density lipoprotein; LNG-IUD = levonorgestrel-releasing IUD; NA = not applicable; PE = pulmonary embolism; PID = pelvic inflammatory disease; POP = progestin-only pill; SSRI = selective serotonin reuptake inhibitor; STD = sexually transmitted disease.

* Consult the respective appendix for each contraceptive method in the 2016 *U.S. Medical Eligibility Criteria for Contraceptive Use* (*1*) for clarifications to the numeric categories.

References

1. Curtis KM, Tepper NK, Jatlaoui TC, et al. U.S. medical eligibility criteria for contraceptive use, 2016. MMWR Recomm Rep 2016;65(No. RR-3).
2. The Criteria Committee of the New York Heart Association. Nomenclature and criteria for diagnosis of diseases of the heart and great vessels. 9th ed. Boston, MA: Little, Brown & Co; 1994.

Appendix B

When To Start Using Specific Contraceptive Methods

Contraceptive method	When to start (if the provider is reasonably certain that the woman is not pregnant)	Additional contraception (i.e., back-up) needed	Examinations or tests needed before initiation*
Copper-containing IUD	Anytime	Not needed	Bimanual examination and cervical inspection†
Levonorgestrel-releasing IUD	Anytime	If >7 days after menses started, use back-up method or abstain for 7 days.	Bimanual examination and cervical inspection†
Implant	Anytime	If >5 days after menses started, use back-up method or abstain for 7 days.	None
Injectable	Anytime	If >7 days after menses started, use back-up method or abstain for 7 days.	None
Combined hormonal contraceptive	Anytime	If >5 days after menses started, use back-up method or abstain for 7 days.	Blood pressure measurement
Progestin-only pill	Anytime	If >5 days after menses started, use back-up method or abstain for 2 days.	None

Abbreviations: BMI = body mass index; HIV = human immunodeficiency virus; IUD = intrauterine device; STD = sexually transmitted disease; U.S. MEC = *U.S. Medical Eligibility Criteria for Contraceptive Use*.

* Weight (BMI) measurement is not needed to determine medical eligibility for any methods of contraception because all methods can be used (U.S. MEC 1) or generally can be used (U.S. MEC 2) among obese women (Box 1). However, measuring weight and calculating BMI (weight [kg] / height [m]^2) at baseline might be helpful for monitoring any changes and counseling women who might be concerned about weight change perceived to be associated with their contraceptive method.

† Most women do not require additional STD screening at the time of IUD insertion. If a woman with risk factors for STDs has not been screened for gonorrhea and chlamydia according to CDC's *STD Treatment Guidelines* (http://www.cdc.gov/std/treatment), screening can be performed at the time of IUD insertion, and insertion should not be delayed. Women with current purulent cervicitis or chlamydial infection or gonococcal infection should not undergo IUD insertion (U.S. MEC 4).

Appendix C

Examinations and Tests Needed Before Initiation of Contraceptive Methods

The examinations or tests noted apply to women who are presumed to be healthy (Table C1). Those with known medical problems or other special conditions might need additional examinations or tests before being determined to be appropriate candidates for a particular method of contraception. The 2016 *U.S. Medical Eligibility Criteria for Contraceptive Use* (U.S. MEC) might be useful in such circumstances (*1*). The following classification was considered useful in differentiating the applicability of the various examinations or tests:

- **Class A:** essential and mandatory in all circumstances for safe and effective use of the contraceptive method.
- **Class B:** contributes substantially to safe and effective use, but implementation may be considered within the public health and/or service context; risk of not performing an examination or test should be balanced against the benefits of making the contraceptive method available.
- **Class C:** does not contribute substantially to safe and effective use of the contraceptive method.

These classifications focus on the relationship of the examinations or tests to safe initiation of a contraceptive method. They are not intended to address the appropriateness of these examinations or tests in other circumstances. For example, some of the examinations or tests that are not deemed necessary for safe and effective contraceptive use might be appropriate for good preventive health care or for diagnosing or assessing suspected medical conditions. Any additional screening needed for preventive health care can be performed at the time of contraception initiation and initiation should not be delayed for test results.

No examinations or tests are needed before initiating condoms or spermicides. A bimanual examination is necessary for diaphragm fitting. A bimanual examination and cervical inspection are needed for cervical cap fitting.

References

1. Curtis KM, Tepper NK, Jatlaoui TC, et al. U.S. medical eligibility criteria for contraceptive use, 2016. MMWR Recomm Rep 2016;65(No. RR-3).

TABLE C1. Examinations and tests needed before initiation of contraceptive methods

	Contraceptive method and class							
Examination or test	**Cu-IUD and LNG-IUD**	**Implant**	**Injectable**	**CHC**	**POP**	**Condom**	**Diaphragm or cervical cap**	**Spermicide**
Examination								
Blood pressure	C	C	C	A*	C	C	C	C
Weight (BMI) (weight [kg] / height [m]^2)	—†	—†	—†	—†	—†	C	C	C
Clinical breast examination	C	C	C	C	C	C	C	C
Bimanual examination and cervical inspection	A	C	C	C	C	C	A§	C
Laboratory test								
Glucose	C	C	C	C	C	C	C	C
Lipids	C	C	C	C	C	C	C	C
Liver enzymes	C	C	C	C	C	C	C	C
Hemoglobin	C	C	C	C	C	C	C	C
Thrombogenic mutations	C	C	C	C	C	C	C	C
Cervical cytology (Papanicolaou test)	C	C	C	C	C	C	C	C
STD screening with laboratory tests	—¶	C	C	C	C	C	C	C
HIV screening with laboratory tests	C	C	C	C	C	C	C	C

Abbreviations: BMI = body mass index; CHC = combined hormonal contraceptive; Cu-IUD = copper-containing intrauterine device; HIV = human immunodeficiency virus; LNG-IUD = levonorgestrel-releasing intrauterine device; POP = progestin-only pill; STD = sexually transmitted disease; U.S. MEC = *U.S. Medical Eligibility Criteria for Contraceptive Use*.

* In instances in which blood pressure cannot be measured by a provider, blood pressure measured in other settings can be reported by the woman to her provider.

† Weight (BMI) measurement is not needed to determine medical eligibility for any methods of contraception because all methods can be used (U.S. MEC 1) or generally can be used (U.S. MEC 2) among obese women (Box 1). However, measuring weight and calculating BMI at baseline might be helpful for monitoring any changes and counseling women who might be concerned about weight change perceived to be associated with their contraceptive method.

§ A bimanual examination (not cervical inspection) is needed for diaphragm fitting.

¶ Most women do not require additional STD screening at the time of IUD insertion. If a woman with risk factors for STDs has not been screened for gonorrhea and chlamydia according to CDC's *STD Treatment Guidelines* (http://www.cdc.gov/std/treatment), screening can be performed at the time of IUD insertion, and insertion should not be delayed. Women with current purulent cervicitis or chlamydial infection or gonococcal infection should not undergo IUD insertion (U.S. MEC 4).

Appendix D

Routine Follow-Up After Contraceptive Initiation

These recommendations address when routine follow-up is recommended for safe and effective continued use of contraception for healthy women (Table D1). The recommendations refer to general situations and might vary for different users and different situations. Specific populations who might benefit from frequent follow-up visits include adolescents, those with certain medical conditions or characteristics, and those with multiple medical conditions.

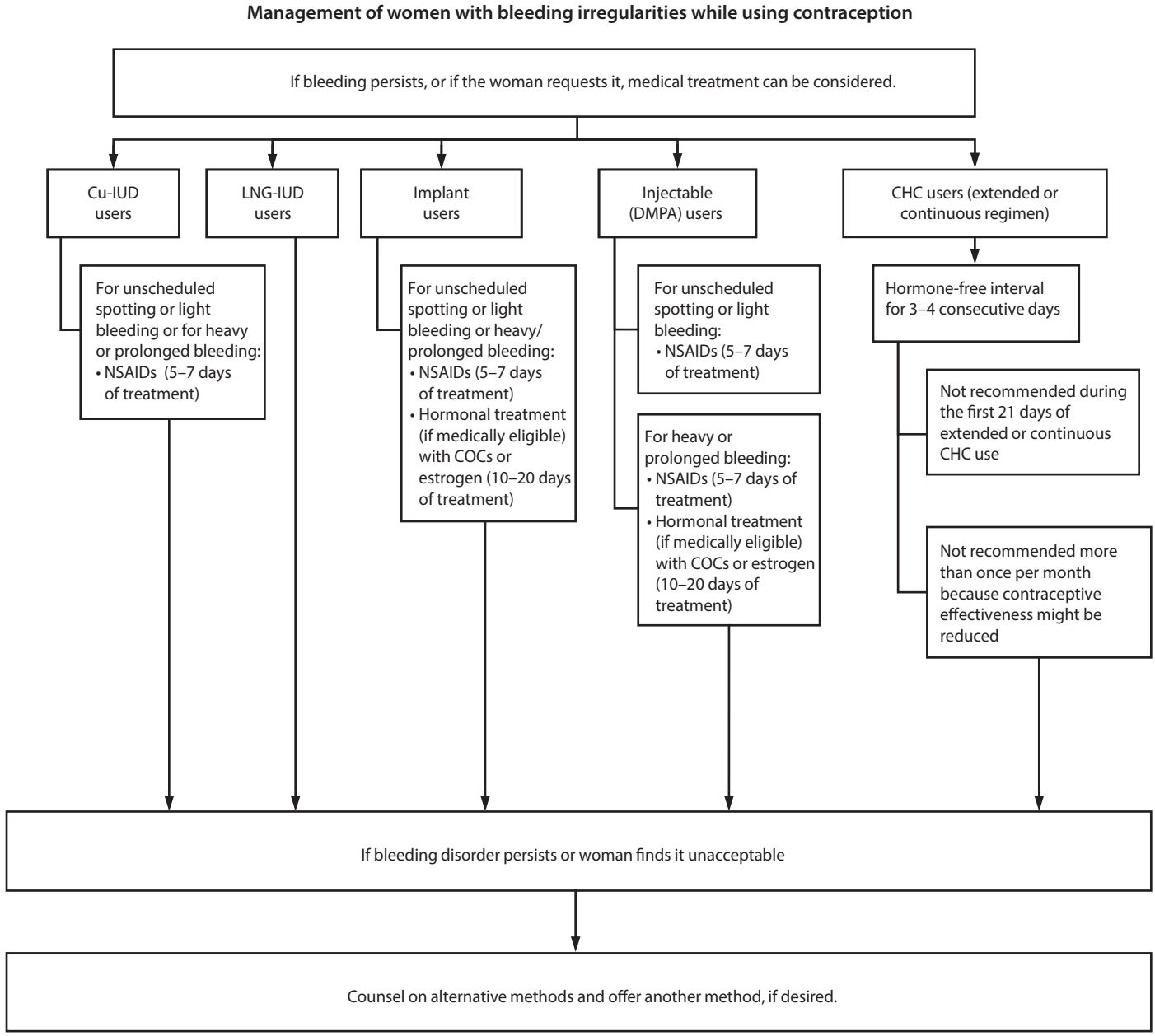
TABLE D1. Routine follow-up after contraceptive initiation

	Contraceptive method				
Action	**Cu-IUD or LNG-IUD**	**Implant**	**Injectable**	**CHC**	**POP**
General follow-up					
Advise women to return at any time to discuss side effects or other problems or if they want to change the method. Advise women using IUDs, implants, or injectables when the IUD or implant needs to be removed or when a reinjection is needed. No routine follow-up visit is required.	X	X	X	X	X
Other routine visits					
Assess the woman's satisfaction with her current method and whether she has any concerns about method use.	X	X	X	X	X
Assess any changes in health status, including medications, that would change the method's appropriateness for safe and effective continued use based on U.S. MEC (i.e., category 3 and 4 conditions and characteristics) (Box 1).	X	X	X	X	X
Consider performing an examination to check for the presence of IUD strings.	X	—	—	—	—
Consider assessing weight changes and counseling women who are concerned about weight change perceived to be associated with their contraceptive method.	X	X	X	X	X
Measure blood pressure.	—	—	—	X	—

Abbreviations: CHC = combined hormonal contraceptives; Cu-IUD = copper-containing intrauterine device; HIV = human immunodeficiency virus; IUD = intrauterine device; LNG-IUD = levonorgestrel-releasing intrauterine device; POP = progestin-only pills; U.S. MEC = *U.S. Medical Eligibility Criteria for Contraceptive Use*.

Appendix E

Management of Women with Bleeding Irregularities While Using Contraception*



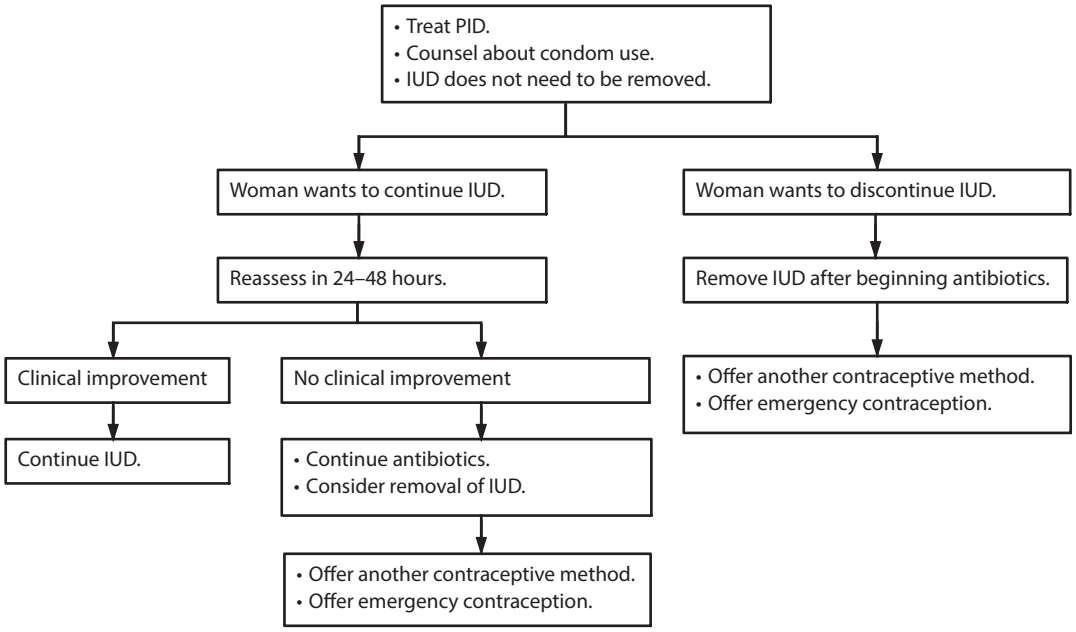
Abbreviations: CHC = combined hormonal contraceptive; COC = combined oral contraceptive; Cu-IUD = copper-containing intrauterine device; DMPA = depot medroxyprogesterone acetate; LNG-IUD = levonorgestrel-releasing intrauterine device; NSAIDs = nonsteroidal antiinflammatory drugs.

* If clinically warranted, evaluate for underlying condition. Treat the condition or refer for care. Heavy or prolonged bleeding, either unscheduled or menstrual, is uncommon among LNG-IUD users and implant users.

Appendix F

Management of Intrauterine Devices When Users are Found To Have Pelvic Inflammatory Disease*

Management of intrauterine devices when users of copper-containing intrauterine devices or levonorgestrel-releasing intrauterine devices are found to have pelvic inflammatory disease



Abbreviations: IUD = intrauterine device; PID = pelvic inflammatory disease.
* Treat according to the CDC *Sexually Transmitted Diseases Treatment Guidelines* (http://www.cdc.gov/std/treatment).

The *Morbidity and Mortality Weekly Report (MMWR)* Series is prepared by the Centers for Disease Control and Prevention (CDC) and is available free of charge in electronic format. To receive an electronic copy each week, visit *MMWR*'s free subscription page at *http://www.cdc.gov/mmwr/mmwrsubscribe.html*. Paper copy subscriptions are available through the Superintendent of Documents, U.S. Government Printing Office, Washington, DC 20402; telephone 202-512-1800.

Readers who have difficulty accessing this PDF file may access the HTML file at http://www.cdc.gov/mmwr/volumes/65/rr/rr6504a1.htm?s_cid=rr6504a1_w. Address all inquiries about the *MMWR* Series, including material to be considered for publication, to Executive Editor, *MMWR* Series, Mailstop E-90, CDC, 1600 Clifton Rd., N.E., Atlanta, GA 30329-4027 or to *mmwrq@cdc.gov.*

ISSN: 1057-5987 (Print)

Centers for Disease Control and Prevention
MMWR
Morbidity and Mortality Weekly Report
Recommendations and Reports / Vol. 65 / No. 3
July 29, 2016

U.S. Medical Eligibility Criteria for Contraceptive Use, 2016

U.S. Department of Health and Human Services
Centers for Disease Control and Prevention

CONTENTS

Disclosure of Relationship

CDC, our planners, and our content experts wish to disclose they have no financial interest or other relationships with the manufacturers of commercial products, suppliers of commercial services, or commercial supporters. Planners have reviewed content to ensure there is no bias. This document will not include any discussion of the unlabeled use of a product or a product under investigational use, with the exception that some of the recommendations in this document might be inconsistent with package labeling.

The *MMWR* series of publications is published by the Center for Surveillance, Epidemiology, and Laboratory Services, Centers for Disease Control and Prevention (CDC), U.S. Department of Health and Human Services, Atlanta, GA 30329-4027.

Suggested citation: [Author names; first three, then et al., if more than six.] [Title]. MMWR Recomm Rep 2016;65(No. RR-#):[inclusive page numbers].

U.S. Medical Eligibility Criteria for Contraceptive Use, 2016

Kathryn M. Curtis, PhD[1]
Naomi K. Tepper, MD[1]
Tara C. Jatlaoui, MD[1]
Erin Berry-Bibee, MD[1,2]
Leah G. Horton, MSPH[1]
Lauren B. Zapata, PhD[1]
Katharine B. Simmons, MD[1,2]
H. Pamela Pagano, MPH[1]
Denise J. Jamieson, MD[1]
Maura K. Whiteman, PhD[1]

[1]*Division of Reproductive Health, National Center for Chronic Disease Prevention and Health Promotion, CDC, Atlanta, Georgia*
[2]*Department of Obstetrics and Gynecology, University of North Carolina School of Medicine, Chapel Hill, North Carolina*

Summary

The 2016 U.S. Medical Eligibility Criteria for Contraceptive Use *(U.S. MEC) comprises recommendations for the use of specific contraceptive methods by women and men who have certain characteristics or medical conditions. These recommendations for health care providers were updated by CDC after review of the scientific evidence and consultation with national experts who met in Atlanta, Georgia, during August 26–28, 2015. The information in this report updates the 2010 U.S. MEC* (CDC. U.S. medical eligibility criteria for contraceptive use, 2010. MMWR 2010:59 [No. RR-4]). *Notable updates include the addition of recommendations for women with cystic fibrosis, women with multiple sclerosis, and women receiving certain psychotropic drugs or St. John's wort; revisions to the recommendations for emergency contraception, including the addition of ulipristal acetate; and revisions to the recommendations for postpartum women; women who are breastfeeding; women with known dyslipidemias, migraine headaches, superficial venous disease, gestational trophoblastic disease, sexually transmitted diseases, and human immunodeficiency virus; and women who are receiving antiretroviral therapy. The recommendations in this report are intended to assist health care providers when they counsel women, men, and couples about contraceptive method choice. Although these recommendations are meant to serve as a source of clinical guidance, health care providers should always consider the individual clinical circumstances of each person seeking family planning services. This report is not intended to be a substitute for professional medical advice for individual patients. Persons should seek advice from their health care providers when considering family planning options.*

Introduction

Approximately 45% of all pregnancies that occur in the United States are unintended (*1*), with associated increased risks for adverse maternal and infant health outcomes (*2*) and increased health care costs (*3*). Women, men, and couples have increasing numbers of safe and effective choices for contraceptive methods, including long-acting reversible contraception methods such as intrauterine devices (IUDs) and implants, to reduce the risk for an unintended pregnancy. However, with these expanded options comes the need for evidence-based guidance to help health care providers offer quality family planning care to their patients, including choosing the most appropriate contraceptive method for individual circumstances and using that method correctly, consistently, and continuously to maximize effectiveness.

In 2010, CDC published the first *U.S. Medical Eligibility Criteria for Contraceptive Use* (U.S. MEC), which provided recommendations on safe use of contraceptive methods for women with various medical conditions and other characteristics (and was adapted from global guidance developed by the World Health Organization [WHO MEC]) (*4*,*5*). U.S. MEC is a companion document to the *U.S. Selected Practice Recommendations for Contraceptive Use* (U.S. SPR), which provides guidance on how to use contraceptive methods safely and effectively once they are deemed to be medically appropriate (*6*). WHO intended for the global guidance to be used by local or national policy makers, family planning program managers, and the scientific community as a reference when they develop family planning guidance at the country or program level. During 2008–2010, CDC participated in a formal process to adapt the global guidance for appropriateness

Corresponding author: Kathryn M. Curtis, PhD, Division of Reproductive Health, National Center for Chronic Disease Prevention and Health Promotion, CDC. Telephone: 770-488-5200; E-mail: kmc6@cdc.gov.

for use in the United States, which included rigorous identification and critical appraisal of the scientific evidence through systematic reviews, and input from national experts on how to translate that evidence into recommendations for U.S. health care providers (*5*). At that time, CDC committed to keeping this guidance up to date and based on the best available evidence, with full review every few years (*5*).

This document updates CDC's U.S. MEC 2010 (*5*), based on new evidence and input from experts. A summary of changes from U.S. MEC 2010 is provided (Appendix A). Notable updates include the following:

- addition of recommendations for women with cystic fibrosis, women with multiple sclerosis, and women receiving certain psychotropic drugs or St. John's wort
- revisions to the recommendations for emergency contraception, including the addition of ulipristal acetate
- revisions to the recommendations for postpartum women; women who are breastfeeding; women with known dyslipidemias, migraine headaches, superficial venous disease, gestational trophoblastic disease, sexually transmitted diseases (STDs), and human immunodeficiency virus (HIV); and women who are receiving antiretroviral therapy

The goal of these recommendations is to remove unnecessary medical barriers to accessing and using contraception, thereby decreasing the number of unintended pregnancies. These recommendations are meant to serve as a source of clinical guidance for health care providers; health care providers should always consider the individual clinical circumstances of each person seeking family planning services. This report is not intended to be a substitute for professional medical advice for individual patients, who should seek advice from their health care providers when considering family planning options.

Methods

Since publication of U.S. MEC 2010, CDC has monitored the literature for new evidence relevant to the recommendations through the WHO/CDC continuous identification of research evidence (CIRE) system. This system identifies new evidence as it is published and allows WHO and CDC to update systematic reviews and facilitate updates to recommendations as new evidence warrants. Automated searches are run in PubMed weekly, and the results are reviewed. Abstracts that meet specific criteria are added to the web-based CIRE system, which facilitates coordination and peer review of systematic reviews for both WHO and CDC (*7*). In 2014, CDC reviewed all of the existing recommendations in U.S. MEC 2010 for new evidence identified by CIRE that had the potential to lead to a changed recommendation. During August 27–28, 2014, CDC held a meeting in Atlanta, Georgia, of 11 family planning experts and representatives from partner organizations to solicit their input on the scope of and process for updating both U.S. MEC 2010 and U.S. SPR 2013. The participants were experts in family planning and represented various types of health care providers, as well as health care provider organizations. A list of participants is provided at the end of this report. Meeting participants discussed topics to be addressed in the update of U.S. MEC based on new evidence published since 2010 (identified through the CIRE system), topics addressed at a 2014 WHO meeting to update global guidance, and suggestions CDC received from health care providers for the addition of recommendations for women with medical conditions not yet included in U.S. MEC (e.g., from provider feedback through e-mail, public inquiry, and questions received at conferences). CDC identified several topics to consider when updating the guidance, including revision of existing recommendations for certain medical conditions or characteristics (breastfeeding, postpartum, HIV, receiving antiretroviral therapy, obesity, dyslipidemia, increased risk for STDs, superficial venous thrombosis, gestational trophoblastic disease, and migraine headaches), addition of recommendations for new medical conditions (cystic fibrosis, multiple sclerosis, use of certain psychotropic drugs, and St. John's wort), and addition of recommendations for new contraceptive methods (ulipristal acetate for emergency contraception). CDC determined that all other recommendations in U.S. MEC 2010 were up to date and consistent with the existing body of evidence for that recommendation.

In preparation for a subsequent expert meeting held during August 26–28, 2015, to review the scientific evidence for potential recommendations, CDC staff members and other invited authors listed at the end of this report conducted independent systematic reviews for each of the topics being considered. The purpose of these systematic reviews was to identify direct evidence about the safety of contraceptive method use by women with selected conditions (e.g., risk for disease progression or other adverse health effects in women with multiple sclerosis who use combined hormonal contraceptives [CHCs]). Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines were followed for reporting systematic reviews (*8*,*9*), and strength and quality of the evidence were assigned using the system of the U.S. Preventive Services Task Force (*10*). When direct evidence was limited or not available, indirect evidence (e.g., evidence on surrogate outcomes or among healthy women) and theoretical issues were considered and either added to direct evidence within a systematic review or separately compiled for presentation to the meeting participants. Completed systematic

reviews were peer reviewed by two or three experts and then provided to participants before the expert meeting. Reviews are referenced and cited throughout this document; the full reviews appear in the published literature and contain the details of each review, including the systematic review question, literature search protocol, inclusion and exclusion criteria, evidence tables, and quality assessments. CDC staff continued to monitor new evidence identified through the CIRE system during the preparation for the August 2015 meeting.

During August 26–28, 2015, in Atlanta, Georgia, CDC held a meeting with 44 participants who were invited to provide their individual perspectives on the scientific evidence presented and potential recommendations. Twenty-nine of the participants represented a wide range of expertise in family planning provision and research, and included obstetricians/gynecologists, pediatricians, family physicians, nurse practitioners, epidemiologists, and others with research and clinical practice expertise in contraceptive safety, effectiveness, and management; these individuals participated in the entire meeting. Fifteen participants with expertise relevant to specific topics on the meeting agenda provided information and participated in the discussion (e.g., an expert in cystic fibrosis was asked to provide general information about the condition and to assist in interpreting the evidence and any theoretical concerns on the use of contraceptive methods in women with the condition); these participants provided input only during the session for which their topics were discussed. Lists of participants and any potential conflicts of interest are provided at the end of this report. During the meeting, the evidence from the systematic review for each topic was presented, including direct evidence and any indirect evidence or theoretical concerns. Participants provided their perspectives on using the evidence to develop recommendations that would meet the needs of U.S. health care providers. After the meeting, CDC determined the recommendations in this report, taking into consideration the perspectives provided by the meeting participants. Feedback also was received from three external reviewers, composed of health care providers and researchers who had not participated in the update meetings. These reviewers were asked to provide comments on the accuracy, feasibility, and clarity of the recommendations. Areas of research that need additional investigation also were considered during the meeting (*11*).

How to Use This Document

These recommendations are intended to help health care providers determine the safe use of contraceptive methods among women and men with various characteristics and medical conditions. Providers also can use the information in these recommendations when consulting with women, men, and couples about their selection of contraceptive methods. The tables in this document include recommendations for the use of contraceptive methods by women and men with particular characteristics or medical conditions. Each condition is defined as representing either an individual's characteristics (e.g., age or history of pregnancy) or a known preexisting medical or pathologic condition (e.g., diabetes or hypertension). The recommendations refer to contraceptive methods being used for contraceptive purposes; the recommendations do not consider the use of contraceptive methods for treatment of medical conditions because the eligibility criteria in these situations might differ. The conditions affecting eligibility for the use of each contraceptive method are classified into one of four categories (Box 1).

Using the Categories in Practice

Health care providers can use the eligibility categories when assessing the safety of contraceptive method use for women and men with specific medical conditions or characteristics. Category 1 comprises conditions for which no restrictions exist for use of the contraceptive method. Classification of a method/condition as category 2 indicates the method generally can be used, although careful follow-up might be required. For a method/condition classified as category 3, use of that method usually is not recommended unless other more appropriate methods are not available or acceptable. The severity of the condition and the availability, practicality, and acceptability of alternative methods should be considered, and careful follow-up is required. Hence, provision of a contraceptive method to a woman with a condition classified as category 3 requires careful clinical judgement and access to clinical services. Category 4 comprises conditions that represent an unacceptable health risk if the method is used. For example, a smoker aged <35 years generally can use combined oral contraceptives (COCs) (category 2). However, for a woman

BOX 1. Categories of medical eligibility criteria for contraceptive use

1 = A condition for which there is no restriction for the use of the contraceptive method.
2 = A condition for which the advantages of using the method generally outweigh the theoretical or proven risks.
3 = A condition for which the theoretical or proven risks usually outweigh the advantages of using the method.
4 = A condition that represents an unacceptable health risk if the contraceptive method is used.

aged ≥35 years who smokes <15 cigarettes per day, the use of COCs usually is not recommended unless other methods are not available or acceptable to her (category 3). A woman aged ≥35 years who smokes ≥15 cigarettes per day should not use COCs because of unacceptable health risks, primarily the risk for myocardial infarction and stroke (category 4). The programmatic implications of these categories might depend on the circumstances of particular professional or service organizations. For example, in some settings, a category 3 might mean that a special consultation is warranted.

The recommendations address medical eligibility criteria for the initiation and continued use of all methods evaluated. The issue of continuation criteria is clinically relevant whenever a medical condition develops or worsens during use of a contraceptive method. When the categories differ for initiation and continuation, these differences are noted in the Initiation and Continuation columns. When initiation and continuation are not indicated, the category is the same for initiation and continuation of use.

On the basis of this classification system, the eligibility criteria for initiating and continuing use of a specific contraceptive method are presented in tables (Appendices A–K). In these tables, the first column indicates the condition. Several conditions are divided into subconditions to differentiate between varying types or severity of the condition. The second column classifies the condition for initiation or continuation (or both) into category 1, 2, 3, or 4. For certain conditions, the numeric classification does not adequately capture the recommendation; in these cases, the third column clarifies the numeric category. These clarifications were determined during the discussions of the scientific evidence and are considered a necessary element of the recommendation. The third column also summarizes the evidence for the recommendation if evidence exists. The recommendations for which no evidence is cited are based on expert opinion from either the WHO or U.S. expert meeting in which these recommendations were developed, and might be based on evidence from sources other than systematic reviews. For certain recommendations, additional comments appear in the third column and generally come from the WHO meeting or the U.S. meeting.

Recommendations for Use of Contraceptive Methods

The classifications for whether women with certain medical conditions or characteristics can use specific contraceptive methods are provided for intrauterine contraception, including the copper-containing IUD and levonorgestrel-releasing IUDs (Appendix B); progestin-only contraceptives (POCs), including etonogestrel implants, depot medroxyprogesterone acetate injections, and progestin-only pills (Appendix C); CHCs, including low-dose (containing ≤35 μg ethinyl estradiol) COCs, combined hormonal patch, and combined vaginal ring (Appendix D); barrier contraceptive methods, including male and female condoms, spermicides, diaphragm with spermicide, and cervical cap (Appendix E); fertility awareness–based methods (Appendix F); lactational amenorrhea method (Appendix G); coitus interruptus (Appendix H); female and male sterilization (Appendix I); and emergency contraception, including emergency use of the copper-containing IUD and emergency contraceptive pills (Appendix J). A table at the end of this report summarizes the classifications for the hormonal and intrauterine methods (Appendix K).

Contraceptive Method Choice

Many elements need to be considered by women, men, or couples at any given point in their lifetimes when choosing the most appropriate contraceptive method. These elements include safety, effectiveness, availability (including accessibility and affordability), and acceptability. The guidance in this report focuses primarily on the safety of a given contraceptive method for a person with a particular characteristic or medical condition. Therefore, the classification of category 1 means that the method can be used in that circumstance with no restrictions with regard to safety but does not necessarily imply that the method is the best choice for that person; other factors, such as effectiveness, availability, and acceptability, might play an important role in determining the most appropriate choice. Voluntary informed choice of contraceptive methods is an essential guiding principle, and contraceptive counseling, when applicable, might be an important contributor to the successful use of contraceptive methods.

In choosing a method of contraception, dual protection from the simultaneous risk for HIV and other STDs also should be considered. Although hormonal contraceptives and IUDs are highly effective at preventing pregnancy, they do not protect against STDs, including HIV. Consistent and correct use of the male latex condom reduces the risk for HIV infection and other STDs, including chlamydial infection, gonococcal infection, and trichomoniasis (*12*). Although evidence is limited, use of female condoms can provide protection from acquisition and transmission of STDs (*12*). All patients, regardless of contraceptive choice, should be counseled about the use of condoms and the risk for STDs, including HIV infection (*12*). Additional information about prevention and treatment of STDs is available from the CDC *Sexually Transmitted Diseases Treatment Guidelines* (http://www.cdc.gov/std/treatment) (*12*).

Contraceptive Method Effectiveness

Contraceptive method effectiveness is critical for minimizing the risk for an unintended pregnancy, particularly among women for whom an unintended pregnancy would pose additional health risks. The effectiveness of contraceptive methods depends both on the inherent effectiveness of the method itself and on how consistently and correctly it is used (Figure). Methods that depend on consistent and correct use have a wide range of effectiveness. IUDs and implants are considered long-acting, reversible contraception (LARC); these methods are highly effective because they do not depend on regular compliance from the user. LARC methods are appropriate for most women, including adolescents and nulliparous women. All women should be counseled about the full range and effectiveness of contraceptive options for which they are medically eligible so that they can identify the optimal method.

Unintended Pregnancy and Increased Health Risk

For women with conditions that might make pregnancy an unacceptable health risk, long-acting, highly effective contraceptive methods might be the best choice to avoid unintended pregnancy (Figure). Women with these conditions should be advised that sole use of barrier methods for contraception and behavior-based methods of contraception might not be the most appropriate choice because of their relatively higher typical-use rates of failure (Figure). Conditions included in U.S. MEC that are associated with increased risk for adverse health events as a result of pregnancy are identified throughout the document (Box 2). Some of the medical conditions included in U.S. MEC recommendations are treated with teratogenic drugs. While the woman's medical condition may not affect her eligibility to use certain contraceptive methods, women using teratogenic drugs are at increased risk for poor pregnancy outcomes; long-acting, highly effective contraceptive methods might be the best option to avoid unintended pregnancy or delay pregnancy until teratogenic drugs are no longer needed.

BOX 2. Conditions associated with increased risk for adverse health events as a result of pregnancy*

- Breast cancer
- Complicated valvular heart disease
- Cystic fibrosis
- Diabetes: insulin dependent; with nephropathy, retinopathy, or neuropathy or other vascular disease; or of >20 years' duration
- Endometrial or ovarian cancer
- Epilepsy
- Hypertension (systolic ≥160 mm Hg or diastolic ≥100 mm Hg)
- History of bariatric surgery within the past 2 years
- HIV: not clinically well or not receiving antiretroviral therapy
- Ischemic heart disease
- Gestational trophoblastic disease
- Hepatocellular adenoma and malignant liver tumors (hepatoma)
- Peripartum cardiomyopathy
- Schistosomiasis with fibrosis of the liver
- Severe (decompensated) cirrhosis
- Sickle cell disease
- Solid organ transplantation within the past 2 years
- Stroke
- Systemic lupus erythematosus
- Thrombogenic mutations
- Tuberculosis

* Long-acting, highly effective contraceptive methods might be the best choice for women with conditions that are associated with increased risk for adverse health events as a result of pregnancy. These women should be advised that sole use of barrier methods for contraception and behavior-based methods of contraception might not be the most appropriate choice because of their relatively higher typical-use rates of failure.

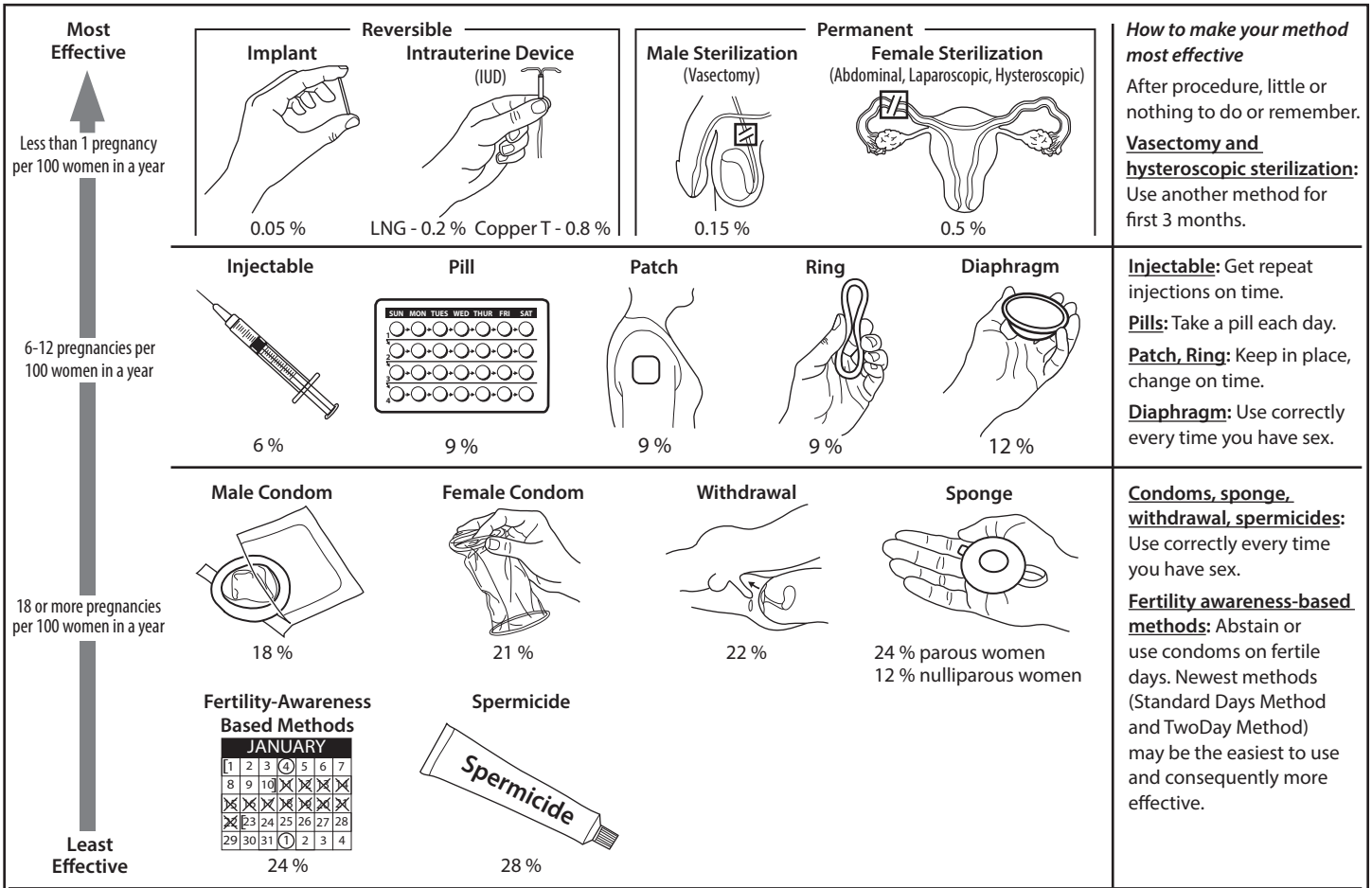
Keeping Guidance Up to Date

Updating the evidence-based recommendations as new scientific evidence becomes available is a challenge. CDC will continue to work with WHO to identify and assess new relevant evidence as it becomes available and to determine whether changes in the recommendations are warranted (*7*). In most cases, U.S. MEC follows the WHO guidance updates, which typically occur every 5 years (or sooner if warranted by new data). However, CDC will review all WHO updates for their application in the United States. CDC also will identify and assess any new literature for the recommendations and medical conditions that are not included in the WHO guidance. CDC will completely review U.S. MEC every 5 years as well. Updates to the guidance will appear on the CDC U.S. MEC website (http://www.cdc.gov/reproductivehealth/UnintendedPregnancy/USMEC.htm).

Acknowledgments

This report is based, in part, on the work of the Promoting Family Planning Team, Department of Reproductive Health and Research, World Health Organization, and its development of *Medical Eligibility Criteria for Contraceptive Use, 5th edition*.

FIGURE. Effectiveness of family planning methods*



CONDOMS SHOULD ALWAYS BE USED TO REDUCE THE RISK OF SEXUALLY TRANSMITTED INFECTIONS.

Other Methods of Contraception

Lactational Amenorrhea Method: LAM is a highly effective, *temporary* method of contraception.

Emergency Contraception: Emergency contraceptive pills or a copper IUD after unprotected intercourse substantially reduces risk of pregnancy.

Sources: Adapted from World Health Organization (WHO) Department of Reproductive Health and Research, Johns Hopkins Bloomberg School of Public Health/ Center for Communication Programs (CCP). Knowledge for health project. Family planning: a global handbook for providers (2011 update). Baltimore, MD; Geneva, Switzerland: CCP and WHO; 2011; and Trussell J. Contraceptive failure in the United States. Contraception 2011;83:397–404.

* The percentages indicate the number out of every 100 women who experienced an unintended pregnancy within the first year of typical use of each contraceptive method.

U.S. Medical Eligibility Criteria for Contraceptive Use Participants

CDC Guideline Development Group for *U.S. Medical Eligibility Criteria for Contraceptive Use* and *U.S. Selected Practice Recommendations for Contraceptive Use*

Kathryn M. Curtis, PhD (Chair), Erin Berry-Bibee, MD, Suzanne G. Folger, PhD, Leah G. Horton, MSPH, Denise J. Jamieson, MD, Tara C. Jatlaoui, MD, Polly A. Marchbanks, PhD, H. Pamela Pagano, MPH, Halley E.M. Riley, MPH, Mirelys Rodriguez, Katharine B. Simmons, MD, Naomi K. Tepper, MD, Maura K. Whiteman, PhD, Lauren B. Zapata, PhD, CDC, Atlanta, Georgia.

Invited Meeting Participants, August 27–28, 2014, Atlanta, Georgia

Herbert Peterson, MD, University of North Carolina, Chapel Hill, North Carolina (Chair); Gale Burstein, MD, Erie County Department of Health, Buffalo, New York; Alison Edelman, MD, Oregon Health and Science University, Portland, Oregon; Eve Espey, MD, University of New Mexico, Albuquerque, New Mexico; Emily Godfrey, MD, University of Washington, Seattle, Washington; Andrew Kaunitz, MD, University of Florida, Jacksonville, Florida; Susan Moskosky, MS, U.S. Department of Health and Human Services, Rockville, Maryland; Kavita Nanda, MD, FHI360 and American College of Obstetricians and Gynecologists, Durham, North Carolina; Deborah Nucatola, MD, Planned Parenthood Federation of America, New York, New York; Michael Policar, MD, University of California, San Francisco, California; Carolyn Westhoff, MD, Columbia University, New York, New York.

Systematic Review Presenters and Authors, Meeting, August 26–28, 2015, Atlanta, Georgia

Erin Berry-Bibee, MD, Kathryn M. Curtis, PhD, Leah G. Horton, MSPH, Denise J. Jamieson, MD, Tara C. Jatlaoui, MD, Polly A. Marchbanks PhD, Titilope Oduyebo, MD, Pamela Pagano, MPH, Halley E.M. Riley, MPH, Katharine B. Simmons, MD, Naomi K. Tepper, MD, Maura K. Whiteman, PhD, Lauren B. Zapata, PhD, CDC, Atlanta, Georgia; Monica Dragoman, MD, Mary E. Gaffield, PhD, Sharon J. Phillips, MD, Marleen Temmerman, MD, World Health Organization, Geneva, Switzerland; Andra James, MD, Duke University Medical Center, Durham, North Carolina; Nathalie Kapp, MD, HRA Pharma, Paris, France; Kavita Nanda, MD, FHI360, Durham, North Carolina; Seth Walker, MD, Emory University, Atlanta, Georgia.

Invited Meeting Participants, August 26–28, 2015, Atlanta, Georgia

Herbert Peterson, MD, University of North Carolina, Chapel Hill, North Carolina (Chair); Rebecca Allen, MD, American Society for Reproductive Medicine and Women and Infants Hospital, Providence, Rhode Island; Jean Anderson, MD, Johns Hopkins University, Baltimore, Maryland; Abbey Berenson, MD, University of Texas Medical Branch, Galveston, Texas; Amanda Black, MD, University of Ottawa, Ontario, Canada; Cora Collette Breuner, MD, American Academy of Pediatrics and Seattle Children's Hospital, Seattle, Washington; Gale Burstein MD, Erie County Department of Health, Buffalo, New York; Anne Calhoun, MD, University of North Carolina, Chapel Hill, North Carolina; Nahida Chakhtoura, MD, National Institutes of Health, Rockville, Maryland; Alicia Christy, MD, National Institutes of Health, Rockville, Maryland; Mitchell D. Creinin, MD, University of California, Davis, California; Linda Dominguez, Southwest Women's Health, Albuquerque, New Mexico; Alison Edelman, MD, Oregon Health and Science University, Portland, Oregon; Eve Espey, MD, University of New Mexico, Albuquerque, New Mexico; Emily Godfrey, MD, University of Washington, Seattle, Washington; Marji Gold, MD, Albert Einstein College of Medicine, Bronx, New York; Donald Goldstein, MD, Harvard Medical School, Boston, Massachusetts; Robert Hatcher, MD, Emory University, Atlanta, Georgia; Mark Hathaway, MD, JHPIEGO and Unity Healthcare, Washington, DC; Stephen Heartwell, MD, Susan Thompson Buffett Foundation, Omaha, Nebraska; Paula Hillard, MD, Stanford University, Palo Alto, California; Maria Houtchens, MD, Harvard Medical School, Boston, Massachusetts; Andra James, MD, Duke University Medical Center, Durham, North Carolina; Andrew Kaunitz, MD, University of Florida, Jacksonville, Florida; Myong-Jin Kim, Pharm-D, Food and Drug Administration, Rockville, Maryland; Barbara Konkle, MD, Bloodworks Northwest, Seattle, Washington; Tobias Kurth, MD, INSERM, Bordeaux, France; Miriam Labbok, MD, Academy of Breastfeeding Medicine and University of North Carolina, Chapel Hill, North Carolina; Reagan McDonald-Mosley, MD, Planned Parenthood Federation of America, New York, New York; Joan Meek, MD, American Academy of Pediatrics and Florida State University College of Medicine, Orlando, Florida; Mark Mirochnick, MD, Boston University, Boston, Massachusetts; Susan Moskosky, MS, U.S. Department of Health and Human Services, Rockville, Maryland; Kavita Nanda, MD, FHI360, Durham, North Carolina; Jeffrey Peipert, MD, Washington University, St. Louis, Missouri; Michael Policar, MD, University of California, San Francisco, California; Sarah Prager, MD, University of Washington, Seattle Washington; David Soper, MD, Medical University of South Carolina, Charleston, South Carolina; Lisa Soule, MD, Food and Drug Administration, Rockville, Maryland; Alison Stuebe, MD, American College of Obstetricians and Gynecologist and University of North Carolina, Chapel Hill, North Carolina; James Trussell, PhD, Princeton University, Princeton, New Jersey; Seth Walker, MD, Emory University, Atlanta, Georgia; Nanette Wenger, MD, Emory University, Atlanta, Georgia; Carolyn Westhoff, MD, Columbia University, New York, New York; Christopher Zahn, MD, American College of Obstetricians and Gynecologists, Washington, DC.

CDC Attendees

Wanda D. Barfield, MD, Peter Briss, MD, W. Craig Hooper, PhD, Jill Huppert, MD, Caroline King, PhD, Michele Mandel, Titilope Oduyebo, MD, Cria Perrine, MD, Sam Posner, PhD.

External Reviewers

Abigail R.A. Aiken, PhD, Princeton University, Princeton, New Jersey; Linda Burdette, PA-C, Premier Women's Health of Yakima, Yakima, Washington; Mimi Zieman, MD, Planned Parenthood Southeast, Atlanta, Georgia.

Conflicts of Interest for Invited Meeting Participants, August 26–28, 2015, Atlanta, Georgia

Rebecca Allen, Nexplanon trainer for Merck and Liletta trainer for Actavis, consultant, advisory board and education grant from Bayer; Mitchell D. Creinin, Nexplanon trainer for Merck, litigation consultant for Bayer, advisory board for Merck and Teva Pharmaceutical Industries, Ltd., consultant for Lemonaid – PolkaDoc app, research support to University of California, Davis from Medicines360, Contramed, Merck, Eunice Kennedy Shriver National Institute of Child Health and Human Development, and Society of Family Planning; Linda Dominguez, speaker for Bayer, Merck, and Actavis; Alison Edelman, royalties from Up to Date, Inc., consultant for Genzyme, grant support from the National Institutes of Health and the Gates Foundation, travel funds from the World Health Organization, grant support and honorarium from Society of Family Planning, honorarium and travel funds from Contemporary Forum, trainer for Merck, consultant for Gynuity Health Projects, honorarium from CDC, Projects In Knowledge, and American Congress of Obstetricians and Gynecologists, advisory board for Agile Therapeutics; Eve Espey, travel funds from the American Congress of Obstetricians and Gynecologists, Society for Family Planning, and U.S. Food and Drug Administration, Reproductive and Drug Advisory Committee for U.S. Food and Drug Administration, travel funds and honoraria from Wayne State University, Telluride Conference, New Mexico Department of Health Clinician Conference, Planned Parenthood National Medical Conference and Society of Family Planning, British Columbia Contraception Access Research Team Conference, and American Congress of Obstetricians and Gynecologists annual meeting; Emily Godfrey, research funding from Bayer Women's Health, Prima-Temp, and Teva Pharmaceutical Industries, Ltd., trainer for Merck and Upstream USA, grant reviewer for Fellowship of Family Planning and Society of Family Planning Research Fund; Mark Hathaway, Liletta trainer and speaker for Actavis and Medicines360, Nexplanon trainer for Merck, advisory board for Contramed and Afaxys Pharmaceuticals; Paula Hillard, consultant for American Civil Liberties Union, Advanced Health Media, CMEology, National Sleep Foundation, and Planned Parenthood Federation of America, honoraria from National Sleep Foundation, Dignity Health, CMEology, Advance Health Media, and Medscape, editorial board for Advanstar–Contemporary OB/GYN, board examiner for the American Board of Obstetrics and Gynecology, contract reviewer for the U.S. Department of Health and Human Services, editorial board for EBSCO–PEMSoft, Nexplanon trainer for Merck, scientific advisor to Proctor and Gamble, publication royalties from Wiley Blackwell Publishing; Andrew Kaunitz, advisory board participant of Allergan, Bayer, Merck, and Pfizer, clinical trial funding to University of Florida from Agile Therapeutics, Bayer, Merck; Mark Mirochnick, data and safety monitoring board for Merck and ViiV Healthcare, advisory board for Merck; Jeffrey Peipert, research funding from Bayer and Teva Pharmaceutical Industries, Ltd., advisory board for Perrigo; Michael Policar, litigation consultant for Bayer; James Trussell, advisory board for Merck and Teva Pharmaceutical Industries, Ltd., consultant for Bayer; Nanette Wenger, research grants from Alnylam Pharmaceuticals, Gilead Sciences, National Heart, Lung, and Blood Institute, Pfizer, and Society for Women's Health Research, consultant for Amgen, AstraZeneca, Gilead Sciences and Merck; Carolyn Westhoff, data and safety monitoring board for Merck and Bayer, advisory board for Agile Therapeutics, MicroChips Biotech, and Actavis, research support to Columbia University from Medicines360, León Farma, and ContraMed.

Handling Conflicts of Interest

To promote transparency, all participants were asked to disclose any potential conflicts of interest to CDC prior to the expert meeting and to report any potential conflicts of interest during the introductory portion of the expert meeting. All potential conflicts of interest are listed above. No participants were excluded from discussion based on potential conflicts of interest. CDC staff who ultimately decided and developed these recommendations have no financial interests or other relationships with the manufacturers of commercial products, suppliers of commercial services, or commercial supporters relevant to these recommendations.

References

1. Finer LB, Zolna MR. Declines in unintended pregnancy in the United States, 2008–2011. N Engl J Med 2016;374:843–52.http://dx.doi.org/10.1056/NEJMsa1506575
2. Gipson JD, Koenig MA, Hindin MJ. The effects of unintended pregnancy on infant, child, and parental health: a review of the literature. Stud Fam Plann 2008;39:18–38. http://dx.doi.org/10.1111/j.1728-4465.2008.00148.x
3. Sonfield A, Kost K. Public costs from unintended pregnancies and the role of public insurance programs in paying for pregnancy-related care: national and state estimates for 2010. New York: Guttmacher Institute; 2015.
4. World Health Organization. Medical eligibility criteria for contraceptive use. 4th ed. Geneva, Switzerland: World Health Organization; 2009.
5. CDC. U.S. medical eligibility criteria for contraceptive use, 2010. MMWR Recomm Rep 2010;59(No. RR-4).
6. Curtis KM, Jatlaoui TC, Tepper NK, et al. U.S. selected practice recommendations for contraceptive use, 2016. MMWR Recomm Rep 2016;65(No. RR-4).
7. Mohllajee AP, Curtis KM, Flanagan RG, Rinehart W, Gaffield ML, Peterson HB. Keeping up with evidence a new system for WHO's evidence-based family planning guidance. Am J Prev Med 2005;28:483–90.http://dx.doi.org/10.1016/j.amepre.2005.02.008
8. Liberati A, Altman DG, Tetzlaff J, et al. The PRISMA statement for reporting systematic reviews and meta-analyses of studies that evaluate health care interventions: explanation and elaboration. J Clin Epidemiol 2009;62:e1–34. http://dx.doi.org/10.1016/j.jclinepi.2009.06.006
9. Moher D, Liberati A, Tetzlaff J, Altman DG; PRISMA Group. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. Int J Surg 2010;8:336–41. http://dx.doi.org/10.1016/j.ijsu.2010.02.007
10. Harris RP, Helfand M, Woolf SH, et al; Methods Work Group, Third US Preventive Services Task Force. Current methods of the US Preventive Services Task Force: a review of the process. Am J Prev Med 2001;20(Suppl):21–35. http://dx.doi.org/10.1016/S0749-3797(01)00261-6
11. Horton L, Folger SG, Berry-Bibee E, Jatlaoui TC, Tepper NK, Curtis KM. Research gaps from evidence-based contraception guidance: the U.S. Medical Eligibility Criteria for Contraceptive Use, 2016, and the U.S. Selected Practice Recommendations for Contraceptive Use, 2016. Contraception. In press 2016.
12. Workowski KA, Bolan GA. Sexually transmitted diseases treatment guidelines, 2015. MMWR Recomm Rep 2015;64(No. RR-3).

Abbreviations and Acronyms

ARV = antiretroviral [therapy]
BMD = bone mineral density
BMI = body mass index
CHC = combined hormonal contraceptive
COC = combined oral contraceptive
Cu-IUD = copper-containing intrauterine device
DMPA = depot medroxyprogesterone acetate
DVT = deep venous thrombosis
ECP = emergency contraceptive pills
FAB = fertility awareness–based [methods]
hCG = human chorionic gonadotropin
HDL = high-density lipoprotein
HIV = human immunodeficiency virus
IBD = inflammatory bowel disease
IUD = intrauterine device
LARC = long-acting reversible contraception
LDL = low-density lipoprotein
LNG = levonorgestrel
LNG-IUD = levonorgestrel-releasing intrauterine device
NET-EN = norethisterone enantate
NNRTI = nonnucleoside reverse transcriptase inhibitor
NRTI = nucleoside reverse transcriptase inhibitor
PE = pulmonary embolism
PID = pelvic inflammatory disease
POC = progestin-only contraceptive
POP = progestin-only pill
SLE = systemic lupus erythematosus
SSRI = selective serotonin reuptake inhibitors
STD = sexually transmitted disease
UPA = ulipristal acetate
U.S. MEC = U.S. Medical Eligibility Criteria for Contraceptive Use
U.S. SPR = U.S. Selected Practice Recommendations for Contraceptive Use
VTE = venous thromboembolism

Appendix A

Summary of Changes from *U.S. Medical Eligibility Criteria for Contraceptive Use, 2010*

The classification additions, deletions, and modifications from the 2010 *U.S. Medical Eligibility Criteria for Contraceptive Use* (U.S. MEC) are summarized in the following tables (Box A1) (Tables A1 and A2). For conditions for which classifications changed for one or more contraceptive methods or the condition description underwent a major modification, the changes or modifications are in bold italics (Tables A1 and A2). Conditions that do not appear in this table remain unchanged from the 2010 U.S. MEC.

BOX A1. Categories for classifying intrauterine devices and hormonal contraceptives

1 = A condition for which there is no restriction for the use of the contraceptive method.
2 = A condition for which the advantages of using the method generally outweigh the theoretical or proven risks.
3 = A condition for which the theoretical or proven risks usually outweigh the advantages of using the method.
4 = A condition that represents an unacceptable health risk if the contraceptive method is used.

TABLE A1. Summary of changes in classifications from *U.S. Medical Eligibility Criteria for Contraceptive Use, 2010**

Condition	Cu-IUD	LNG-IUD	Implants	DMPA	POP	CHCs	Clarification
Breastfeeding							
a. <21 days postpartum	—	—	2	2	2	***4***	Breastfeeding provides important health benefits for mother and infant. The U.S. Department of Health and Human Services recommends increasing the proportion of infants initially breastfed, exclusively breastfed through 6 months of life, and continuing breastfeeding through at least 1 year of life as key public health goals (*1*).
b. 21 to <30 days postpartum							
i. With other risk factors for VTE (e.g., age ≥35 years, previous VTE, thrombophilia, immobility, transfusion at delivery, peripartum cardiomyopathy, BMI ≥30 kg/m², postpartum hemorrhage, postcesarean delivery, preeclampsia, or smoking)	—	—	***2***	***2***	***2***	***3***	Breastfeeding provides important health benefits for mother and infant. The U.S. Department of Health and Human Services recommends increasing the proportion of infants initially breastfed, exclusively breastfed through 6 months of life, and continuing breastfeeding through at least 1 year of life as key public health goals (*1*). ***CHCs: For women with other risk factors for VTE, these risk factors might increase the classification to a category 4.***
ii. Without other risk factors for VTE	—	—	***2***	***2***	***2***	***3***	Breastfeeding provides important health benefits for mother and infant. The U.S. Department of Health and Human Services recommends increasing the proportion of infants initially breastfed, exclusively breastfed through 6 months of life, and continuing breastfeeding through at least 1 year of life as key public health goals (*1*).
c. 30–42 days postpartum							
i. With other risk factors for VTE (e.g., age ≥35 years, previous VTE, thrombophilia, immobility, transfusion at delivery, peripartum cardiomyopathy, BMI ≥30 kg/m², postpartum hemorrhage, postcesarean delivery, preeclampsia, or smoking)	—	—	***1***	***1***	***1***	***3***	Breastfeeding provides important health benefits for mother and infant. The U.S. Department of Health and Human Services recommends increasing the proportion of infants initially breastfed, exclusively breastfed through 6 months of life, and continuing breastfeeding through at least 1 year of life as key public health goals (*1*). ***CHCs: For women with other risk factors for VTE, these risk factors might increase the classification to a category 4.***
ii. Without other risk factors for VTE	—	—	***1***	***1***	***1***	***2***	Breastfeeding provides important health benefits for mother and infant. The U.S. Department of Health and Human Services recommends increasing the proportion of infants initially breastfed, exclusively breastfed through 6 months of life, and continuing breastfeeding through at least 1 year of life as key public health goals (*1*).
d. >42 days postpartum	—	—	***1***	***1***	***1***	***2***	Breastfeeding provides important health benefits for mother and infant. The U.S. Department of Health and Human Services recommends increasing the proportion of infants initially breastfed, exclusively breastfed through 6 months of life, and continuing breastfeeding through at least 1 year of life as key public health goals (*1*).

See table footnotes on page 16.

TABLE A1. (*Continued*) Summary of changes in classifications from *U.S. Medical Eligibility Criteria for Contraceptive Use, 2010**

Condition	Cu-IUD	LNG-IUD	Implants	DMPA	POP	CHCs	Clarification
Postpartum (nonbreastfeeding women)							
a. <21 days postpartum	—	—	1	1	1	***4***	—
b. 21–42 days postpartum							
i. With other risk factors for VTE (e.g., age ≥35 years, previous VTE, thrombophilia, immobility, transfusion at delivery, peripartum cardiomyopathy, BMI ≥30 kg/m^2, postpartum hemorrhage, postcesarean delivery, preeclampsia, or smoking)	—	—	***1***	***1***	***1***	***3***	***CHCs: For women with other risk factors for VTE, these risk factors might increase the classification to a category 4.***
ii. Without other risk factors for VTE	—	—	***1***	***1***	***1***	***2***	—
c. >42 days postpartum	—	—	***1***	***1***	***1***	***1***	—
Postpartum (including cesarean delivery)							
a. <10 minutes after delivery of the placenta							***IUDs: Insertion of IUDs among postpartum women is safe and does not appear to increase health risks associated with IUD use such as infection. Higher rates of expulsion during the postpartum period should be considered as they relate to effectiveness, along with patient access to interval placement (i.e., not related to pregnancy) when expulsion rates are lower.*** ***Breastfeeding: Breastfeeding provides important health benefits for mother and infant. The U.S. Department of Health and Human Services recommends increasing the proportion of infants initially breastfed, exclusively breastfed through 6 months of life, and continuing breastfeeding through at least 1 year of life as key public health goals (1).***
i. Breastfeeding	***1***	***2***	—	—	—	—	
ii. Nonbreastfeeding	***1***	***1***	—	—	—	—	
b. 10 minutes after delivery of the placenta to <4 weeks (breastfeeding or nonbreastfeeding)	2	2	—	—	—	—	***IUDs: Insertion of IUDs among postpartum women is safe and does not appear to increase health risks associated with IUD use such as infection. Higher rates of expulsion during the postpartum period should be considered as they relate to effectiveness, along with patient access to interval placement (i.e., not related to pregnancy) when expulsion rates are lower.*** ***Breastfeeding: Breastfeeding provides important health benefits for mother and infant. The U.S. Department of Health and Human Services recommends increasing the proportion of infants initially breastfed, exclusively breastfed through 6 months of life, and continuing breastfeeding through at least 1 year of life as key public health goals (1).***
c. ≥4 weeks (breastfeeding or nonbreastfeeding)	1	1	—	—	—	—	***IUDs: Insertion of IUDs among postpartum women is safe and does not appear to increase health risks associated with IUD use such as infection. Higher rates of expulsion during the postpartum period should be considered as they relate to effectiveness, along with patient access to interval placement (i.e., not related to pregnancy) when expulsion rates are lower.*** ***Breastfeeding: Breastfeeding provides important health benefits for mother and infant. The U.S. Department of Health and Human Services recommends increasing the proportion of infants initially breastfed, exclusively breastfed through 6 months of life, and continuing breastfeeding through at least 1 year of life as key public health goals (1).***
d. Postpartum sepsis	4	4	—	—	—	—	—

See table footnotes on page 16.

TABLE A1. (*Continued*) Summary of changes in classifications from *U.S. Medical Eligibility Criteria for Contraceptive Use, 2010**

Condition	Cu-IUD	LNG-IUD	Implants	DMPA	POP	CHCs	Clarification
Multiple risk factors for atherosclerotic cardiovascular disease (e.g., older age, smoking, diabetes, hypertension, low HDL, high LDL, or high triglyceride levels)	1	2	2	3	2	3/4	**Implants, DMPA, POP:** When multiple major risk factors exist, risk for cardiovascular disease might increase substantially. Certain POCs might increase the risk for thrombosis, although this increase is substantially less than with COCs. The effects of DMPA might persist for some time after discontinuation. **CHCs:** When a woman has multiple major risk factors, any of which alone would substantially increase her risk for cardiovascular disease, use of CHCs might increase her risk to an unacceptable level. However, a simple addition of categories for multiple risk factors is not intended; for example, a combination of two category 2 risk factors might not necessarily warrant a higher category. **Implants, DMPA, POP, CHCs:** The recommendations apply to known preexisting medical conditions or characteristics. Few if any screening tests are needed before initiation of contraception. See the *U.S. Selected Practice Recommendations for Contraceptive Use* (http://www.cdc.gov/reproductivehealth/unintendedpregnancy/usspr.htm)
Superficial venous disorders							
a. Varicose veins	1	1	1	1	1	1	—
b. Superficial venous thrombosis (acute or history)	1	1	1	1	1	***3***	***CHCs: Superficial venous thrombosis might be associated with an increased risk for VTE. If a woman has risk factors for concurrent DVT (e.g., known thrombophilia or cancer) or has current or history of DVT, see recommendations for DVT/PE. Superficial venous thrombosis associated with a peripheral intravenous catheter is less likely to be associated with additional thrombosis and use of CHCs may be considered.***
Headaches							
a. Nonmigraine (mild or severe)	1	1	1	1	1	***1***	**CHCs:** Classification depends on accurate diagnosis of those severe headaches that are migraines and those headaches that are not, as well as diagnosis of ever experiencing aura. Aura is a specific focal neurologic symptom. For more information about headache classification see *The International Headache Classification, 3rd edition* (http://www.ihs-classification.org/_downloads/mixed/International-Headache-Classification-III-ICHD-III-2013-Beta.pdf). Any new headaches or marked changes in headaches should be evaluated.
b. Migraine							
i. Without aura (This category of migraine includes menstrual migraine.)	1	***1***	***1***	***1***	***1***	***2***	**CHCs:** Classification depends on accurate diagnosis of those severe headaches that are migraines and those headaches that are not, as well as diagnosis of ever experiencing aura. Aura is a specific focal neurologic symptom. For more information about headache classification see *The International Headache Society Classification, 3rd edition* (http://www.ihs-classification.org/_downloads/mixed/International-Headache-Classification-III-ICHD-III-2013-Beta.pdf). Any new headaches or marked changes in headaches should be evaluated. **CHCs:** Classification is for women without any other risk factors for stroke (e.g., age, hypertension, and smoking).
ii. With aura	1	***1***	***1***	***1***	***1***	4	
Multiple sclerosis							
a. With prolonged immobility	***1***	***1***	***1***	***2***	***1***	***3***	—
b. Without prolonged immobility	***1***	***1***	***1***	***2***	***1***	***1***	—
Gestational trophoblastic disease This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).							***For all subconditions of gestational trophoblastic disease, classifications are based on the assumption that women are under close medical supervision because of the need for monitoring of β-hCG levels for appropriate disease surveillance.***
a. Suspected gestational trophoblastic disease (immediate postevacuation)							***For all subconditions of gestational trophoblastic disease, classifications are based on the assumption that women are under close medical supervision because of the need for monitoring of β-hCG levels for appropriate disease surveillance.***
i. Uterine size first trimester	***1***	***1***	***1***	***1***	***1***	***1***	
ii. Uterine size second trimester	***2***	***2***	***1***	***1***	***1***	***1***	

See table footnotes on page 16.

TABLE A1. (*Continued*) Summary of changes in classifications from *U.S. Medical Eligibility Criteria for Contraceptive Use, 2010**

Condition	Cu-IUD		LNG-IUD		Implants	DMPA	POP	CHCs	Clarification
b. Confirmed gestational trophoblastic disease (after initial evacuation and during monitoring)	Initiation	Continuation	Initiation	Continuation					
i. Undetectable/nonpregnant β-hCG levels	***1***	***1***	***1***	***1***	***1***	***1***	***1***	***1***	***For all subconditions of gestational trophoblastic disease, classifications are based on the assumption that women are under close medical supervision because of the need for monitoring of β-hCG levels for appropriate disease surveillance.***
ii. Decreasing β-hCG levels	***2***	***1***	***2***	***1***	***1***	***1***	***1***	***1***	***For all subconditions of gestational trophoblastic disease, classifications are based on the assumption that women are under close medical supervision because of the need for monitoring of β-hCG levels for appropriate disease surveillance.***
									IUD: For women at higher risk for disease progression, the benefits of effective contraception must be weighed against the potential need for early IUD removal.
iii. Persistently elevated β-hCG levels or malignant disease, with no evidence or suspicion of intrauterine disease	***2***	***1***	***2***	***1***	***1***	***1***	***1***	***1***	***For all subconditions of gestational trophoblastic disease, classifications are based on the assumption that women are under close medical supervision because of the need for monitoring of β-hCG levels for appropriate disease surveillance.***
iv. Persistently elevated β-hCG levels or malignant disease, with evidence or suspicion of intrauterine disease	***4***	***2***	***4***	***2***	***1***	***1***	***1***	***1***	***For all subconditions of gestational trophoblastic disease, classifications are based on the assumption that women are under close medical supervision because of the need for monitoring of β-hCG levels for appropriate disease surveillance.***
Sexually transmitted diseases	Initiation	Continuation	Initiation	Continuation					
a. Current purulent cervicitis or chlamydial infection or gonococcal infection	4	2	4	2	1	1	1	1	**IUD continuation:** Treat the STD using appropriate antibiotics. The IUD usually does not need to be removed if the woman wants to continue using it. Continued use of an IUD depends on the woman's informed choice and her current risk factors for STDs and PID.
b. Vaginitis (including *Trichomonas vaginalis* and bacterial vaginosis)	2	2	2	2	1	1	1	1	—
c. Other factors related to STDs	***2***	***2***	***2***	***2***	1	1	1	1	***IUD initiation: Most women do not require additional STD screening at the time of IUD insertion. If a woman with risk factors for STDs has not been screened for gonorrhea and chlamydia according to CDC STD treatment guidelines (2), screening may be performed at the time of IUD insertion and insertion should not be delayed.***
High risk for HIV	Initiation 2	Continuation 2	Initiation 2	Continuation 2	1	1	1	1	***DMPA: Some studies suggest that women using progestin-only injectable contraception might be at increased risk for HIV acquisition; other studies do not show this association. CDC reviewed all available evidence and agreed that the data were not sufficiently conclusive to change current guidance. However, because of the inconclusive nature of the body of evidence on possible increased risk for HIV acquisition, women using progestin-only injectable contraception should be strongly advised to also always use condoms (male or female) and take other HIV preventive measures. Expansion of contraceptive method mix and further research on the relationship between hormonal contraception and HIV infection are essential. These recommendations will be continually reviewed in light of new evidence.***
HIV infection ***For women with HIV infection who are not clinically well or not receiving ARV therapy, this condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).***	—	—	—	—	1	1	1	1	**Implants, DMPA, POP, CHCs:** Drug interactions might exist between hormonal contraceptives and ARV drugs; see Drug Interactions section.
a. Clinically well receiving ARV therapy	***1***	***1***	***1***	***1***	—	—	—	—	—
b. Not clinically well or not receiving ARV therapy	***2***	***1***	***2***	***1***	—	—	—	—	—

See table footnotes on page 16.

TABLE A1. (*Continued*) Summary of changes in classifications from *U.S. Medical Eligibility Criteria for Contraceptive Use, 2010**

Condition	Cu-IUD		LNG-IUD		Implants	DMPA	POP	CHCs	Clarification
Cystic fibrosis *This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).*	*1*		*1*		*1*	*2*	*1*	*1*	***Persons with cystic fibrosis are at increased risk for diabetes, liver disease, gallbladder disease, and VTE (particularly related to use of central venous catheters) and are frequently prescribed antibiotics. Categories assigned to such conditions in U.S. MEC should be the same for women with cystic fibrosis who have these conditions. For cystic fibrosis, classifications are based on the assumption that no other conditions are present; these classifications must be modified in the presence of such conditions.*** ***Implants, DMPA, POP, CHCs: Certain drugs to treat cystic fibrosis (e.g., lumacaftor) might reduce effectiveness of hormonal contraceptives, including oral, injectable, transdermal, and implantable contraceptives.***
Antiretroviral therapy	Initiation	Continuation	Initiation	Continuation					**IUD:** No known interaction exists between ARV therapy and IUD use. However, IUD insertion is classified as category 2 if the woman is not clinically well or not receiving ARV therapy. Otherwise, both insertion and continuation are classified as category 1 (see HIV Infection section).
a. Nucleoside reverse transcriptase inhibitors (NRTIs)									
i. Abacavir (ABC)	*1/2*	*1*	*1/2*	*1*	*1*	*1*	*1*	*1*	—
ii. Tenofovir (TDF)	*1/2*	*1*	*1/2*	*1*	*1*	*1*	*1*	*1*	—
iii. Zidovudine (AZT)	*1/2*	*1*	*1/2*	*1*	*1*	*1*	*1*	*1*	—
iv. Lamivudine (3TC)	*1/2*	*1*	*1/2*	*1*	*1*	*1*	*1*	*1*	—
v. Didanosine (DDI)	*1/2*	*1*	*1/2*	*1*	*1*	*1*	*1*	*1*	—
vi. Emtricitabine (FTC)	*1/2*	*1*	*1/2*	*1*	*1*	*1*	*1*	*1*	—
vii. Stavudine (D4T)	*1/2*	*1*	*1/2*	*1*	*1*	*1*	*1*	*1*	—
b. Nonnucleoside reverse transcriptase inhibitors (NNRTIs)									
i. Efavirenz (EFV)	*1/2*	*1*	*1/2*	*1*	*2*	*1*	*2*	*2*	***Implants, DMPA, POP, CHCs: Evidence suggests drug interactions between efavirenz and certain hormonal contraceptives. These interactions might reduce the effectiveness of the hormonal contraceptive.***
ii. Etravirine (ETR)	*1/2*	*1*	*1/2*	*1*	*1*	*1*	*1*	*1*	—
iii. Nevirapine (NVP)	*1/2*	*1*	*1/2*	*1*	*1*	*1*	*1*	*1*	—
iv. Rilpivirine (RPV)	*1/2*	*1*	*1/2*	*1*	*1*	*1*	*1*	*1*	—
c. Ritonavir-boosted protease inhibitors									
i. Ritonavir-boosted atazanavir (ATV/r)	*1/2*	*1*	*1/2*	*1*	*2*	*1*	*2*	*2*	***Implants, DMPA, POP: Theoretically, drug interactions might occur between certain ritonavir-boosted protease inhibitors and certain hormonal contraceptives that might reduce the effectiveness of the hormonal contraceptive. Any potential effect on contraceptive effectiveness is likely to be lower with DMPA than with other POCs because of the higher dose of DMPA.*** ***CHCs: Theoretically, drug interactions might occur between certain ritonavir-boosted protease inhibitors and certain hormonal contraceptives that might reduce the effectiveness of the hormonal contraceptive.***
ii. Ritonavir-boosted darunavir (DRV/r)	*1/2*	*1*	*1/2*	*1*	*2*	*1*	*2*	*2*	***Implants, DMPA, POP: Theoretically, drug interactions might occur between certain ritonavir-boosted protease inhibitors and certain hormonal contraceptives that might reduce the effectiveness of the hormonal contraceptive. Any potential effect on contraceptive effectiveness is likely to be lower with DMPA than with other POCs because of the higher dose of DMPA.*** ***CHCs: Theoretically, drug interactions might occur between certain ritonavir-boosted protease inhibitors and certain hormonal contraceptives that might reduce the effectiveness of the hormonal contraceptive.***

See table footnotes on page 16.

TABLE A1. (*Continued*) Summary of changes in classifications from *U.S. Medical Eligibility Criteria for Contraceptive Use, 2010**

Condition	Cu-IUD		LNG-IUD		Implants	DMPA	POP	CHCs	Clarification
iii. Ritonavir-boosted fosamprenavir (FPV/r)	*1/2*	*1*	*1/2*	*1*	*2*	*1*	*2*	*2*	*Implants, DMPA, POP: Theoretically, drug interactions might occur between certain ritonavir-boosted protease inhibitors and certain hormonal contraceptives that might reduce the effectiveness of the hormonal contraceptive. Any potential effect on contraceptive effectiveness is likely to be lower with DMPA than with other POCs because of the higher dose of DMPA.* *CHCs: Theoretically, drug interactions might occur between certain ritonavir-boosted protease inhibitors and certain hormonal contraceptives that might reduce the effectiveness of the hormonal contraceptive.*
iv. Ritonavir-boosted lopinavir (LPV/r)	*1/2*	*1*	*1/2*	*1*	*1*	*1*	*1*	*1*	—
v. Ritonavir-boosted saquinavir (SQV/r)	*1/2*	*1*	*1/2*	*1*	*2*	*1*	*2*	*2*	*Implants, DMPA, POP: Theoretically, drug interactions might occur between certain ritonavir-boosted protease inhibitors and certain hormonal contraceptives that might reduce the effectiveness of the hormonal contraceptive. Any potential effect on contraceptive effectiveness is likely to be lower with DMPA than with other POCs because of the higher dose of DMPA.* *CHCs: Theoretically, drug interactions might occur between certain ritonavir-boosted protease inhibitors and certain hormonal contraceptives that might reduce the effectiveness of the hormonal contraceptive.*
vi. Ritonavir-boosted tipranavir (TPV/r)	*1/2*	*1*	*1/2*	*1*	*2*	*1*	*2*	*2*	*Implants, DMPA, POP: Theoretically, drug interactions might occur between certain ritonavir-boosted protease inhibitors and certain hormonal contraceptives that might reduce the effectiveness of the hormonal contraceptive. Any potential effect on contraceptive effectiveness is likely to be lower with DMPA than with other POCs because of the higher dose of DMPA.* *CHCs: Theoretically, drug interactions might occur between certain ritonavir-boosted protease inhibitors and certain hormonal contraceptives that might reduce the effectiveness of the hormonal contraceptive.*
d. Protease inhibitors without ritonavir									
i. Atazanavir (ATV)	*1/2*	*1*	*1/2*	*1*	*1*	*1*	*1*	*2*	*CHCs: Theoretical concern exists that increased levels of ethinyl estradiol because of interactions with ATV might increase the risk for adverse events.*
ii. Fosamprenavir (FPV)	*1/2*	*1*	*1/2*	*1*	*2*	*2*	*2*	*3*	*Implants, DMPA, POP: Theoretical concern exists that interactions between FPV and hormonal contraceptives leading to decreased levels of FPV might diminish effectiveness of the antiretroviral drug. The drug interaction likely involves CYP3A4 pathways; POCs have less effect on CYP3A4 enzymes than CHCs.* *CHCs: Concern exists that interactions between FPV and hormonal contraceptives leading to decreased levels of FPV might diminish effectiveness of the antiretroviral drug.*
iii. Indinavir (IDV)	*1/2*	*1*	*1/2*	*1*	*1*	*1*	*1*	*1*	—
iv. Nelfinavir (NFV)	*1/2*	*1*	*1/2*	*1*	*2*	*1*	*2*	*2*	*Implants, DMPA, POP: Theoretically, drug interactions might occur between certain protease inhibitors and certain hormonal contraceptives that might reduce the effectiveness of the hormonal contraceptive. Any potential effect on contraceptive effectiveness is likely to be lower with DMPA than with other POCs because of the higher dose of DMPA. Concern exists that interactions between NFV and POCs might decrease NFV levels.* *CHCs: Evidence suggests drug interactions between certain protease inhibitors and certain hormonal contraceptives. These interactions might reduce the effectiveness of the hormonal contraceptive.*

See table footnotes on page 16.

TABLE A1. (*Continued*) Summary of changes in classifications from *U.S. Medical Eligibility Criteria for Contraceptive Use, 2010

Condition	Cu-IUD		LNG-IUD		Implants	DMPA	POP	CHCs	Clarification
e. CCR5 co-receptor antagonists									
i. Maraviroc (MVC)	***1/2***	***1***	***1/2***	***1***	***1***	***1***	***1***	***1***	—
f. HIV integrase strand transfer inhibitors									
i. Raltegravir (RAL)	***1/2***	***1***	***1/2***	***1***	***1***	***1***	***1***	***1***	—
ii. Dolutegravir (DTG)	***1/2***	***1***	***1/2***	***1***	***1***	***1***	***1***	***1***	—
iii. Elvitegravir (EVG)	***1/2***	***1***	***1/2***	***1***	***1***	***1***	***1***	***1***	—
g. Fusion inhibitors									
i. Enfuvirtide	***1/2***	***1***	***1/2***	***1***	***1***	***1***	***1***	***1***	—
Psychotropic medications									
a. SSRIs		***1***		***1***	***1***	***1***	***1***	***1***	—
St. John's wort		***1***		***1***	***2***	***1***	***2***	***2***	—

Abbreviations: ARV = antiretroviral; BMI = body mass index; CHC = combined hormonal contraceptive; COC = combined oral contraceptive; Cu-IUD = copper-containing intrauterine device; DMPA = depot medroxyprogesterone acetate; DVT = deep venous thrombosis; hCG = human chorionic gonadotropin; HDL = high-density lipoprotein; HIV = human immunodeficiency virus; LDL = low-density lipoprotein; LNG-IUD = levonorgestrel-releasing intrauterine device; PE = pulmonary embolism; PID = pelvic inflammatory disease; POC = progestin-only contraceptive; POP = progestin-only pill; SSRI = selective serotonin uptake inhibitor; STD = sexually transmitted disease; VTE = venous thromboembolism.

* For conditions for which classification changed for one or more contraceptive methods or the condition description underwent a major modification, the changes or modifications are in bold italics.

TABLE A2. Summary of changes for emergency contraception from *U.S. Medical Eligibility Criteria for Contraceptive Use, 2010

Condition	Category				Clarification
	Cu-IUD	UPA	LNG	COC	
Pregnancy	4	***NA***	NA	NA	***IUD: The IUD is not indicated during pregnancy and should not be used because of the risk for serious pelvic infection and septic spontaneous abortion.*** ***ECPs: Although this method is not indicated for a woman with a known or suspected pregnancy, no harm to the woman, the course of her pregnancy, or the fetus if ECPs are inadvertently used is known to exist.***
Breastfeeding	***1***	***1***	1	1	***UPA: Breastfeeding is not recommended for 24 hours after taking UPA because it is excreted in breast milk with highest concentrations in the first 24 hours, and maximum maternal serum levels are reached 1-3 hours after administration. Mean UPA concentrations in breast milk decrease markedly from 0 to 24–48 hours and then slowly decrease over 5 days (3). Breast milk should be expressed and discarded for 24 hours after taking UPA.***
Past ectopic pregnancy	***1***	***1***	1	1	—
History of bariatric surgery This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).					
a. Restrictive procedures: decrease storage capacity of the stomach (vertical banded gastroplasty, laparoscopic adjustable gastric band, or laparoscopic sleeve gastrectomy)	***1***	***1***	1	1	—
b. Malabsorptive procedures: decrease absorption of nutrients and calories by shortening the functional length of the small intestine (Roux-en-Y gastric bypass or biliopancreatic diversion)	***1***	***1***	1	1	—
History of severe cardiovascular disease (ischemic heart disease, cerebrovascular attack, or other thromboembolic conditions) This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	***1***	***2***	2	2	—
Rheumatoid arthritis					
a. Receiving immunosuppressive therapy	***2***	***1***	1	1	—
b. Not receiving immunosuppressive therapy	***1***	***1***	1	1	—
Migraine	***1***	***1***	***1***	2	—
Inflammatory bowel disease (ulcerative colitis or Crohn's disease)	***1***	***1***	1	1	—
Severe liver disease (including jaundice) This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	***1***	***2***	2	2	—

See table footnotes on page 17.

TABLE A2. (*Continued*) Summary of changes for emergency contraception from *U.S. Medical Eligibility Criteria for Contraceptive Use, 2010**

Condition	Category				Clarification
	Cu-IUD	UPA	LNG	COC	
Solid organ transplantation This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).					
a. Complicated: graft failure (acute or chronic), rejection, or cardiac allograft vasculopathy	***3***	***1***	1	1	—
b. Uncomplicated	***2***	***1***	1	1	—
Repeated ECP use	***1***	***1***	1	1	**ECPs:** Recurrent ECP use is an indication that the woman requires further counseling about other contraceptive options. Frequently repeated ECP use might be harmful for women with conditions classified as 2, 3, or 4 for CHC or POC use.
Sexual assault	***2***	***1***	1	1	***IUD: Women who have experienced sexual assault are at increased risk for STDs. According to CDC STD treatment guidelines, routine presumptive treatment of chlamydia, gonorrhea, and trichomonas is recommended after sexual assault (2). Women with current purulent cervicitis or chlamydial infection or gonococcal infection should not undergo IUD insertion (category 4).***
Obesity (BMI ≥30 kg/m^2)	***1***	***2***	***2***	***2***	***ECPs: ECPs might be less effective among women with BMI ≥30 kg/m^2 than among women with BMI <25 kg/m^2. Despite this, no safety concerns exist.***
CYP3A4 inducers (e.g., bosentan, carbamazepine, felbamate, griseofulvin, oxcarbazepine, phenytoin, rifampin, St. John's wort, topiramate, efavirenz, and lumacaftor)	***1***	***2***	***2***	***2***	***ECPs: Strong CYP3A4 inducers might reduce the effectiveness of ECPs.***

Abbreviations: BMI = body mass index; CHC = combined hormonal contraceptive; COC = combined oral contraceptive; Cu-IUD = copper-containing intrauterine device; ECP = emergency contraceptive pill; IUD = intrauterine device; LNG = levonorgestrel; NA = not applicable; POC = progestin-only contraceptive; STD = sexually transmitted disease; UPA = ulipristal acetate.
* For conditions for which classification changed for one or more contraceptive methods or the condition description underwent a major modification, the changes or modifications are in bold italics.

References

1. US Department of Health and Human Services. Healthy people 2020: maternal, infant, and child health objectives. Washington, DC: US Department of Health and Human Services; 2015. http://www.healthypeople.gov/2020/topics-objectives/topic/maternal-infant-and-child-health/objectives
2. Workowski KA, Bolan GA. Sexually transmitted diseases treatment guidelines, 2015. MMWR Recomm Rep 2015;64(No. RR-03).
3. Watson Pharmaceuticals. Ella [Prescribing information]. Morristown, NJ; 2010. https://www.accessdata.fda.gov/drugsatfda_docs/label/2010/022474s000lbl.pdf

Appendix B

Classifications for Intrauterine Devices

Classifications for intrauterine devices (IUDs) are for the copper-containing IUD and levonorgestrel-releasing IUD (containing a total of either 13.5 mg or 52 mg levonorgestrel) (Box B1) (Table B1). IUDs do not protect against sexually transmitted diseases (STDs), including human immunodeficiency virus (HIV), and women using these methods should be counseled that consistent and correct use of the male latex condom reduces the risk for transmission of HIV and other STDs. Use of female condoms can provide protection from transmission of STDs, although data are limited.

BOX B1. Categories for classifying intrauterine devices

1 = A condition for which there is no restriction for the use of the contraceptive method.
2 = A condition for which the advantages of using the method generally outweigh the theoretical or proven risks.
3 = A condition for which the theoretical or proven risks usually outweigh the advantages of using the method.
4 = A condition that represents an unacceptable health risk if the contraceptive method is used.

TABLE B1. Classifications for intrauterine devices, including the copper-containing intrauterine device and levonorgestrel-releasing intrauterine device

Condition	Category		Clarifications/Evidence/Comments
	Cu-IUD	LNG-IUD	
Personal Characteristics and Reproductive History			
Pregnancy	4	4	**Clarification:** The IUD is not indicated during pregnancy and should not be used because of the risk for serious pelvic infection and septic spontaneous abortion.
Age			
a. Menarche to <20 years	2	2	**Comment:** Concern exists both about the risk for expulsion from nulliparity and for STDs from sexual behavior in younger age groups.
b. ≥20 years	1	1	—
Parity			
a. Nulliparous	2	2	**Evidence:** Data conflict about whether IUD use is associated with infertility among nulliparous women, although well-conducted studies suggest no increased risk (*1–9*).
b. Parous	1	1	—
Postpartum (including cesarean delivery)			
a. <10 minutes after delivery of the placenta			**Clarification:** Insertion of IUDs among postpartum women is safe and does not appear to increase health risks associated with IUD use such as infection. Higher rates of expulsion during the postpartum period should be considered as they relate to effectiveness, along with patient access to interval placement (i.e., not related to pregnancy) when expulsion rates are lower.
i. Breastfeeding	1	2	**Clarification (breastfeeding):** Breastfeeding provides important health benefits for mother and infant. The U.S. Department of Health and Human Services recommends increasing the proportion of infants initially breastfed, exclusively breastfed through 6 months of life, and continuing breastfeeding through at least 1 year of life as key public health goals (*10*).
ii. Nonbreastfeeding	1	1	**Evidence:** Studies suggest that immediate postplacental (<10 minutes) and early postpartum (10 minutes up until 72 hours) placement of Cu-IUDs and LNG-IUDs is associated with increased risk for expulsion compared with interval placement (i.e., not related to pregnancy). Early postpartum placement has similar or increased risk for expulsion compared with immediate postplacental placement. Although immediate postplacental placement at the time of cesarean delivery might have increased risk for expulsion compared with interval placement, risk appears lower than that for placement at the time of vaginal delivery. Evidence for infection, perforation, and removals for pain or bleeding are limited; however, these events are rare (*11–62*).
			Evidence (breastfeeding): Two randomized controlled trials found conflicting results on breastfeeding outcomes when LNG-IUDs were initiated immediately postpartum compared with 6–8 weeks postpartum. Initiation of LNG-IUDs immediately postpartum had no other harmful effect on infant health, growth, or development (*63*,*64*). Breastfeeding women using IUDs do not have an increased risk for certain IUD-related adverse events including expulsion, infection, pain, or bleeding compared with nonbreastfeeding women. The risk for perforation is increased independently among breastfeeding women and among women ≤36 weeks postpartum, compared with non-postpartum women; however, the absolute risk for perforation remains low (*11–62*,*65*).
			Comment (breastfeeding): Certain women might be at risk for breastfeeding difficulties, such as women with previous breastfeeding difficulties, certain medical conditions, or certain perinatal complications and those who deliver preterm. For these women, as for all women, discussions about contraception for breastfeeding women should include information about risks, benefits, and alternatives.

See table footnotes on page 30.

TABLE B1. (*Continued*) Classifications for intrauterine devices, including the copper-containing intrauterine device and levonorgestrel-releasing intrauterine device

Condition	Category		Clarifications/Evidence/Comments
	Cu-IUD	LNG-IUD	
b. 10 minutes after delivery of the placenta to <4 weeks (breastfeeding or nonbreastfeeding)	2	2	**Clarification:** Insertion of IUDs among postpartum women is safe and does not appear to increase health risks associated with IUD use such as infection. Higher rates of expulsion during the postpartum period should be considered as they relate to effectiveness, along with patient access to interval placement (i.e., not related to pregnancy) when expulsion rates are lower. **Clarification (breastfeeding):** Breastfeeding provides important health benefits for mother and infant. The U.S. Department of Health and Human Services recommends increasing the proportion of infants initially breastfed, exclusively breastfed through 6 months of life, and continuing breastfeeding through at least 1 year of life as key public health goals (*10*). **Evidence:** Studies suggest that immediate postplacental (<10 minutes) and early postpartum (10 minutes up until 72 hours) placement of Cu-IUDs and LNG-IUDs is associated with increased risk for expulsion compared with interval placement (i.e., not related to pregnancy). Early postpartum placement has similar or increased risk for expulsion compared with immediate postplacental placement. Although immediate postplacental placement at the time of cesarean delivery might have increased risk for expulsion compared with interval placement, risk appears lower than that for placement at the time of vaginal delivery. Evidence for infection, perforation, and removals for pain or bleeding are limited; however, these events are rare (*11*–*62*). **Evidence (breastfeeding):** Two randomized controlled trials found conflicting results on breastfeeding outcomes when LNG-IUDs were initiated immediately postpartum compared with 6–8 weeks postpartum. Initiation of LNG-IUDs immediately postpartum had no other harmful effect on infant health, growth, or development (*63*,*64*). Breastfeeding women using IUDs do not have an increased risk for certain IUD-related adverse events including expulsion, infection, pain, or bleeding compared with nonbreastfeeding women. The risk for perforation is increased independently among breastfeeding women and among women ≤36 weeks postpartum, compared with non-postpartum women; however, the absolute risk for perforation remains low (*11*–*62*,*65*). **Comment (breastfeeding):** Certain women might be at risk for breastfeeding difficulties, such as women with previous breastfeeding difficulties, certain medical conditions, or certain perinatal complications and those who deliver preterm. For these women, as for all women, discussions about contraception for breastfeeding women should include information about risks, benefits, and alternatives.
c. ≥4 weeks (breastfeeding or nonbreastfeeding)	1	1	**Clarification:** Insertion of IUDs among postpartum women is safe and does not appear to increase health risks associated with IUD use such as infection. Higher rates of expulsion during the postpartum period should be considered as they relate to effectiveness, along with patient access to interval placement (i.e., not related to pregnancy) when expulsion rates are lower. **Clarification (breastfeeding):** Breastfeeding provides important health benefits for mother and infant. The U.S. Department of Health and Human Services recommends increasing the proportion of infants initially breastfed, exclusively breastfed through 6 months of life, and continuing breastfeeding through at least 1 year of life as key public health goals (*10*). **Evidence (breastfeeding):** Initiation of LNG-IUDs at 4 weeks postpartum or later demonstrated no detrimental effect on breastfeeding outcomes and no harmful effect on infant health, growth, or development (*63*,*64*). Breastfeeding women using IUDs do not have an increased risk for certain IUD-related adverse events including expulsion, infection, pain, or bleeding compared with nonbreastfeeding women. The risk for perforation is increased independently among breastfeeding women and among women ≤36 weeks postpartum, compared with non-postpartum women; however, the absolute risk for perforation remains low (*11*–*62*,*65*). **Comment (breastfeeding):** Certain women might be at risk for breastfeeding difficulties, such as women with previous breastfeeding difficulties, certain medical conditions, or certain perinatal complications and those who deliver preterm. For these women, as for all women, discussions about contraception for breastfeeding women should include information about risks, benefits, and alternatives.
d. Postpartum sepsis	4	4	**Comment:** Theoretical concern exists that postpartum insertion of an IUD in a women with recent chorioamnionitis or current endometritis might be associated with increased complications.

See table footnotes on page 30.

TABLE B1. (*Continued*) Classifications for intrauterine devices, including the copper-containing intrauterine device and levonorgestrel-releasing intrauterine device

Condition	Category		Clarifications/Evidence/Comments
	Cu-IUD	LNG-IUD	
Postabortion			
a. First trimester	1	1	**Clarification:** IUDs can be inserted immediately after spontaneous or induced abortion. **Evidence:** Risk for complications from immediate versus delayed insertion of an IUD after abortion did not differ. Expulsion was greater when an IUD was inserted after a second trimester abortion than when inserted after a first trimester abortion. Safety or expulsion for postabortion insertion of an LNG-IUD did not differ from that of a Cu-IUD (*66*).
b. Second trimester	2	2	
c. Immediate postseptic abortion	4	4	**Comment:** Insertion of an IUD might substantially worsen the condition.
Past ectopic pregnancy	1	1	**Comment:** The absolute risk for ectopic pregnancy is extremely low because of the high effectiveness of IUDs. However, when a woman becomes pregnant during IUD use, the relative likelihood of ectopic pregnancy increases substantially.
History of pelvic surgery (see Postpartum [Including Cesarean Delivery] section)	1	1	—
Smoking			
a. Age <35 years	1	1	—
b. Age ≥35 years			
i. <15 cigarettes per day	1	1	—
ii. ≥15 cigarettes per day	1	1	—
Obesity			
a. BMI ≥30 kg/m^2	1	1	—
b. Menarche to <18 years and BMI ≥30 kg/m^2	1	1	—
History of bariatric surgery This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).			
a. Restrictive procedures: decrease storage capacity of the stomach (vertical banded gastroplasty, laparoscopic adjustable gastric band, or laparoscopic sleeve gastrectomy)	1	1	—
b. Malabsorptive procedures: decrease absorption of nutrients and calories by shortening the functional length of the small intestine (Roux-en-Y gastric bypass or biliopancreatic diversion)	1	1	—
Cardiovascular Disease			
Multiple risk factors for atherosclerotic cardiovascular disease (e.g., older age, smoking, diabetes, hypertension, low HDL, high LDL, or high triglyceride levels)	1	2	—
Hypertension Systolic blood pressure ≥160 mm Hg or diastolic blood pressure ≥100 mm Hg are associated with increased risk for adverse health events as a result of pregnancy (Box 2).			
a. Adequately controlled hypertension	1	1	**Clarification:** For all categories of hypertension, classifications are based on the assumption that no other risk factors for cardiovascular disease exist. When multiple risk factors do exist, risk for cardiovascular disease might increase substantially. A single reading of blood pressure level is not sufficient to classify a woman as hypertensive.
b. Elevated blood pressure levels (properly taken measurements)			**Clarification:** For all categories of hypertension, classifications are based on the assumption that no other risk factors for cardiovascular disease exist. When multiple risk factors do exist, risk for cardiovascular disease might increase substantially. A single reading of blood pressure level is not sufficient to classify a woman as hypertensive. **Comment:** Theoretical concern exists about the effect of LNG on lipids. Use of Cu-IUDs has no restrictions.
i. Systolic 140–159 mm Hg or diastolic 90–99 mm Hg	1	1	
ii. Systolic ≥160 mm Hg or diastolic ≥100 mm Hg	1	2	

See table footnotes on page 30.

TABLE B1. (*Continued*) Classifications for intrauterine devices, including the copper-containing intrauterine device and levonorgestrel-releasing intrauterine device

Condition	Category		Clarifications/Evidence/Comments
	Cu-IUD	LNG-IUD	
c. Vascular disease	1	2	**Clarification:** For all categories of hypertension, classifications are based on the assumption that no other risk factors for cardiovascular disease exist. When multiple risk factors do exist, risk for cardiovascular disease might increase substantially. A single reading of blood pressure level is not sufficient to classify a woman as hypertensive. **Comment:** Theoretical concern exists about the effect of LNG on lipids. Use of Cu-IUDs has no restrictions
History of high blood pressure during pregnancy (when current blood pressure is measurable and normal)	1	1	—
Deep venous thrombosis/ Pulmonary embolism			
a. History of DVT/PE, not receiving anticoagulant therapy			
i. Higher risk for recurrent DVT/PE (one or more risk factors) • History of estrogen-associated DVT/PE • Pregnancy-associated DVT/PE • Idiopathic DVT/PE • Known thrombophilia, including antiphospholipid syndrome • Active cancer (metastatic, receiving therapy, or within 6 months after clinical remission), excluding nonmelanoma skin cancer • History of recurrent DVT/PE	1	2	—
ii. Lower risk for recurrent DVT/PE (no risk factors)	1	2	—
b. Acute DVT/PE	2	2	**Evidence:** No direct evidence exists on the use of POCs among women with acute DVT/PE. Although findings on the risk for venous thrombosis with the use of POCs in otherwise healthy women are inconsistent, any small increased risk is substantially less than that with COCs (*67–69*).
c. DVT/PE and established anticoagulant therapy for at least 3 months			**Evidence:** No direct evidence exists on the use of POCs among women with acute DVT/PE. Although findings on the risk for venous thrombosis with the use of POCs in otherwise healthy women are inconsistent, any small increased risk is substantially less than that with COCs (*67–69*). **Evidence:** Limited evidence indicates that insertion of the LNG-IUD does not pose major bleeding risks in women receiving chronic anticoagulant therapy (*70–73*). **Comment:** The LNG-IUD might be a useful treatment for menorrhagia in women receiving long-term anticoagulation therapy.
i. Higher risk for recurrent DVT/PE (one or more risk factors) • Known thrombophilia, including antiphospholipid syndrome • Active cancer (metastatic, receiving therapy, or within 6 months after clinical remission), excluding nonmelanoma skin cancer • History of recurrent DVT/PE	2	2	—
ii. Lower risk for recurrent DVT/PE (no risk factors)	2	2	—
d. Family history (first-degree relatives)	1	1	—
e. Major surgery			
i. With prolonged immobilization	1	2	—
ii. Without prolonged immobilization	1	1	—
f. Minor surgery without immobilization	1	1	

See table footnotes on page 30.

TABLE B1. (*Continued*) Classifications for intrauterine devices, including the copper-containing intrauterine device and levonorgestrel-releasing intrauterine device

Condition	Category				Clarifications/Evidence/Comments
	Cu-IUD		LNG-IUD		
Known thrombogenic mutations (e.g., factor V Leiden; prothrombin mutation; and protein S, protein C, and antithrombin deficiencies) This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	1		2		**Clarification:** Routine screening is not appropriate because of the rarity of the conditions and the high cost of screening.
Superficial venous disorders					
a. Varicose veins	1		1		—
b. Superficial venous thrombosis (acute or history)	1		1		—
Current and history of ischemic heart disease This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	1	Initiation 2		Continuation 3	**Comment:** Theoretical concern exists about the effect of LNG on lipids. Use of Cu-IUDs has no restrictions.
Stroke (history of cerebrovascular accident) This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	1		2		**Comment:** Theoretical concern exists about the effect of LNG on lipids. Use of Cu-IUDs has no restrictions.
Valvular heart disease Complicated valvular heart disease is a condition associated with increased risk for adverse health events as a result of pregnancy (Box 2).					**Comment**: According to the American Heart Association, administration of prophylactic antibiotics solely to prevent endocarditis is not recommended for patients who undergo genitourinary tract procedures, including insertion or removal of IUDs (*74*).
a. Uncomplicated	1		1		
b. Complicated (pulmonary hypertension, risk for atrial fibrillation, or history of subacute bacterial endocarditis)	1		1		
Peripartum cardiomyopathy This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).					**Evidence:** No direct evidence exists on the safety of IUDs among women with peripartum cardiomyopathy. Limited indirect evidence from noncomparative studies did not demonstrate any cases of arrhythmia or infective endocarditis in women with cardiac disease who used IUDs (*75*).
a. Normal or mildly impaired cardiac function (New York Heart Association Functional Class I or II: patients with no limitation of activities or patients with slight, mild limitation of activity) (*76*)					**Comment:** IUD insertion might induce cardiac arrhythmias in healthy women; women with peripartum cardiomyopathy have a high incidence of cardiac arrhythmias.
i. <6 months	2		2		
ii. ≥6 months	2		2		
b. Moderately or severely impaired cardiac function (New York Heart Association Functional Class III or IV: patients with marked limitation of activity or patients who should be at complete rest) (*76*)	2		2		
Rheumatic Diseases					
Systemic lupus erythematosus This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	Initiation	Continuation			
a. Positive (or unknown) antiphospholipid antibodies	1	1	3		**Clarification:** Persons with SLE are at increased risk for ischemic heart disease, stroke, and VTE. Categories assigned to such conditions in U.S. MEC should be the same for women with SLE who have these conditions. For all subconditions of SLE, classifications are based on the assumption that no other risk factors for cardiovascular disease are present; these classifications must be modified in the presence of such risk factors. Many women with SLE can be considered good candidates for most contraceptive methods, including hormonal contraceptives (*73*,*77*–*94*).
					Evidence: Antiphospholipid antibodies are associated with a higher risk for both arterial and venous thrombosis (*95*,*96*)

See table footnotes on page 30.

TABLE B1. (*Continued*) Classifications for intrauterine devices, including the copper-containing intrauterine device and levonorgestrel-releasing intrauterine device

Condition	Category				Clarifications/Evidence/Comments
	Cu-IUD		LNG-IUD		
b. Severe thrombocytopenia	3	2	2		**Clarification:** Persons with SLE are at increased risk for ischemic heart disease, stroke, and VTE. Categories assigned to such conditions in U.S. MEC should be the same for women with SLE who have these conditions. For all subconditions of SLE, classifications are based on the assumption that no other risk factors for cardiovascular disease are present; these classifications must be modified in the presence of such risk factors. Many women with SLE can be considered good candidates for most contraceptive methods, including hormonal contraceptives (*73*,*77*–*94*). **Clarification:** Severe thrombocytopenia increases the risk for bleeding. The category should be assessed according to the severity of thrombocytopenia and its clinical manifestations. In women with very severe thrombocytopenia who are at risk for spontaneous bleeding, consultation with a specialist and certain pretreatments might be warranted. **Evidence:** The LNG-IUD might be a useful treatment for menorrhagia in women with severe thrombocytopenia (*73*).
c. Immunosuppressive therapy	2	1	2		**Clarification:** Persons with SLE are at increased risk for ischemic heart disease, stroke, and VTE. Categories assigned to such conditions in U.S. MEC should be the same for women with SLE who have these conditions. For all subconditions of SLE, classifications are based on the assumption that no other risk factors for cardiovascular disease are present; these classifications must be modified in the presence of such risk factors. Many women with SLE can be considered good candidates for most contraceptive methods, including hormonal contraceptives (*73*,*77*–*94*).
d. None of the above	1	1	2		**Clarification:** Persons with SLE are at increased risk for ischemic heart disease, stroke, and VTE. Categories assigned to such conditions in U.S. MEC should be the same for women with SLE who have these conditions. For all subconditions of SLE, classifications are based on the assumption that no other risk factors for cardiovascular disease are present; these classifications must be modified in the presence of such risk factors. Many women with SLE can be considered good candidates for most contraceptive methods, including hormonal contraceptives (*73*,*77*–*94*).
Rheumatoid arthritis	Initiation	Continuation	Initiation	Continuation	
a. Receiving immunosuppressive therapy	2	1	2	1	—
b. Not receiving immunosuppressive therapy	1		1		—
Neurologic Conditions					
Headaches					
a. Nonmigraine (mild or severe)	1		1		—
b. Migraine					
i. Without aura (This category of migraine includes menstrual migraine.)	1		1		**Evidence:** No studies directly examined the risk for stroke among women with migraine using LNG-IUDs (*97*). Limited evidence demonstrated that women using LNG-IUDs do not have an increased risk for ischemic stroke compared with women not using hormonal contraceptives (*98*). **Comment:** Menstrual migraine is a subtype of migraine without aura. For more information see *The International Headache Society Classification, 3rd edition* (http://www.ihs-classification.org/_downloads/mixed/International-Headache-Classification-III-ICHD-III-2013-Beta.pdf).
ii. With aura	1		1		
Epilepsy This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	1		1		—
Multiple sclerosis					
a. With prolonged immobility	1		1		—
b. Without prolonged immobility	1		1		—
Depressive Disorders					
Depressive disorders	1		1		**Clarification**: If a woman is receiving psychotropic medications or St. John's wort, see Drug Interactions section. **Evidence:** The frequency of psychiatric hospitalizations for women with bipolar disorder or depression did not significantly differ among women using DMPA, LNG-IUD, Cu-IUD, or sterilization (*99*).

See table footnotes on page 30.

TABLE B1. (*Continued*) Classifications for intrauterine devices, including the copper-containing intrauterine device and levonorgestrel-releasing intrauterine device

Condition	Category				Clarifications/Evidence/Comments
	Cu-IUD		LNG-IUD		
Reproductive Tract Infections and Disorders					
Vaginal bleeding patterns			Initiation	Continuation	
a. Irregular pattern without heavy bleeding	1		1	1	—
b. Heavy or prolonged bleeding (includes regular and irregular patterns)	2		1	2	**Clarification:** Unusually heavy bleeding should raise suspicion of a serious underlying condition. **Evidence:** Evidence from studies examining the treatment effects of the LNG-IUD among women with heavy or prolonged bleeding reported no increase in adverse effects and found the LNG-IUD to be beneficial in treating menorrhagia (*100–107*).
Unexplained vaginal bleeding (suspicious for serious condition) before evaluation	Initiation 4	Continuation 2	Initiation 4	Continuation 2	**Clarification:** If pregnancy or an underlying pathological condition (e.g., pelvic malignancy) is suspected, it must be evaluated and the category adjusted after evaluation. The IUD does not need to be removed before evaluation.
Endometriosis	2		1		**Evidence:** LNG-IUD use among women with endometriosis decreased dysmenorrhea, pelvic pain, and dyspareunia (*108–112*).
Benign ovarian tumors (including cysts)	1		1		—
Severe dysmenorrhea	2		1		**Comment:** Dysmenorrhea might intensify with Cu-IUD use. LNG-IUD use has been associated with reduction of dysmenorrhea.
Gestational trophoblastic disease This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).					
a. Suspected gestational trophoblastic disease (immediate postevacuation)					**Clarification:** For all subconditions of gestational trophoblastic disease, classifications are based on the assumption that women are under close medical supervision because of the need for monitoring of β-hCG levels for appropriate disease surveillance.
i. Uterine size first trimester	1		1		
ii. Uterine size second trimester	2		2		**Evidence:** Limited evidence suggests that women using an IUD after uterine evacuation for a molar pregnancy are not at greater risk for postmolar trophoblastic disease than are women using other methods of contraception (*113*). **Comment:** The risk for expulsion immediately postevacuation for gestational trophoblastic disease is unknown. Expulsion is greater after IUD insertion immediately postevacuation for a spontaneous or induced abortion in the second trimester compared with IUD insertion after a first trimester abortion.
b. Confirmed gestational trophoblastic disease (after initial evacuation and during monitoring)	Initiation	Continuation	Initiation	Continuation	
i. Undetectable/nonpregnant β-hCG levels	1	1	1	1	**Clarification:** For all subconditions of gestational trophoblastic disease, classifications are based on the assumption that women are under close medical supervision because of the need for monitoring of β-hCG levels for appropriate disease surveillance. **Evidence:** Limited evidence suggests that women using an IUD after uterine evacuation for a molar pregnancy are not at greater risk for postmolar trophoblastic disease than are women using other methods of contraception (*113*). **Comment:** Once β-hCG levels have decreased to nonpregnant levels, the risk for disease progression is likely to be very low.
ii. Decreasing β-hCG levels	2	1	2	1	**Clarification:** For all subconditions of gestational trophoblastic disease, classifications are based on the assumption that women are under close medical supervision because of the need for monitoring of β-hCG levels for appropriate disease surveillance. **Clarification:** For women at higher risk for disease progression, the benefits of effective contraception must be weighed against the potential need for early IUD removal. **Evidence:** Limited evidence suggests that women using an IUD after uterine evacuation for a molar pregnancy are not at greater risk for postmolar trophoblastic disease than are women using other methods of contraception (*113*).

See table footnotes on page 30.

TABLE B1. (*Continued*) Classifications for intrauterine devices, including the copper-containing intrauterine device and levonorgestrel-releasing intrauterine device

Condition	Category				Clarifications/Evidence/Comments
	Cu-IUD		LNG-IUD		
iii. Persistently elevated β-hCG levels or malignant disease, with no evidence or suspicion of intrauterine disease	2	1	2	1	**Clarification:** For all subconditions of gestational trophoblastic disease, classifications are based on the assumption that women are under close medical supervision because of the need for monitoring of β-hCG levels for appropriate disease surveillance. **Evidence:** Limited evidence suggests that women using an IUD after uterine evacuation for a molar pregnancy are not at greater risk for postmolar trophoblastic disease than are women using other methods of contraception (*113*).
iv. Persistently elevated β-hCG levels or malignant disease, with evidence or suspicion of intrauterine disease	4	2	4	2	**Clarification:** For all subconditions of gestational trophoblastic disease, classifications are based on the assumption that women are under close medical supervision because of the need for monitoring of β-hCG levels for appropriate disease surveillance. **Evidence:** Limited evidence suggests that women using an IUD after uterine evacuation for a molar pregnancy are not at greater risk for postmolar trophoblastic disease than are women using other methods of contraception (*113*). **Comment:** For women with suspected or confirmed intrauterine disease, an IUD should not be inserted because of theoretical risk for perforation, infection, and hemorrhage. For women who already have an IUD in place, individual circumstance along with the benefits of effective contraception must be weighed against theoretical risks of either removal or continuation of the IUD.
Cervical ectropion		1		1	—
Cervical intraepithelial neoplasia		1		2	**Comment:** Theoretical concern exists that LNG-IUDs might enhance progression of cervical intraepithelial neoplasia.
Cervical cancer (awaiting treatment)	Initiation 4	Continuation 2	Initiation 4	Continuation 2	**Comment:** Concern exists about the increased risk for infection and bleeding at insertion. The IUD most likely will need to be removed at the time of treatment but until then, the woman is at risk for pregnancy.
Breast disease Breast cancer is associated with increased risk for adverse health events as a result of pregnancy (Box 2).					
a. Undiagnosed mass		1		2	—
b. Benign breast disease		1		1	—
c. Family history of cancer		1		1	—
d. Breast cancer					**Comment:** Breast cancer is a hormonally sensitive tumor. Concerns about progression of the disease might be less with LNG-IUDs than with COCs or higher-dose POCs.
i. Current		1		4	
ii. Past and no evidence of current disease for 5 years		1		3	
Endometrial hyperplasia		1		1	**Evidence:** Among women with endometrial hyperplasia, no adverse health events occurred with LNG-IUD use; most women experienced disease regression (*114*).
Endometrial cancer This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	Initiation 4	Continuation 2	Initiation 4	Continuation 2	**Comment:** Concern exists about the increased risk for infection, perforation, and bleeding at insertion. The IUD most likely will need to be removed at the time of treatment, but until then, the woman is at risk for pregnancy.
Ovarian cancer This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).		1		1	**Comment:** Women with ovarian cancer who undergo fertility-sparing treatment and need contraception may use an IUD.
Uterine fibroids		2		2	**Evidence:** Among women with uterine fibroids using an LNG-IUD, most experienced improvements in serum levels of hemoglobin, hematocrit, and ferritin and in menstrual blood loss (*115*). Rates of LNG-IUD expulsion were higher in women with uterine fibroids (11%) than in women without fibroids (0%–3%); these findings were either not statistically significant or significance testing was not conducted (*115*). Rates of expulsion found in noncomparative studies ranged from 0%–20% (*115*). **Comment:** Women with heavy or prolonged bleeding should be assigned the category for that condition.

See table footnotes on page 30.

TABLE B1. (*Continued*) Classifications for intrauterine devices, including the copper-containing intrauterine device and levonorgestrel-releasing intrauterine device

Condition	Category				Clarifications/Evidence/Comments
	Cu-IUD		LNG-IUD		
Anatomical abnormalities					
a. Distorted uterine cavity (any congenital or acquired uterine abnormality distorting the uterine cavity in a manner that is incompatible with IUD insertion)	4		4		**Comment:** An anatomical abnormality that distorts the uterine cavity might preclude proper IUD placement.
b. Other abnormalities (including cervical stenosis or cervical lacerations) not distorting the uterine cavity or interfering with IUD insertion	2		2		—
Pelvic inflammatory disease	Initiation	Continuation	Initiation	Continuation	
a. Past PID					**Comment:** IUDs do not protect against STDs, including HIV, or PID. In women at low risk for STDs, IUD insertion poses little risk for PID.
i. With subsequent pregnancy	1	1	1	1	
ii. Without subsequent pregnancy	2	2	2	2	
b. Current PID	4	2	4	2	**Clarification (continuation):** Treat the PID using appropriate antibiotics. The IUD usually does not need to be removed if the woman wants to continue using it. Continued use of an IUD depends on the woman's informed choice and her current risk factors for STDs and PID. **Evidence:** Among IUD users treated for PID, clinical course did not differ regardless of whether the IUD was removed or left in place (*116*).
Sexually transmitted diseases	Initiation	Continuation	Initiation	Continuation	
a. Current purulent cervicitis or chlamydial infection or gonococcal infection	4	2	4	2	**Clarification (continuation):** Treat the STD using appropriate antibiotics. The IUD usually does not need to be removed if the woman wants to continue using it. Continued use of an IUD depends on the woman's informed choice and her current risk factors for STDs and PID. **Evidence:** Among women who had an IUD inserted, the absolute risk for subsequent PID was low among women with STD at the time of insertion but greater than among women with no STD at the time of IUD insertion (*117–123*).
b. Vaginitis (including *Trichomonas vaginalis* and bacterial vaginosis)	2	2	2	2	—
c. Other factors related to STDs	2	2	2	2	**Clarification (initiation):** Most women do not require additional STD screening at the time of IUD insertion. If a woman with risk factors for STDs has not been screened for gonorrhea and chlamydia according to CDC STD treatment guidelines (*124*), screening may be performed at the time of IUD insertion and insertion should not be delayed. **Evidence:** Women who undergo same-day STD screening and IUD insertion have low incidence rates of PID. Algorithms for predicting PID among women with risk factors for STDs have poor predictive value. Risk for PID among women with risk factors for STDs is low (*125*).
HIV					
High risk for HIV	Initiation	Continuation	Initiation	Continuation	**Evidence:** Among women at risk for HIV, Cu-IUD use did not increase risk for HIV acquisition (*126–136*).
	2	2	2	2	
HIV infection For women with HIV infection who are not clinically well or not receiving ARV therapy, this condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).					**Evidence:** Among IUD users, limited evidence shows a low risk for PID among HIV-infected women using IUDs and no higher risk for pelvic infectious complications in HIV-infected than in HIV-noninfected women or among women with varying degrees of HIV severity. IUD use did not adversely affect progression of HIV during 6–45 months of follow-up or when compared with hormonal contraceptive use among HIV-infected women. Furthermore, IUD use among HIV-infected women was not associated with increased risk for transmission to sex partners or with increased genital viral shedding (*137*).
a. Clinically well receiving ARV therapy	1	1	1	1	
b. Not clinically well or not receiving ARV therapy	2	1	2	1	

See table footnotes on page 30.

TABLE B1. (*Continued*) Classifications for intrauterine devices, including the copper-containing intrauterine device and levonorgestrel-releasing intrauterine device

Condition	Category				Clarifications/Evidence/Comments
	Cu-IUD		LNG-IUD		
Other Infections					
Schistosomiasis					
Schistosomiasis with fibrosis of the liver is associated with increased risk for adverse health events as a result of pregnancy (Box 2).					
a. Uncomplicated	1		1		—
b. Fibrosis of the liver (if severe, see Cirrhosis section)	1		1		—
Tuberculosis	Initiation	Continuation	Initiation	Continuation	
This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).					
a. Nonpelvic	1	1	1	1	—
b. Pelvic	4	3	4	3	**Comment:** Insertion of an IUD might substantially worsen the condition.
Malaria	1		1		—
Endocrine Conditions					
Diabetes					
Insulin-dependent diabetes; diabetes with nephropathy, retinopathy, or neuropathy; diabetes with other vascular disease; or diabetes of >20 years' duration are associated with increased risk for adverse health events as a result of pregnancy (Box 2).					
a. History of gestational disease	1		1		—
b. Nonvascular disease					**Evidence:** Limited evidence on the use of the LNG-IUD among women with insulin-dependent or non–insulin-dependent diabetes suggests that these methods have little effect on short-term or long-term diabetes control (e.g., glycosylated hemoglobin levels), hemostatic markers, or lipid profile (*138*,*139*).
i. Non-insulin dependent	1		2		
ii. Insulin dependent	1		2		
c. Nephropathy, retinopathy, or neuropathy	1		2		—
d. Other vascular disease or diabetes of >20 years' duration	1		2		—
Thyroid disorders					
a. Simple goiter	1		1		—
b. Hyperthyroid	1		1		—
c. Hypothyroid	1		1		—
Gastrointestinal Conditions					
Inflammatory bowel disease (ulcerative colitis or Crohn's disease)	1		1		**Evidence:** Although two case reports described three women with IBD who experienced exacerbation of disease 5 days–25 months after LNG-IUD insertion, no comparative studies have examined the safety of IUD use among women with IBD (*140*).
Gallbladder disease					
a. Symptomatic					
i. Treated by cholecystectomy	1		2		—
ii. Medically treated	1		2		—
iii. Current	1		2		—
b. Asymptomatic	1		2		—
History of cholestasis					
a. Pregnancy related	1		1		—
b. Past COC related	1		2		**Comment:** Concern exists that history of COC related cholestasis might predict subsequent cholestasis with LNG use. Whether risk exists with use of LNG-IUD is unclear.
Viral hepatitis					
a. Acute or flare	1		1		—
b. Carrier	1		1		—
c. Chronic	1		1		—
Cirrhosis					
Severe cirrhosis is associated with increased risk for adverse health events as a result of pregnancy (Box 2).					
a. Mild (compensated)	1		1		—
b. Severe (decompensated)	1		3		—

See table footnotes on page 30.

TABLE B1. (*Continued*) Classifications for intrauterine devices, including the copper-containing intrauterine device and levonorgestrel-releasing intrauterine device

Condition	Category				Clarifications/Evidence/Comments
	Cu-IUD		LNG-IUD		
Liver tumors Hepatocellular adenoma and malignant liver tumors are associated with increased risk for adverse health events as a result of pregnancy (Box 2).					
a. Benign					
i. Focal nodular hyperplasia	1		2		—
ii. Hepatocellular adenoma	1		3		**Comment:** No evidence is available about hormonal contraceptive use in women with hepatocellular adenoma. COC use in healthy women is associated with development and growth of hepatocellular adenoma; whether other hormonal contraceptives have similar effects is not known.
b. Malignant (hepatoma)	1		3		—
Respiratory Conditions					
Cystic fibrosis This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	1		1		**Clarification:** Persons with cystic fibrosis are at increased risk for diabetes, liver disease, gallbladder disease, and VTE (particularly related to use of central venous catheters) and are frequently prescribed antibiotics. Categories assigned to such conditions in U.S. MEC should be the same for women with cystic fibrosis who have these conditions. For cystic fibrosis, classifications are based on the assumption that no other conditions are present; these classifications must be modified in the presence of such conditions.
Anemias					
Thalassemia	2		1		**Comment:** Concern exists about an increased risk for blood loss with Cu-IUDs.
Sickle cell disease This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	2		1		**Comment:** Concern exists about an increased risk for blood loss with Cu-IUDs.
Iron deficiency anemia	2		1		**Comment:** Concern exists about an increased risk for blood loss with Cu-IUDs.
Solid Organ Transplantation					
Solid organ transplantation This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	Initiation	Continuation	Initiation	Continuation	**Evidence:** No comparative studies have examined IUD use among transplant patients. Four case reports of transplant patients using IUDs provided inconsistent results, including beneficial effects and contraceptive failures (*141*).
a. Complicated: graft failure (acute or chronic), rejection, or cardiac allograft vasculopathy	3	2	3	2	
b. Uncomplicated	2	2	2	2	
Drug Interactions					
Antiretroviral therapy	Initiation	Continuation	Initiation	Continuation	**Clarification:** No known interaction exists between ARV therapy and IUD use. However, IUD insertion is classified as category 2 if the woman is not clinically well or not receiving ARV therapy. Otherwise, both insertion and continuation are classified as category 1 (see HIV Infection section).
a. Nucleoside reverse transcriptase inhibitors (NRTIs)					
i. Abacavir (ABC)	1/2	1	1/2	1	—
ii. Tenofovir (TDF)	1/2	1	1/2	1	—
iii. Zidovudine (AZT)	1/2	1	1/2	1	—
iv. Lamivudine (3TC)	1/2	1	1/2	1	—
v. Didanosine (DDI)	1/2	1	1/2	1	—
vi. Emtricitabine (FTC)	1/2	1	1/2	1	—
vii. Stavudine (D4T)	1/2	1	1/2	1	—
b. Nonnucleoside reverse transcriptase inhibitors (NNRTIs)					
i. Efavirenz (EFV)	1/2	1	1/2	1	—
ii. Etravirine (ETR)	1/2	1	1/2	1	—
iii. Nevirapine (NVP)	1/2	1	1/2	1	—
iv. Rilpivirine (RPV)	1/2	1	1/2	1	—
c. Ritonavir-boosted protease inhibitors					
i. Ritonavir-boosted atazanavir (ATV/r)	1/2	1	1/2	1	—
ii. Ritonavir-boosted darunavir (DRV/r)	1/2	1	1/2	1	—

See table footnotes on page 30.

TABLE B1. (*Continued*) Classifications for intrauterine devices, including the copper-containing intrauterine device and levonorgestrel-releasing intrauterine device

Condition	Category				Clarifications/Evidence/Comments
	Cu-IUD		LNG-IUD		
iii. Ritonavir-boosted fosamprenavir (FPV/r)	1/2	1	1/2	1	—
iv. Ritonavir-boosted lopinavir (LPV/r)	1/2	1	1/2	1	—
v. Ritonavir-boosted saquinavir (SQV/r)	1/2	1	1/2	1	—
vi. Ritonavir-boosted tipranavir (TPV/r)	1/2	1	1/2	1	—
d. Protease inhibitors without ritonavir					
i. Atazanavir (ATV)	1/2	1	1/2	1	—
ii. Fosamprenavir (FPV)	1/2	1	1/2	1	—
iii. Indinavir (IDV)	1/2	1	1/2	1	—
iv. Nelfinavir (NFV)	1/2	1	1/2	1	—
e. CCR5 co-receptor antagonists					
i. Maraviroc (MVC)	1/2	1	1/2	1	—
f. HIV integrase strand transfer inhibitors					
i. Raltegravir (RAL)	1/2	1	1/2	1	—
ii. Dolutegravir (DTG)	1/2	1	1/2	1	—
iii. Elvitegravir (EVG)	1/2	1	1/2	1	—
g. Fusion inhibitors					
i. Enfuvirtide	1/2	1	1/2	1	—
Anticonvulsant therapy					
a. Certain anticonvulsants (phenytoin, carbamazepine, barbiturates, primidone, topiramate, and oxcarbazepine)	1		1		**Evidence:** Limited evidence suggests use of certain anticonvulsants does not interfere with the contraceptive effectiveness of the LNG-IUD (*142*).
b. Lamotrigine	1		1		**Evidence:** No drug interactions have been reported among women with epilepsy who are receiving lamotrigine and using the LNG-IUD (*143*).
Antimicrobial therapy					
a. Broad-spectrum antibiotics	1		1		—
b. Antifungals	1		1		—
c. Antiparasitics	1		1		—
d. Rifampin or rifabutin therapy	1		1		**Evidence:** One cross-sectional survey found that rifabutin had no impact on the effectiveness of the LNG-IUD (*142*).
Psychotropic medications					**Comment:** For many common psychotropic agents, limited or no theoretical concern exists for clinically significant drug interactions when co-administered with hormonal contraceptives. However, either no or very limited data exist examining potential interactions for these classes of medications.
a. SSRIs	1		1		—
St. John's wort	1		1		—

Abbreviations: ARV = antiretroviral; BMI = body mass index; COC = combined oral contraceptive; Cu-IUD = copper-containing IUD; DVT = deep venous thrombosis; hCG = human chorionic gonadotropin; HDL = high-density lipoprotein; HIV = human immunodeficiency virus; IBD = inflammatory bowel disease; IUD = intrauterine device; LDL = low-density lipoprotein; LNG = levonorgestrel; LNG-IUD = levonorgestrel-releasing IUD; PE = pulmonary embolism; PID = pelvic inflammatory disease; POC = progestin-only contraceptive; SLE = systemic lupus erythematosus; SSRI = selective serotonin reuptake inhibitor; STD = sexually transmitted disease; VTE = venous thromboembolism.

References

1. Cramer DW, Schiff I, Schoenbaum SC, et al. Tubal infertility and the intrauterine device. N Engl J Med 1985;312:941–7. http://dx.doi.org/10.1056/NEJM198504113121502
2. Daling JR, Weiss NS, Metch BJ, et al. Primary tubal infertility in relation to the use of an intrauterine device. N Engl J Med 1985;312:937–41. http://dx.doi.org/10.1056/NEJM198504113121501
3. Daling JR, Weiss NS, Voigt LF, McKnight B, Moore DE. The intrauterine device and primary tubal infertility. N Engl J Med 1992;326:203–4. http://dx.doi.org/10.1056/NEJM199201163260314
4. Delbarge W, Bátár I, Bafort M, et al. Return to fertility in nulliparous and parous women after removal of the GyneFix intrauterine contraceptive system. Eur J Contracept Reprod Health Care 2002;7:24–30. http://dx.doi.org/10.1080/ejc.7.1.24.30
5. Doll H, Vessey M, Painter R. Return of fertility in nulliparous women after discontinuation of the intrauterine device: comparison with women discontinuing other methods of contraception. BJOG 2001;108:304–14. http://dx.doi.org/10.1111/j.1471-0528.2001.00075.x
6. Hubacher D, Lara-Ricalde R, Taylor DJ, Guerra-Infante F, Guzmán-Rodríguez R. Use of copper intrauterine devices and the risk of tubal infertility among nulligravid women. N Engl J Med 2001;345:561–7. http://dx.doi.org/10.1056/NEJMoa010438
7. Skjeldestad FE, Bratt H. Return of fertility after use of IUDs (Nova-T, MLCu250 and MLCu375). Adv Contracept 1987;3:139–45. http://dx.doi.org/10.1007/BF01890702
8. Urbach DR, Marrett LD, Kung R, Cohen MM. Association of perforation of the appendix with female tubal infertility. Am J Epidemiol 2001;153:566–71. http://dx.doi.org/10.1093/aje/153.6.566
9. Wilson JC. A prospective New Zealand study of fertility after removal of copper intrauterine contraceptive devices for conception and because of complications: a four-year study. Am J Obstet Gynecol 1989;160:391–6. http://dx.doi.org/10.1016/0002-9378(89)90455-9
10. US Department of Health and Human Services. Healthy people 2020: Maternal, infant, and child health objectives. Washington, DC: US Department of Health and Human Services; 2015. http://www.healthypeople.gov/2020/topics-objectives/topic/maternal-infant-and-child-health/objectives

11. Annus J, Brat T, Diethelm MP, et al.; World Health Organization Special. Comparative multicentre trial of three IUDs inserted immediately following delivery of the placenta. Contraception 1980;22:9–18.http://dx.doi.org/10.1016/0010-7824(80)90112-2
12. Apelo RA, Waszak CS. Postpartum IUD insertions in Manila, Philippines. Adv Contracept 1985;1:319–28.
13. Baldwin MK, Edelman AB, Lim JY, Nichols MD, Bednarek PH, Jensen JT. Intrauterine device placement at 3 versus 6weeks postpartum: a randomized trial. Contraception 2016;93:356–63.http://dx.doi.org/10.1016/j.contraception.2015.12.006
14. Bonilla Rosales F, Aguilar Zamudio ME, Cázares Montero ML, Hernández Ortiz ME, Luna Ruiz MA. [Factors for expulsion of intrauterine device Tcu380A applied immediately postpartum and after a delayed period]. Rev Med Inst Mex Seguro Soc 2005;43:5–10.
15. Braniff K, Gomez E, Muller R. A randomised clinical trial to assess satisfaction with the levonorgestrel- releasing intrauterine system inserted at caesarean section compared to postpartum placement. Aust N Z J Obstet Gynaecol 2015;55:279–83.http://dx.doi.org/10.1111/ajo.12335
16. Bryant AG, Kamanga G, Stuart GS, Haddad LB, Meguid T, Mhango C. Immediate postpartum versus 6-week postpartum intrauterine device insertion: a feasibility study of a randomized controlled trial. Afr J Reprod Health 2013;17:72–9.
17. Caliskan E, Ozturk N, Dilbaz BO, Dilbaz S. Analysis of risk factors associated with uterine perforation by intrauterine devices. Eur J Contracep Reprod 2003;8:150–5.
18. Çelen S, Möröy P, Sucak A, Aktulay A, Danişman N. Clinical outcomes of early postplacental insertion of intrauterine contraceptive devices. Contraception 2004;69:279–82.http://dx.doi.org/10.1016/j.contraception.2003.12.004
19. Çelen Ş, Sucak A, Yıldız Y, Danışman N. Immediate postplacental insertion of an intrauterine contraceptive device during cesarean section. Contraception 2011;84:240–3.http://dx.doi.org/10.1016/j.contraception.2011.01.006
20. Chen BA, Reeves MF, Hayes JL, Hohmann HL, Perriera LK, Creinin MD. Postplacental or delayed insertion of the levonorgestrel intrauterine device after vaginal delivery: a randomized controlled trial. Obstet Gynecol 2010;116:1079–87.http://dx.doi.org/10.1097/AOG.0b013e3181f73fac
21. Chen JH, Wu SC, Shao WQ, et al. The comparative trial of TCu 380A IUD and progesterone-releasing vaginal ring used by lactating women. Contraception 1998;57:371–9.http://dx.doi.org/10.1016/S0010-7824(98)00043-2
22. Cohen R, Sheeder J, Arango N, Teal SB, Tocce K. Twelve-month contraceptive continuation and repeat pregnancy among young mothers choosing postdelivery contraceptive implants or postplacental intrauterine devices. Contraception 2016;93:178–83.http://dx.doi.org/10.1016/j.contraception.2015.10.001
23. Dahlke JD, Terpstra ER, Ramseyer AM, Busch JM, Rieg T, Magann EF. Postpartum insertion of levonorgestrel—intrauterine system at three time periods: a prospective randomized pilot study. Contraception 2011;84:244–8.http://dx.doi.org/10.1016/j.contraception.2011.01.007
24. Dias T, Abeykoon S, Kumarasiri S, Gunawardena C, Padeniya T, D'Antonio F. Use of ultrasound in predicting success of intrauterine contraceptive device insertion immediately after delivery. Ultrasound Obstet Gynecol 2015;46:104–8.
25. Elsedeek MS. Puerperal and menstrual bleeding patterns with different types of contraceptive device fitted during elective cesarean delivery. Int J Gynaecol Obstet 2012;116:31–4.http://dx.doi.org/10.1016/j.ijgo.2011.07.036
26. Elsedeek MS. Five-year follow-up of two types of contraceptive device fitted during elective cesarean delivery. Int J Gynaecol Obstet 2015;130:179–82.http://dx.doi.org/10.1016/j.ijgo.2015.02.031
27. El-Shafei MM, Mashali A, Hassan EO, El-Boghdad L, El-Lakkany N. Postpartum and postabortion intrauterine device insertion unmet needs of safe reproductive health: three years experience of Mansoura University Hospital. J Egypt Soc Obstet Gynecol 2000;26:253–62.
28. Eroğlu K, Akkuzu G, Vural G, et al. Comparison of efficacy and complications of IUD insertion in immediate postplacental/early postpartum period with interval period: 1 year follow-up. Contraception 2006;74:376–81.http://dx.doi.org/10.1016/j.contraception.2006.07.003
29. Gueye M, Gaye YF, Diouf AA, et al. Trancesarean intra-uterine device. Pilot study performed at Dakar teaching hospital. [French]. J Gynecol Obstet Biol Reprod (Paris) 2013;42:585–90.http://dx.doi.org/10.1016/j.jgyn.2013.06.003
30. Gupta S, Malik S, Sinha R, Shyamsunder S, Mittal MK. Association of the Position of the Copper T 380A as Determined by the Ultrasonography Following its Insertion in the Immediate Postpartum Period with the Subsequent Complications: An Observational Study. J Obstet Gynaecol India 2014;64:349–53.http://dx.doi.org/10.1007/s13224-014-0532-5
31. Hagbard L, Ingemanson CA, Sorbe B. Early postpartum insertion of copper IUD. Contraception 1978;17:355–63.http://dx.doi.org/10.1016/0010-7824(78)90081-1
32. Hayes JL, Cwiak C, Goedken P, Zieman M. A pilot clinical trial of ultrasound-guided postplacental insertion of a levonorgestrel intrauterine device. Contraception 2007;76:292–6.http://dx.doi.org/10.1016/j.contraception.2007.06.003
33. Jatlaoui TC, Marcus M, Jamieson DJ, Goedken P, Cwiak C. Postplacental intrauterine device insertion at a teaching hospital. Contraception 2014;89:528–33.http://dx.doi.org/10.1016/j.contraception.2013.10.008
34. Kumar S, Sethi R, Balasubramaniam S, et al. Women's experience with postpartum intrauterine contraceptive device use in India. Reprod Health 2014;11:32.http://dx.doi.org/10.1186/1742-4755-11-32
35. Laes E, Lehtovirta P, Weintraub D, Pyörälä T, Luukkainen T. Early puerperal insertions of copper-T-200. Contraception 1975;11:289–95.http://dx.doi.org/10.1016/0010-7824(75)90037-2
36. Lara Ricalde R, Menocal Tobías G, Ramos Pérez C, Velázquez Ramírez N. [Random comparative study between intrauterine device Multiload Cu375 and TCu 380a inserted in the postpartum period]. Ginecol Obstet Mex 2006;74:306–11.
37. Lavin P, Bravo C, Waszak C. Comparison of T Cu 200 and Progestasert IUDs. Contracept Deliv Syst 1983;4:143–7.
38. Lavin P, Waszak C, Bravo C. Preliminary report on a postpartum CuT 200 study, Santiago, Chile. Int J Gynaecol Obstet 1983;21:71–5.http://dx.doi.org/10.1016/0020-7292(83)90073-5
39. Lester F, Kakaire O, Byamugisha J, et al. Intracesarean insertion of the Copper T380A versus 6 weeks postcesarean: a randomized clinical trial. Contraception 2015;91:198–203.http://dx.doi.org/10.1016/j.contraception.2014.12.002
40. Letti Müller AL, Lopes Ramos JG, Martins-Costa SH, et al. Transvaginal ultrasonographic assessment of the expulsion rate of intrauterine devices inserted in the immediate postpartum period: a pilot study. Contraception 2005;72:192–5.http://dx.doi.org/10.1016/j.contraception.2005.03.014
41. Levi E, Cantillo E, Ades V, Banks E, Murthy A. Immediate postplacental IUD insertion at cesarean delivery: a prospective cohort study. Contraception 2012;86:102–5.http://dx.doi.org/10.1016/j.contraception.2011.11.019
42. Levi EE, Stuart GS, Zerden ML, Garrett JM, Bryant AG. Intrauterine device placement during cesarean delivery and continued use 6 months postpartum: a randomized controlled trial. Obstet Gynecol 2015;126:5–11.http://dx.doi.org/10.1097/AOG.0000000000000882
43. Mishra S. Evaluation of Safety, Efficacy, and Expulsion of Post-Placental and Intra-Cesarean Insertion of Intrauterine Contraceptive Devices (PPIUCD). J Obstet Gynaecol India 2014;64:337–43.http://dx.doi.org/10.1007/s13224-014-0550-3
44. Morrison C, Waszak C, Katz K, Diabaté F, Mate EM. Clinical outcomes of two early postpartum IUD insertion programs in Africa. Contraception 1996;53:17–21.http://dx.doi.org/10.1016/0010-7824(95)00254-5
45. Nelson AL, Chen S, Eden R. Intraoperative placement of the Copper T-380 intrauterine devices in women undergoing elective cesarean delivery: a pilot study. Contraception 2009;80:81–3.http://dx.doi.org/10.1016/j.contraception.2009.01.014

46. Newton J, Harper M, Chan KK. Immediate post-placental insertion of intrauterine contraceptive devices. Lancet 1977;310:272–4.http://dx.doi.org/10.1016/S0140-6736(77)90955-2
47. Prema K, Gayathri TL, Philips FS. Comparative study of early postpartum, postabortal and interval insertion of Cu T 200 mm2 device. J Obstet Gynaecol India 1978;28:946–8.
48. Puzey M. Mirena at caesarean section. Eur J Contracep Reprod 2005;10:164–7.
49. Ragab A, Hamed HO, Alsammani MA, et al. Expulsion of Nova-T380, Multiload 375, and Copper-T380A contraceptive devices inserted during cesarean delivery. Int J Gynaecol Obstet 2015;130:174–8.http://dx.doi.org/10.1016/j.ijgo.2015.03.025
50. Shukla M, Qureshi S; Chandrawati. Post-placental intrauterine device insertion—a five year experience at a tertiary care centre in north India. Indian J Med Res 2012;136:432–5.
51. Singal S, Bharti R, Dewan R, et al. Clinical Outcome of Postplacental Copper T 380A Insertion in Women Delivering by Caesarean Section. J Clin Diagn Res 2014;8:OC01–04.
52. Stuart GS, Bryant AG, O'Neill E, Doherty IA. Feasibility of postpartum placement of the levonorgestrel intrauterine system more than 6 h after vaginal birth. Contraception 2012;85:359–62.http://dx.doi.org/10.1016/j.contraception.2011.08.005
53. Stuart GS, Lesko CR, Stuebe AM, Bryant AG, Levi EE, Danvers AI. A randomized trial of levonorgestrel intrauterine system insertion 6 to 48 h compared to 6 weeks after vaginal delivery; lessons learned. Contraception 2015;91:284–8.http://dx.doi.org/10.1016/j.contraception.2014.12.009
54. Thiery M, Van Kets H, Van der Pas H. Immediate post-placental IUD insertion: the expulsion problem. Contraception 1985;31:331–49.http://dx.doi.org/10.1016/0010-7824(85)90002-2
55. Van Der Pas MT, Delbeke L, Van Dets H. Comparative performance of two copper-wired IUDs (ML Cu 250 and T Cu 200: immediate postpartum and interval insertion. Contracept Deliv Syst 1980;1:27–35.
56. Welkovic S, Costa LO, Faúndes A, de Alencar Ximenes R, Costa CF. Post-partum bleeding and infection after post-placental IUD insertion. Contraception 2001;63:155–8.http://dx.doi.org/10.1016/S0010-7824(01)00180-9
57. Whitaker AK, Endres LK, Mistretta SQ, Gilliam ML. Postplacental insertion of the levonorgestrel intrauterine device after cesarean delivery vs. delayed insertion: a randomized controlled trial. Contraception 2014;89:534–9.http://dx.doi.org/10.1016/j.contraception.2013.12.007
58. Woo CJ, Alamgir H, Potter JE. Women's experiences after Planned Parenthood's exclusion from a family planning program in Texas. Contraception 2016;93:298–302.http://dx.doi.org/10.1016/j.contraception.2015.12.004
59. Wu SC; Research Group on Failure Causes and Prevention Measures of Intrauterine Device. [Efficacy of intrauterine device TCu380A when inserted in four different periods]. Zhonghua Fu Chan Ke Za Zhi 2009;44:431–5.
60. Xu J, Yang X, Gu X, et al. Comparison between two techniques used in immediate postplacental insertion of TCu 380A intrauterine device: 36-month follow-up. Reprod Contracept 1999;10:156–62.
61. Xu J, Zhuang L, Yu G. [Comparison of two techniques used in immediate postplacental insertion of TCu 380A intrauterine device: 12 month follow-up of 910 cases]. Zhonghua Fu Chan Ke Za Zhi 1997;32:354–7.
62. Xu JX, Rivera R, Dunson TR, et al. A comparative study of two techniques used in immediate postplacental insertion (IPPI) of the Copper T-380A IUD in Shanghai, People's Republic of China. Contraception 1996;54:33–8.http://dx.doi.org/10.1016/0010-7824(96)00117-5
63. Braniff K, Gomez E, Muller R. A randomised clinical trial to assess satisfaction with the levonorgestrel- releasing intrauterine system inserted at caesarean section compared to postpartum placement. Aust N Z J Obstet Gynaecol 2015;55:279–83. http://dx.doi.org/10.1111/ajo.12335
64. Phillips SJ, Tepper NK, Kapp N, Nanda K, Temmerman M, Curtis KM. Progestogen-only contraceptive use among breastfeeding women: a systematic review. Contraception 2015;S0010-7824(15)00585-5.
65. Berry-Bibee E, Tepper N, Jatlaoui T, Whiteman M, Jamieson D, Curtis K. The safety of intrauterine devices in breastfeeding women: a systematic review. Contraception. In press 2016.
66. Steenland MW, Tepper NK, Curtis KM, Kapp N. Intrauterine contraceptive insertion postabortion: a systematic review. Contraception 2011;84:447–64. http://dx.doi.org/10.1016/j.contraception.2011.03.007
67. World Health Organization Collaborative Study of Cardiovascular Disease and Steroid Hormone Contraception. Cardiovascular disease and use of oral and injectable progestogen-only contraceptives and combined injectable contraceptives. Results of an international, multicenter, case-control study. Contraception 1998;57:315–24. http://dx.doi.org/10.1016/S0010-7824(98)00041-9.
68. Heinemann LA, Assmann A, DoMinh T, Garbe E. Oral progestogen-only contraceptives and cardiovascular risk: results from the Transnational Study on Oral Contraceptives and the Health of Young Women. Eur J Contracept Reprod Health Care 1999;4:67–73. http://dx.doi.org/10.3109/13625189909064007
69. Vasilakis C, Jick H, del Mar Melero-Montes M. Risk of idiopathic venous thromboembolism in users of progestagens alone. Lancet 1999;354:1610–1. http://dx.doi.org/10.1016/S0140-6736(99)04394-9
70. Kingman CE, Kadir RA, Lee CA, Economides DL. The use of levonorgestrel-releasing intrauterine system for treatment of menorrhagia in women with inherited bleeding disorders. BJOG 2004;111:1425–8. http://dx.doi.org/10.1111/j.1471-0528.2004.00305.x
71. Lukes AS, Reardon B, Arepally G. Use of the levonorgestrel-releasing intrauterine system in women with hemostatic disorders. Fertil Steril 2008;90:673–7. http://dx.doi.org/10.1016/j.fertnstert.2007.07.1315
72. Pisoni CN, Cuadrado MJ, Khamashta MA, Hunt BJ. Treatment of menorrhagia associated with oral anticoagulation: efficacy and safety of the levonorgestrel releasing intrauterine device (Mirena coil). Lupus 2006;15:877–80. http://dx.doi.org/10.1177/0961203306071706
73. Schaedel ZE, Dolan G, Powell MC. The use of the levonorgestrel-releasing intrauterine system in the management of menorrhagia in women with hemostatic disorders. Am J Obstet Gynecol 2005;193:1361–3. http://dx.doi.org/10.1016/j.ajog.2005.05.002
74. Wilson W, Taubert KA, Gewitz M, et al; American Heart Association Rheumatic Fever, Endocarditis, and Kawasaki Disease Committee; American Heart Association Council on Cardiovascular Disease in the Young; American Heart Association Council on Clinical Cardiology; American Heart Association Council on Cardiovascular Surgery and Anesthesia; Quality of Care and Outcomes Research Interdisciplinary Working Group. Prevention of infective endocarditis: guidelines from the American Heart Association: a guideline from the American Heart Association Rheumatic Fever, Endocarditis, and Kawasaki Disease Committee, Council on Cardiovascular Disease in the Young, and the Council on Clinical Cardiology, Council on Cardiovascular Surgery and Anesthesia, and the Quality of Care and Outcomes Research Interdisciplinary Working Group. Circulation 2007;116:1736–54. http://dx.doi.org/10.1161/CIRCULATIONAHA.106.183095
75. Tepper NK, Paulen ME, Marchbanks PA, Curtis KM. Safety of contraceptive use among women with peripartum cardiomyopathy: a systematic review. Contraception 2010;82:95–101. http://dx.doi.org/10.1016/j.contraception.2010.02.004
76. The Criteria Committee of the New York Heart Association. Nomenclature and criteria for diagnosis of diseases of the heart and great vessels. 9th ed. Boston, MA: Little, Brown and Co; 1994.
77. Bernatsky S, Clarke A, Ramsey-Goldman R, et al. Hormonal exposures and breast cancer in a sample of women with systemic lupus erythematosus. Rheumatology (Oxford) 2004;43:1178–81. http://dx.doi.org/10.1093/rheumatology/keh282
78. Bernatsky S, Ramsey-Goldman R, Gordon C, et al. Factors associated with abnormal Pap results in systemic lupus erythematosus. Rheumatology (Oxford) 2004;43:1386–9. http://dx.doi.org/10.1093/rheumatology/keh331
79. Chopra N, Koren S, Greer WL, et al. Factor V Leiden, prothrombin gene mutation, and thrombosis risk in patients with antiphospholipid antibodies. J Rheumatol 2002;29:1683–8.

80. Esdaile JM, Abrahamowicz M, Grodzicky T, et al. Traditional Framingham risk factors fail to fully account for accelerated atherosclerosis in systemic lupus erythematosus. Arthritis Rheum 2001;44:2331–7. http://dx.doi.org/10.1002/1529-0131(200110)44:10<2331::AID-ART395≥3.0.CO;2-I
81. Julkunen HA, Kaaja R, Friman C. Contraceptive practice in women with systemic lupus erythematosus. Br J Rheumatol 1993;32:227–30. http://dx.doi.org/10.1093/rheumatology/32.3.227
82. Manzi S, Meilahn EN, Rairie JE, et al. Age-specific incidence rates of myocardial infarction and angina in women with systemic lupus erythematosus: comparison with the Framingham Study. Am J Epidemiol 1997;145:408–15. http://dx.doi.org/10.1093/oxfordjournals.aje.a009122
83. McAlindon T, Giannotta L, Taub N, D'Cruz D, Hughes G. Environmental factors predicting nephritis in systemic lupus erythematosus. Ann Rheum Dis 1993;52:720–4. http://dx.doi.org/10.1136/ard.52.10.720
84. McDonald J, Stewart J, Urowitz MB, Gladman DD. Peripheral vascular disease in patients with systemic lupus erythematosus. Ann Rheum Dis 1992;51:56–60. http://dx.doi.org/10.1136/ard.51.1.56
85. Mintz G, Gutiérrez G, Delezé M, Rodríguez E. Contraception with progestagens in systemic lupus erythematosus. Contraception 1984;30:29–38. http://dx.doi.org/10.1016/0010-7824(84)90076-3
86. Petri M. Musculoskeletal complications of systemic lupus erythematosus in the Hopkins Lupus Cohort: an update. Arthritis Care Res 1995;8:137–45. http://dx.doi.org/10.1002/art.1790080305
87. Petri M. Lupus in Baltimore: evidence-based 'clinical pearls' from the Hopkins Lupus Cohort. Lupus 2005;14:970–3. http://dx.doi.org/10.1191/0961203305lu2230xx
88. Sánchez-Guerrero J, Uribe AG, Jiménez-Santana L, et al. A trial of contraceptive methods in women with systemic lupus erythematosus. N Engl J Med 2005;353:2539–49. http://dx.doi.org/10.1056/NEJMoa050817
89. Sarabi ZS, Chang E, Bobba R, et al. Incidence rates of arterial and venous thrombosis after diagnosis of systemic lupus erythematosus. Arthritis Rheum 2005;53:609–12. http://dx.doi.org/10.1002/art.21314
90. Somers E, Magder LS, Petri M. Antiphospholipid antibodies and incidence of venous thrombosis in a cohort of patients with systemic lupus erythematosus. J Rheumatol 2002;29:2531–6.
91. Urowitz MB, Bookman AA, Koehler BE, Gordon DA, Smythe HA, Ogryzlo MA. The bimodal mortality pattern of systemic lupus erythematosus. Am J Med 1976;60:221–5. http://dx.doi.org/10.1016/0002-9343(76)90431-9
92. Julkunen HA. Oral contraceptives in systemic lupus erythematosus: side-effects and influence on the activity of SLE. Scand J Rheumatol 1991;20:427–33. http://dx.doi.org/10.3109/03009749109096822
93. Jungers P, Dougados M, Pélissier C, et al. Influence of oral contraceptive therapy on the activity of systemic lupus erythematosus. Arthritis Rheum 1982;25:618–23. http://dx.doi.org/10.1002/art.1780250603
94. Petri M, Kim MY, Kalunian KC, et al; OC-SELENA Trial. Combined oral contraceptives in women with systemic lupus erythematosus. N Engl J Med 2005;353:2550–8. http://dx.doi.org/10.1056/NEJMoa051135
95. Choojitarom K, Verasertniyom O, Totemchokchyakarn K, Nantiruj K, Sumethkul V, Janwityanujit S. Lupus nephritis and Raynaud's phenomenon are significant risk factors for vascular thrombosis in SLE patients with positive antiphospholipid antibodies. Clin Rheumatol 2008;27:345–51. http://dx.doi.org/10.1007/s10067-007-0721-z
96. Wahl DG, Guillemin F, de Maistre E, Perret C, Lecompte T, Thibaut G. Risk for venous thrombosis related to antiphospholipid antibodies in systemic lupus erythematosus—a meta-analysis. Lupus 1997;6:467–73. http://dx.doi.org/10.1177/096120339700600510
97. Tepper NK, Whiteman MK, Zapata LB, Marchbanks PA, Curtis KM. Safety of hormonal contraceptives among women with migraine: a systematic review. Contraception 2016. Epub May 3, 2016. http:// dx.doi.org/10.1016/j.contraception.2016.04.016
98. Tepper NK, Whiteman MK, Marchbanks PA, James AH, Curtis KM. Progestin-only contraception and thromboembolism: a systematic review. Contraception 2016. Epub May 3, 2016. http://dx.doi.org/10.1016/j.contraception.2016.04.014
99. Pagano HP, Zapata LB, Berry-Bibee EN, Nanda K, Curtis KM. Safety of hormonal contraception and intrauterine devices among women with depressive and bipolar disorders: a systematic review. Contraception 2016. Epub June 27, 2016. http://dx.doi.org/10.1016/j.contraception.2016.06.012
100. Barrington JW, Arunkalaivanan AS, Abdel-Fattah M. Comparison between the levonorgestrel intrauterine system (LNG-IUS) and thermal balloon ablation in the treatment of menorrhagia. Eur J Obstet Gynecol Reprod Biol 2003;108:72–4. http://dx.doi.org/10.1016/S0301-2115(02)00408-6
101. Gupta B, Mittal S, Misra R, Deka D, Dadhwal V. Levonorgestrel-releasing intrauterine system vs. transcervical endometrial resection for dysfunctional uterine bleeding. Int J Gynaecol Obstet 2006;95:261–6. http://dx.doi.org/10.1016/j.ijgo.2006.07.004
102. Hurskainen R, Teperi J, Rissanen P, et al. Quality of life and cost-effectiveness of levonorgestrel-releasing intrauterine system versus hysterectomy for treatment of menorrhagia: a randomised trial.[see comment]. Lancet 2001;357:273–7. http://dx.doi.org/10.1016/S0140-6736(00)03615-1
103. Istre O, Trolle B. Treatment of menorrhagia with the levonorgestrel intrauterine system versus endometrial resection. Fertil Steril 2001;76:304–9. http://dx.doi.org/10.1016/S0015-0282(01)01909-4
104. Koh SC, Singh K. The effect of levonorgestrel-releasing intrauterine system use on menstrual blood loss and the hemostatic, fibrinolytic/inhibitor systems in women with menorrhagia. J Thromb Haemost 2007;5:133–8. http://dx.doi.org/10.1111/j.1538-7836.2006.02243.x
105. Lethaby AE, Cooke I, Rees M. Progesterone/progestogen releasing intrauterine systems versus either placebo or any other medication for heavy menstrual bleeding. Cochrane Database Syst Rev 2000;(2):CD002126.
106. Magalhães J, Aldrighi JM, de Lima GR. Uterine volume and menstrual patterns in users of the levonorgestrel-releasing intrauterine system with idiopathic menorrhagia or menorrhagia due to leiomyomas. Contraception 2007;75:193–8. http://dx.doi.org/10.1016/j.contraception.2006.11.004
107. Stewart A, Cummins C, Gold L, Jordan R, Phillips W. The effectiveness of the levonorgestrel-releasing intrauterine system in menorrhagia: a systematic review. BJOG 2001;108:74–86. http://dx.doi.org/10.1111/j.1471-0528.2001.00020.x
108. Fedele L, Bianchi S, Zanconato G, Portuese A, Raffaelli R. Use of a levonorgestrel-releasing intrauterine device in the treatment of rectovaginal endometriosis. Fertil Steril 2001;75:485–8. http://dx.doi.org/10.1016/S0015-0282(00)01759-3
109. Lockhat FBE, Emembolu J, Konje JC. The effect of a levonorgestrel intrauterine system (LNG-IUS) on symptomatic endometriosis. Fertil Steril 2002;77(Suppl 1):S24. http://dx.doi.org/10.1016/S0015-0282(01)03086-2
110. Petta CA, Ferriani RA, Abrao MS, et al. Randomized clinical trial of a levonorgestrel-releasing intrauterine system and a depot GnRH analogue for the treatment of chronic pelvic pain in women with endometriosis. Hum Reprod 2005;20:1993–8. http://dx.doi.org/10.1093/humrep/deh869
111. Vercellini P, Aimi G, Panazza S, De Giorgi O, Pesole A, Crosignani PG. A levonorgestrel-releasing intrauterine system for the treatment of dysmenorrhea associated with endometriosis: a pilot study. Fertil Steril 1999;72:505–8. http://dx.doi.org/10.1016/S0015-0282(99)00291-5
112. Vercellini P, Frontino G, De Giorgi O, Aimi G, Zaina B, Crosignani PG. Comparison of a levonorgestrel-releasing intrauterine device versus expectant management after conservative surgery for symptomatic endometriosis: a pilot study. Fertil Steril 2003;80:305–9. http://dx.doi.org/10.1016/S0015-0282(03)00608-3
113. Gaffield ME, Kapp N, Curtis KM. Combined oral contraceptive and intrauterine device use among women with gestational trophoblastic disease. Contraception 2009;80:363–71. http://dx.doi.org/10.1016/j.contraception.2009.03.022

114. Whiteman MK, Zapata LB, Tepper NK, Marchbanks PA, Curtis KM. Use of contraceptive methods among women with endometrial hyperplasia: a systematic review. Contraception 2010;82:56–63. http://dx.doi.org/10.1016/j.contraception.2010.02.005
115. Zapata LB, Whiteman MK, Tepper NK, Jamieson DJ, Marchbanks PA, Curtis KM. Intrauterine device use among women with uterine fibroids: a systematic review. Contraception 2010;82:41–55. http://dx.doi.org/10.1016/j.contraception.2010.02.011
116. Tepper NK, Steenland MW, Gaffield ME, Marchbanks PA, Curtis KM. Retention of intrauterine devices in women who acquire pelvic inflammatory disease: a systematic review. Contraception 2013;87:655–60. http://dx.doi.org/10.1016/j.contraception.2012.08.011
117. Faúndes A, Telles E, Cristofoletti ML, Faúndes D, Castro S, Hardy E. The risk of inadvertent intrauterine device insertion in women carriers of endocervical Chlamydia trachomatis. Contraception 1998;58:105–9. http://dx.doi.org/10.1016/S0010-7824(98)00064-X
118. Ferraz do Lago R, Simões JA, Bahamondes L, Camargo RP, Perrotti M, Monteiro I. Follow-up of users of intrauterine device with and without bacterial vaginosis and other cervicovaginal infections. Contraception 2003;68:105–9. http://dx.doi.org/10.1016/S0010-7824(03)00109-4
119. Morrison CS, Sekadde-Kigondu C, Miller WC, Weiner DH, Sinei SK. Use of sexually transmitted disease risk assessment algorithms for selection of intrauterine device candidates. Contraception 1999;59:97–106. http://dx.doi.org/10.1016/S0010-7824(99)00006-2
120. Pap-Akeson M, Solheim F, Thorbert G, Akerlund M. Genital tract infections associated with the intrauterine contraceptive device can be reduced by inserting the threads into the uterine cavity. Br J Obstet Gynaecol 1992;99:676–9. http://dx.doi.org/10.1111/j.1471-0528.1992.tb13854.x
121. Sinei SK, Schulz KFLP, Lamptey PR, et al. Preventing IUCD-related pelvic infection: the efficacy of prophylactic doxycycline at insertion. Br J Obstet Gynaecol 1990;97:412–9. http://dx.doi.org/10.1111/j.1471-0528.1990.tb01828.x
122. Skjeldestad FE, Halvorsen LE, Kahn H, Nordbø SA, Saake K. IUD users in Norway are at low risk for genital C. trachomatis infection. Contraception 1996;54:209–12. http://dx.doi.org/10.1016/S0010-7824(96)00190-4
123. Walsh TL, Bernstein GS, Grimes DA, Frezieres R, Bernstein L, Coulson AH; IUD Study Group. Effect of prophylactic antibiotics on morbidity associated with IUD insertion: results of a pilot randomized controlled trial. Contraception 1994;50:319–27. http://dx.doi.org/10.1016/0010-7824(94)90019-1
124. Workowski KA, Bolan GA. Sexually transmitted diseases treatment guidelines, 2015. MMWR Recomm Rep 2015;64(No. RR-03).
125. Jatlaoui TC, Simmons KB, Curtis KM. The safety of intrauterine contraception initiation among women with current asymptomatic cervical infections or at increased risk of sexually transmitted infections. Contraception 2016. Epub June 1, 2016. http://dx.doi.org/10.1016/j.contraception.2016.05.013
126. Carael M, Van de Perre PH, Lepage PH, et al. Human immunodeficiency virus transmission among heterosexual couples in Central Africa. AIDS 1988;2:201–5.
127. European Study Group on Heterosexual Transmission of HIV. Comparison of female to male and male to female transmission of HIV in 563 stable couples. BMJ 1992;304:809–13. http://dx.doi.org/10.1136/bmj.304.6830.809
128. Kapiga SH, Lyamuya EF, Lwihula GK, Hunter DJ. The incidence of HIV infection among women using family planning methods in Dar es Salaam, Tanzania. AIDS 1998;12:75–84. http://dx.doi.org/10.1097/00002030-199801000-00009.
129. Kapiga SH, Shao JF, Lwihula GK, Hunter DJ. Risk factors for HIV infection among women in Dar-es-Salaam, Tanzania. J Acquir Immune Defic Syndr 1994;7:301–9.
130. Mann JM, Nzilambi N, Piot P, et al. HIV infection and associated risk factors in female prostitutes in Kinshasa, Zaire. AIDS 1988;2:249–54. http://dx.doi.org/10.1097/00002030-198808000-00002
131. Martin HL Jr, Nyange PM, Richardson BA, et al. Hormonal contraception, sexually transmitted diseases, and risk of heterosexual transmission of human immunodeficiency virus type 1. J Infect Dis 1998;178:1053–9. http://dx.doi.org/10.1086/515654
132. Mali JK, Hunter DJ, Maggwa BN, Tukei PM. Contraceptive use and the risk of HIV infection in Nairobi, Kenya. Int J Gynaecol Obstet 1995;48:61–7. http://dx.doi.org/10.1016/0020-7292(94)02214-3
133. Nicolosi A, Corrêa Leite ML, Musicco M, Arici C, Gavazzeni G, Lazzarin A; Italian Study Group on HIV Heterosexual Transmission. The efficiency of male-to-female and female-to-male sexual transmission of the human immunodeficiency virus: a study of 730 stable couples. [comment]. Epidemiology 1994;5:570–5. http://dx.doi.org/10.1097/00001648-199411000-00003
134. Plourde PJ, Plummer FA, Pepin J, et al. Human immunodeficiency virus type 1 infection in women attending a sexually transmitted diseases clinic in Kenya.[comment]. J Infect Dis 1992;166:86–92. http://dx.doi.org/10.1093/infdis/166.1.86
135. Sinei SK, Fortney JA, Kigondu CS, et al. Contraceptive use and HIV infection in Kenyan family planning clinic attenders. Int J STD AIDS 1996;7:65–70. http://dx.doi.org/10.1258/0956462961917104
136. Spence MR, Robbins SM, Polansky M, Schable CA. Seroprevalence of human immunodeficiency virus type I (HIV-1) antibodies in a family-planning population. Sex Transm Dis 1991;18:143–5. http://dx.doi.org/10.1097/00007435-199107000-00003
137. Tepper NK, Curtis KM, Nanda K, Jamieson DJ. Safety of intrauterine devices among women with HIV: a systematic review. Contraception 2016. Epub June 22, 2016. http://dx.doi.org/0.1016/j.contraception.2016.06.011
138. Grigoryan OR, Grodnitskaya EE, Andreeva EN, Shestakova MV, Melnichenko GA, Dedov II. Contraception in perimenopausal women with diabetes mellitus. Gynecol Endocrinol 2006;22:198–206. http://dx.doi.org/10.1080/09513590600624317
139. Rogovskaya S, Rivera R, Grimes DA, et al. Effect of a levonorgestrel intrauterine system on women with type 1 diabetes: a randomized trial. Obstet Gynecol 2005;105:811–5. http://dx.doi.org/10.1097/01.AOG.0000156301.11939.56
140. Zapata LB, Paulen ME, Cansino C, Marchbanks PA, Curtis KM. Contraceptive use among women with inflammatory bowel disease: a systematic review. Contraception 2010;82:72–85. http://dx.doi.org/10.1016/j.contraception.2010.02.012
141. Paulen ME, Folger SG, Curtis KM, Jamieson DJ. Contraceptive use among solid organ transplant patients: a systematic review. Contraception 2010;82:102–12. http://dx.doi.org/10.1016/j.contraception.2010.02.007
142. Bounds W, Guillebaud J. Observational series on women using the contraceptive Mirena concurrently with anti-epileptic and other enzyme-inducing drugs. J Fam Plann Reprod Health Care 2002;28:78–80. http://dx.doi.org/10.1783/147118902101195992
143. Reimers A, Helde G, Brodtkorb E. Ethinyl estradiol, not progestogens, reduces lamotrigine serum concentrations. Epilepsia 2005;46:1414–7. http://dx.doi.org/10.1111/j.1528-1167.2005.10105.x

Appendix C

Classifications for Progestin-Only Contraceptives

Classifications for progestin-only contraceptives (POCs) include those for progestin-only implants, depot medroxyprogesterone acetate (DMPA; 150 mg intramuscularly or 104 mg subcutaneously), and progestin-only pills (POPs) (Box C1) (Table C1). POCs do not protect against sexually transmitted diseases (STDs), including human immunodeficiency virus (HIV), and women using these methods should be counseled that consistent and correct use of the male latex condom reduces the risk for transmission of HIV and other STDs. Use of female condoms can provide protection from transmission of STDs, although data are limited.

BOX C1. Categories for classifying progestin-only contraceptives

1 = A condition for which there is no restriction for the use of the contraceptive method.
2 = A condition for which the advantages of using the method generally outweigh the theoretical or proven risks.
3 = A condition for which the theoretical or proven risks usually outweigh the advantages of using the method.
4 = A condition that represents an unacceptable health risk if the contraceptive method is used.

TABLE C1. Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category			Clarifications/Evidence/Comments
	Implants	DMPA	POPs	
Personal Characteristics and Reproductive History				
Pregnancy	NA	NA	NA	**Clarification:** Use of POCs is not required. No known harm to the woman, the course of her pregnancy, or the fetus occurs if POCs are inadvertently used during pregnancy. However, the relation between DMPA use during pregnancy and its effects on the fetus remains unclear.
Age				**Evidence:** Most studies have found that women lose BMD during DMPA use but recover BMD after discontinuation. Limited evidence shows a weak association with fracture. However, one large study suggests that women who choose DMPA might be at higher risk for fracture before initiation (*1*). It is unclear whether adult women with long durations of DMPA use can regain BMD to baseline levels before entering menopause and whether adolescents can reach peak bone mass after discontinuation of DMPA. The relationship between these changes in BMD during the reproductive years and future fracture risk is unknown. Studies generally find no effect of POCs other than DMPA on BMD *(1–48)*.
a. Menarche to <18 years	1	2	1	
b. 18–45 years	1	1	1	
c. >45 years	1	2	1	
Parity				
a. Nulliparous	1	1	1	—
b. Parous	1	1	1	—
Breastfeeding				
a. <21 days postpartum	2	2	2	**Clarification:** Breastfeeding provides important health benefits for mother and infant. The U.S. Department of Health and Human Services recommends increasing the proportion of infants initially breastfed, exclusively breastfed through 6 months of life, and continuing breastfeeding through at least 1 year of life as key public health goals (*49*). **Evidence:** Two small, randomized controlled trials found no adverse impact on breastfeeding with initiation of etonogestrel implants within 48 hours postpartum. Other studies found that initiation of POPs, injectables, and implants at ≤6 weeks postpartum compared with nonhormonal use had no detrimental effect on breastfeeding outcomes or infant health, growth, and development in the first year postpartum. In general, these studies are of poor quality, lack standard definitions of breastfeeding or outcome measures, and have not included premature or ill infants (*50*,*51*). **Comment:** Certain women might be at risk for breastfeeding difficulties, such as women with previous breastfeeding difficulties, certain medical conditions, and certain perinatal complications and those who deliver preterm. For these women, as for all women, discussions about contraception for breastfeeding women should include information about risks, benefits, and alternatives.

See table footnotes on page 49.

TABLE C1. (*Continued*) Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category			Clarifications/Evidence/Comments
	Implants	DMPA	POPs	
b. 21 to <30 days postpartum				
i. With other risk factors for VTE (e.g., age ≥35 years, previous VTE, thrombophilia, immobility, transfusion at delivery, peripartum cardiomyopathy, BMI ≥30 kg/m^2, postpartum hemorrhage, postcesarean delivery, preeclampsia, or smoking)	2	2	2	**Clarification:** Breastfeeding provides important health benefits for mother and infant. The U.S. Department of Health and Human Services recommends increasing the proportion of infants initially breastfed, exclusively breastfed through 6 months of life, and continuing breastfeeding through at least 1 year of life as key public health goals (*49*). **Evidence:** Two small, randomized controlled trials found no adverse impact on breastfeeding with initiation of etonogestrel implants within 48 hours postpartum. Other studies found that initiation of POPs, injectables, and implants at ≤6 weeks postpartum compared with nonhormonal use had no detrimental effect on breastfeeding outcomes or infant health, growth, and development in the first year postpartum. In general, these studies are of poor quality, lack standard definitions of breastfeeding or outcome measures, and have not included premature or ill infants (*50*,*51*). **Comment:** Certain women might be at risk for breastfeeding difficulties, such as women with previous breastfeeding difficulties, certain medical conditions, and certain perinatal complications and those who deliver preterm. For these women, as for all women, discussions about contraception for breastfeeding women should include information about risks, benefits, and alternatives.
ii. Without other risk factors for VTE	2	2	2	
c. 30–42 days postpartum				
i. With other risk factors for VTE (e.g., age ≥35 years, previous VTE, thrombophilia, immobility, transfusion at delivery, peripartum cardiomyopathy, BMI ≥30 kg/m^2, postpartum hemorrhage, postcesarean delivery, preeclampsia, or smoking)	1	1	1	**Clarification:** Breastfeeding provides important health benefits for mother and infant. The U.S. Department of Health and Human Services recommends increasing the proportion of infants initially breastfed, exclusively breastfed through 6 months of life, and continuing breastfeeding through at least 1 year of life as key public health goals (*49*). **Evidence:** Two small, randomized controlled trials found no adverse impact on breastfeeding with initiation of etonogestrel implants within 48 hours postpartum. Other studies found that initiation of POPs, injectables, and implants at ≤6 weeks postpartum compared with nonhormonal use had no detrimental effect on breastfeeding outcomes or infant health, growth, and development in the first year postpartum. In general, these studies are of poor quality, lack standard definitions of breastfeeding or outcome measures, and have not included premature or ill infants (*50*,*51*). **Comment:** Certain women might be at risk for breastfeeding difficulties, such as women with previous breastfeeding difficulties, certain medical conditions, and certain perinatal complications and those who deliver preterm. For these women, as for all women, discussions about contraception for breastfeeding women should include information about risks, benefits, and alternatives.
ii. Without other risk factors for VTE	1	1	1	
d. >42 days postpartum	1	1	1	**Clarification:** Breastfeeding provides important health benefits for mother and infant. The U.S. Department of Health and Human Services recommends increasing the proportion of infants initially breastfed, exclusively breastfed through 6 months of life, and continuing breastfeeding through at least 1 year of life as key public health goals (*49*). **Evidence:** Overall, studies found that initiation of POPs, injectables, and implants at >6 weeks postpartum compared with nonhormonal use had no detrimental effect on breastfeeding outcomes or infant health, growth, and development in the first year postpartum. In general, these studies are of poor quality, lack standard definitions of breastfeeding or outcome measures, and have not included premature or ill infants (*51*). **Comment:** Certain women might be at risk for breastfeeding difficulties, such as women with previous breastfeeding difficulties, certain medical conditions, and certain perinatal complications and those who deliver preterm. For these women, as for all women, discussions about contraception for breastfeeding women should include information about risks, benefits, and alternatives.

See table footnotes on page 49.

TABLE C1. (*Continued*) Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category			Clarifications/Evidence/Comments
	Implants	DMPA	POPs	
Postpartum (nonbreastfeeding women)				
a. <21 days postpartum	1	1	1	—
b. 21–42 days postpartum				
i. With other risk factors for VTE (e.g., age ≥35 years, previous VTE, thrombophilia, immobility, transfusion at delivery, peripartum cardiomyopathy, BMI ≥30 kg/m^2, postpartum hemorrhage, postcesarean delivery, preeclampsia, or smoking)	1	1	1	—
ii. Without other risk factors for VTE	1	1	1	—
c. >42 days postpartum	1	1	1	—
Postabortion				
a. First trimester	1	1	1	**Clarification:** POCs may be started immediately postabortion. **Evidence:** Limited evidence suggests that no adverse side effects occur when implants (Norplant) or progestin-only injectables (NET-EN) are initiated after first trimester abortion (*52–55*).
b. Second trimester	1	1	1	**Clarification:** POCs may be started immediately postabortion.
c. Immediate postseptic abortion	1	1	1	**Clarification:** POCs may be started immediately postabortion.
Past ectopic pregnancy	1	1	2	**Comment:** POP users have a higher absolute rate of ectopic pregnancy than do users of other POCs but still lower than women using no method.
History of pelvic surgery	1	1	1	—
Smoking				
a. Age <35 years	1	1	1	—
b. Age ≥35 years				
i. <15 cigarettes per day	1	1	1	—
ii. ≥15 cigarettes per day	1	1	1	—
Obesity				—
a. BMI ≥30 kg/m^2	1	1	1	—
b. Menarche to <18 years and BMI ≥30 kg/m^2	1	2	1	**Evidence:** Among adult women, generally no association has been found between baseline weight and weight gain among DMPA users compared with nonusers. Evidence is mixed for adolescent DMPA users, with some studies observing greater weight gain among obese compared with normal weight users but other studies showing no association; methodologic differences across studies might account for the differences in findings. Data on other POC methods and other adverse outcomes including weight gain are limited (*56–73*).
History of bariatric surgery This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).				
a. Restrictive procedures: decrease storage capacity of the stomach (vertical banded gastroplasty, laparoscopic adjustable gastric band, or laparoscopic sleeve gastrectomy)	1	1	1	**Evidence:** Limited evidence demonstrated no substantial decrease in effectiveness of oral contraceptives among women who underwent laparoscopic placement of an adjustable gastric band (*74*).
b. Malabsorptive procedures: decrease absorption of nutrients and calories by shortening the functional length of the small intestine (Roux-en-Y gastric bypass or biliopancreatic diversion)	1	1	3	**Evidence:** Limited evidence demonstrated no substantial decrease in effectiveness of oral contraceptives among women who underwent a biliopancreatic diversion; however, evidence from pharmacokinetic studies suggested conflicting results regarding oral contraceptive effectiveness among women who underwent a jejunoileal bypass (*74*). **Comment:** Bariatric surgical procedures involving a malabsorptive component have the potential to decrease oral contraceptive effectiveness, perhaps further decreased by postoperative complications such as long-term diarrhea, vomiting, or both.

See table footnotes on page 49.

TABLE C1. (*Continued*) Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category			Clarifications/Evidence/Comments
	Implants	DMPA	POPs	
Cardiovascular Disease				
Multiple risk factors for atherosclerotic cardiovascular disease (e.g., older age, smoking, diabetes, hypertension, low HDL, high LDL, or high triglyceride levels)	2	3	2	**Clarification:** When multiple major risk factors exist, risk for cardiovascular disease might increase substantially. Certain POCs might increase the risk for thrombosis, although this increase is substantially less than with COCs. The effects of DMPA might persist for some time after discontinuation. **Clarification:** The recommendations apply to known preexisting medical conditions or characteristics. Few if any screening tests are needed before initiation of contraception. See the *U.S. Selected Practice Recommendations for Contraceptive Use* (http://www.cdc.gov/reproductivehealth/unintendedpregnancy/usspr.htm).
Hypertension Systolic blood pressure ≥160 mm Hg or diastolic blood pressure ≥100 mm Hg are associated with increased risk for adverse health events as a result of pregnancy (Box 2).				
a. Adequately controlled hypertension	1	2	1	**Clarification:** For all categories of hypertension, classifications are based on the assumption that no other risk factors exist for cardiovascular disease. When multiple risk factors do exist, risk for cardiovascular disease might increase substantially. A single reading of blood pressure level is not sufficient to classify a woman as hypertensive. **Clarification:** Women adequately treated for hypertension are at lower risk for acute myocardial infarction and stroke than are untreated women. Although no data exist, POC users with adequately controlled and monitored hypertension should be at lower risk for acute myocardial infarction and stroke than are untreated hypertensive POC users.
b. Elevated blood pressure levels (properly taken measurements)				**Clarification:** For all categories of hypertension, classifications are based on the assumption that no other risk factors exist for cardiovascular disease. When multiple risk factors do exist, risk for cardiovascular disease might increase substantially. A single reading of blood pressure level is not sufficient to classify a woman as hypertensive. **Evidence:** Limited evidence suggests that among women with hypertension, those who used POPs or progestin-only injectables had a small increased risk for cardiovascular events compared with women who did not use these methods (*75*).
i. Systolic 140–159 mm Hg or diastolic 90–99 mm Hg	1	2	1	
ii. Systolic ≥160 mm Hg or diastolic ≥100 mm Hg	2	3	2	
c. Vascular disease	2	3	2	**Clarification:** For all categories of hypertension, classifications are based on the assumption that no other risk factors exist for cardiovascular disease. When multiple risk factors do exist, risk for cardiovascular disease might increase substantially. A single reading of blood pressure level is not sufficient to classify a woman as hypertensive. **Comment:** Concern exists about hypoestrogenic effects and reduced HDL levels, particularly among users of DMPA. However, little concern exists about these effects with regard to POPs. The effects of DMPA might persist for some time after discontinuation.
History of high blood pressure during pregnancy (when current blood pressure is measurable and normal)	1	1	1	—
Deep venous thrombosis/Pulmonary embolism				
a. History of DVT/PE, not receiving anticoagulant therapy				

See table footnotes on page 49.

TABLE C1. (*Continued*) Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category					Clarifications/Evidence/Comments
	Implants		DMPA	POPs		
i. Higher risk for recurrent DVT/PE (one or more risk factors) • History of estrogen-associated DVT/PE • Pregnancy-associated DVT/PE • Idiopathic DVT/PE • Known thrombophilia, including antiphospholipid syndrome • Active cancer (metastatic, receiving therapy, or within 6 months after clinical remission), excluding nonmelanoma skin cancer • History of recurrent DVT/PE	2		2	2		—
ii. Lower risk for recurrent DVT/PE (no risk factors)	2		2	2		—
b. Acute DVT/PE	2		2	2		**Evidence:** No direct evidence exists on use of POCs among women with acute DVT/PE. Although findings on the risk for venous thrombosis with use of POCs in otherwise healthy women is inconsistent, any small increased risk is substantially less than that with COCs (*75–77*).
c. DVT/PE and established anticoagulant therapy for at least 3 months						**Evidence:** No direct evidence exists on use of POCs among women with DVT/PE receiving anticoagulant therapy. Although findings on the risk for venous thrombosis with use of POCs is inconsistent in otherwise healthy women, any small increased risk is substantially less than that with COCs (*75–77*). Limited evidence indicates that intramuscular injections of DMPA in women receiving chronic anticoagulation therapy does not pose a significant risk for hematoma at the injection site or increase the risk for heavy or irregular vaginal bleeding (*78*).
i. Higher risk for recurrent DVT/PE (one or more risk factors) • Known thrombophilia, including antiphospholipid syndrome • Active cancer (metastatic, receiving therapy, or within 6 months after clinical remission), excluding nonmelanoma skin cancer • History of recurrent DVT/PE	2		2	2		
ii. Lower risk for recurrent DVT/PE (no risk factors)	2		2	2		
d. Family history (first-degree relatives)	1		1	1		—
e. Major surgery						
i. With prolonged immobilization	2		2	2		—
ii. Without prolonged immobilization	1		1	1		—
f. Minor surgery without immobilization	1		1	1		—
Known thrombogenic mutations (e.g., factor V Leiden; prothrombin mutation; and protein S, protein C, and antithrombin deficiencies) This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	2		2	2		**Clarification:** Routine screening is not appropriate because of the rarity of the conditions and the high cost of screening.
Superficial venous disorders						
a. Varicose veins	1		1	1		—
b. Superficial venous thrombosis (acute or history)	1		1	1		—
Current and history of ischemic heart disease This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	Initiation 2	Continuation 3	3	Initiation 2	Continuation 3	**Comment:** Concern exists about hypoestrogenic effects and reduced HDL levels, particularly among users of DMPA. However, little concern exists about these effects with regard to POPs. The effects of DMPA might persist for some time after discontinuation.
Stroke (history of cerebrovascular accident) This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	Initiation 2	Continuation 3	3	Initiation 2	Continuation 3	**Comment:** Concern exists about hypoestrogenic effects and reduced HDL levels, particularly among users of DMPA. However, little concern exists about these effects with regard to POPs. The effects of DMPA might persist for some time after discontinuation.
Valvular heart disease Complicated valvular heart disease is associated with increased risk for adverse health events as a result of pregnancy (Box 2).						
a. Uncomplicated	1		1	1		—

See table footnotes on page 49.

TABLE C1. (*Continued*) Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category				Clarifications/Evidence/Comments
	Implants	DMPA		POPs	
b. Complicated (pulmonary hypertension, risk for atrial fibrillation, or history of subacute bacterial endocarditis)	1	1		1	—
Peripartum cardiomyopathy This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).					**Evidence:** No direct evidence exists on the safety of POCs among women with peripartum cardiomyopathy. Limited indirect evidence from noncomparative studies of women with cardiac disease demonstrated few cases of hypertension, thromboembolism, and heart failure in women with cardiac disease using POPs and DMPA (*79*).
a. Normal or mildly impaired cardiac function (New York Heart Association Functional Class I or II: patients with no limitation of activities or patients with slight, mild limitation of activity) (*80*)					**Comment:** Progestin-only implants might induce cardiac arrhythmias in healthy women; women with peripartum cardiomyopathy have a high incidence of cardiac arrhythmias.
i. <6 months	1	1		1	
ii. ≥6 months	1	1		1	
b. Moderately or severely impaired cardiac function (New York Heart Association Functional Class III or IV: patients with marked limitation of activity or patients who should be at complete rest) (*80*)	2	2		2	
Rheumatic Diseases					
Systemic lupus erythematosus This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).		Initiation	Continuation		
a. Positive (or unknown) antiphospholipid antibodies	3	3	3	3	**Clarification:** Persons with SLE are at increased risk for ischemic heart disease, stroke, and VTE. Categories assigned to such conditions in U.S. MEC should be the same for women with SLE who have these conditions. For all subconditions of SLE, classifications are based on the assumption that no other risk factors for cardiovascular disease are present; these classifications must be modified in the presence of such risk factors. Many women with SLE can be considered good candidates for most contraceptive methods, including hormonal contraceptives (*81–99*). **Evidence:** Antiphospholipid antibodies are associated with a higher risk for both arterial and venous thrombosis (*100*,*101*).
b. Severe thrombocytopenia	2	3	2	2	**Clarification:** Persons with SLE are at increased risk for ischemic heart disease, stroke, and VTE. Categories assigned to such conditions in U.S. MEC should be the same for women with SLE who have these conditions. For all subconditions of SLE, classifications are based on the assumption that no other risk factors for cardiovascular disease are present; these classifications must be modified in the presence of such risk factors. Many women with SLE can be considered good candidates for most contraceptive methods, including hormonal contraceptives (*81–99*). **Comment:** Severe thrombocytopenia increases the risk for bleeding. POCs might be useful in treating menorrhagia in women with severe thrombocytopenia. However, given the increased or erratic bleeding that might be seen on initiation of DMPA and its irreversibility for 11–13 weeks after administration, initiation of this method in women with severe thrombocytopenia should be done with caution.
c. Immunosuppressive therapy	2	2	2	2	**Clarification:** Persons with SLE are at increased risk for ischemic heart disease, stroke, and VTE. Categories assigned to such conditions in U.S. MEC should be the same for women with SLE who have these conditions. For all subconditions of SLE, classifications are based on the assumption that no other risk factors for cardiovascular disease are present; these classifications must be modified in the presence of such risk factors. Many women with SLE can be considered good candidates for most contraceptive methods, including hormonal contraceptives (*81–99*).

See table footnotes on page 49.

TABLE C1. (*Continued*) Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category				Clarifications/Evidence/Comments
	Implants	DMPA		POPs	
		Initiation	Continuation		
d. None of the above	2	2	2	2	**Clarification:** Persons with SLE are at increased risk for ischemic heart disease, stroke, and VTE. Categories assigned to such conditions in U.S. MEC should be the same for women with SLE who have these conditions. For all subconditions of SLE, classifications are based on the assumption that no other risk factors for cardiovascular disease are present; these classifications must be modified in the presence of such risk factors. Many women with SLE can be considered good candidates for most contraceptive methods, including hormonal contraceptives (*81–99*).
Rheumatoid arthritis					
a. Receiving immunosuppressive therapy	1	2/3		1	**Clarification (DMPA):** DMPA use among women receiving long-term corticosteroid therapy with a history of, or with risk factors for, nontraumatic fractures is classified as category 3. Otherwise, DMPA use for women with rheumatoid arthritis is classified as category 2. **Evidence:** Limited evidence shows no consistent pattern of improvement or worsening of rheumatoid arthritis with use of oral contraceptives, progesterone, or estrogen (*102*).
b. Not receiving immunosuppressive therapy	1	2		1	**Evidence:** Limited evidence shows no consistent pattern of improvement or worsening of rheumatoid arthritis with use of oral contraceptives, progesterone, or estrogen (*102*).
Neurologic Conditions					
Headaches					
a. Nonmigraine (mild or severe)	1	1		1	—
b. Migraine					**Evidence:** No studies directly examined the risk for stroke among women with migraine using POCs (*103*). Limited evidence demonstrated that women using POPs, DMPA, or implants do not have an increased risk for ischemic stroke compared with nonusers (*104*).
i. Without aura (This category of migraine includes menstrual migraine.)	1	1		1	
ii. With aura	1	1		1	**Comment:** Menstrual migraine is a subtype of migraine without aura. For more information, see *The International Headache Society Classification, 3rd edition* (http://www.ihs-classification.org/_downloads/mixed/International-Headache-Classification-III-ICHD-III-2013-Beta.pdf).
Epilepsy This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	1	1		1	**Clarification:** If a woman is taking anticonvulsants, see Drug Interactions section. Certain anticonvulsants lower POC effectiveness.
Multiple sclerosis					**Evidence:** Limited evidence suggests that use of COCs or oral contraceptives (type not specified) among women with multiple sclerosis does not worsen the clinical course of disease (*105*). **Comment:** Women with multiple sclerosis might have compromised bone health from disease-related disability, immobility, and use of corticosteroids. Use of DMPA, which has been associated with small changes in BMD, might be of concern.
a. With prolonged immobility	1	2		1	
b. Without prolonged immobility	1	2		1	
Depressive Disorders					
Depressive disorders	1	1		1	**Clarification:** If a woman is taking psychotropic medications or St. John's wort, see Drug Interactions section. **Evidence:** The frequency of psychiatric hospitalizations for women with bipolar disorder or depression did not significantly differ among women using DMPA, LNG-IUD, Cu-IUD, or sterilization (*106*).
Reproductive Tract Infections and Disorders					
Vaginal bleeding patterns					
a. Irregular pattern without heavy bleeding	2	2		2	**Comment:** Irregular menstrual bleeding patterns are common among healthy women. POC use frequently induces an irregular bleeding pattern. Implant use might induce irregular bleeding patterns, especially during the first 3–6 months, although these patterns might persist longer.
b. Heavy or prolonged bleeding (includes regular and irregular patterns)	2	2		2	**Clarification:** Unusually heavy bleeding should raise the suspicion of a serious underlying condition.

See table footnotes on page 49.

TABLE C1. (*Continued*) Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category			Clarifications/Evidence/Comments
	Implants	DMPA	POPs	
Unexplained vaginal bleeding (suspicious for serious condition) before evaluation	3	3	2	**Clarification:** If pregnancy or an underlying pathological condition (e.g., pelvic malignancy) is suspected, it must be evaluated and the category adjusted after evaluation. **Comment:** POCs might cause irregular bleeding patterns, which might mask symptoms of underlying pathologic conditions. The effects of DMPA might persist for some time after discontinuation.
Endometriosis	1	1	1	—
Benign ovarian tumors (including cysts)	1	1	1	—
Severe dysmenorrhea	1	1	1	—
Gestational trophoblastic disease This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).				**Clarification:** For all subconditions of gestational trophoblastic disease, classifications are based on the assumption that women are under close medical supervision because of the need for monitoring of β-hCG levels for appropriate disease surveillance.
a. Suspected gestational trophoblastic disease (immediate postevacuation)				
i. Uterine size first trimester	1	1	1	
ii. Uterine size second trimester	1	1	1	
b. Confirmed gestational trophoblastic disease (after initial evacuation and during monitoring)				
i. Undetectable/nonpregnant β–hCG levels	1	1	1	
ii. Decreasing β–hCG levels	1	1	1	
iii. Persistently elevated β-hCG levels or malignant disease, with no evidence or suspicion of intrauterine disease	1	1	1	
iv. Persistently elevated β-hCG levels or malignant disease, with evidence or suspicion of intrauterine disease	1	1	1	
Cervical ectropion	1	1	1	—
Cervical intraepithelial neoplasia	2	2	1	**Evidence:** Among women with persistent human papillomavirus infection, long-term DMPA use (≥5 years) might increase the risk for carcinoma in situ and invasive carcinoma (*107*).
Cervical cancer (awaiting treatment)	2	2	1	**Comment:** Theoretical concern exists that POC use might affect prognosis of the existing disease. While awaiting treatment, women may use POCs. In general, treatment of this condition can render a woman sterile.
Breast disease Breast cancer is associated with increased risk for adverse health events as a result of pregnancy (Box 2).				
a. Undiagnosed mass	2	2	2	**Clarification:** Evaluation should be pursued as early as possible.
b. Benign breast disease	1	1	1	—
c. Family history of cancer	1	1	1	—
d. Breast cancer				**Comment:** Breast cancer is a hormonally sensitive tumor, and the prognosis for women with current or recent breast cancer might worsen with POC use.
i. Current	4	4	4	
ii. Past and no evidence of current disease for 5 years	3	3	3	
Endometrial hyperplasia	1	1	1	—
Endometrial cancer This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	1	1	1	**Comment:** While awaiting treatment, women may use POCs. In general, treatment of this condition renders a woman sterile.
Ovarian cancer This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	1	1	1	**Comment:** While awaiting treatment, women may use POCs. In general, treatment of this condition can render a woman sterile.
Uterine fibroids	1	1	1	**Comment:** POCs do not appear to cause growth of uterine fibroids.

See table footnotes on page 49.

TABLE C1. (*Continued*) Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category			Clarifications/Evidence/Comments
	Implants	DMPA	POPs	
Pelvic inflammatory disease				**Comment:** Whether POCs, like COCs, reduce the risk for PID among women with STDs is unknown; however, they do not protect against HIV or lower genital tract STDs.
a. Past PID				
i. With subsequent pregnancy	1	1	1	
ii. Without subsequent pregnancy	1	1	1	
b. Current PID	1	1	1	
Sexually transmitted diseases				
a. Current purulent cervicitis or chlamydial infection or gonococcal infection	1	1	1	—
b. Vaginitis (including *Trichomonas vaginalis* and bacterial vaginosis)	1	1	1	—
c. Other factors related to STDs	1	1	1	—
HIV				
High risk for HIV	1	1	1	**Clarification (DMPA):** Some studies suggest that women using progestin-only injectable contraception might be at increased risk for HIV acquisition; other studies do not show this association. CDC reviewed all available evidence and agreed that the data were not sufficiently conclusive to change current guidance. However, because of the inconclusive nature of the body of evidence on possible increased risk for HIV acquisition, women using progestin-only injectable contraception should be strongly advised to also always use condoms (male or female) and take other HIV preventive measures. Expansion of contraceptive method mix and further research on the relationship between hormonal contraception and HIV infection are essential. These recommendations will be continually reviewed in light of new evidence. **Evidence:** Overall, evidence does not support an association between oral contraceptives and risk for HIV acquisition, evidence is inconsistent regarding an association between DMPA and increased risk for HIV acquisition, and no studies have suggested an increased risk for HIV acquisition with etonogestrel implants although data are limited (*108*).
HIV infection For women with HIV infection who are not clinically well or not using ARV therapy, this condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	1	1	1	**Clarification:** Drug interactions might exist between hormonal contraceptives and ARV drugs; see Drug Interactions section. **Evidence:** Overall, evidence does not support an association between POC use and progression of HIV. Limited direct evidence on an association between POC use and transmission of HIV to noninfected partners, as well as studies measuring genital viral shedding as a proxy for infectivity, have had mixed results. Studies measuring whether hormonal contraceptive methods affect plasma HIV viral load generally have found no effect (*109–111*).
Other Infections				
Schistosomiasis Schistosomiasis with fibrosis of the liver is associated with increased risk for adverse health events as a result of pregnancy (Box 2).				
a. Uncomplicated	1	1	1	**Evidence:** Among women with uncomplicated schistosomiasis, limited evidence showed that DMPA use had no adverse effects on liver function (*112*).
b. Fibrosis of the liver (if severe, see Cirrhosis section)	1	1	1	—
Tuberculosis This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).				**Clarification:** If a woman is taking rifampin, see Drug Interactions section. Rifampin is likely to decrease the effectiveness of some POCs.
a. Nonpelvic	1	1	1	
b. Pelvic	1	1	1	
Malaria	1	1	1	—

See table footnotes on page 49.

TABLE C1. (*Continued*) Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category			Clarifications/Evidence/Comments
	Implants	DMPA	POPs	
Endocrine Conditions				
Diabetes Insulin-dependent diabetes; diabetes with nephropathy, retinopathy, or neuropathy; diabetes with other vascular disease; or diabetes of >20 years' duration are associated with increased risk for adverse health events as a result of pregnancy (Box 2).				
a. History of gestational disease	1	1	1	**Evidence:** POCs had no adverse effects on serum lipid levels in women with a history of gestational diabetes in two small studies (*113*,*114*). Limited evidence is inconsistent about the development of noninsulin-dependent diabetes among users of POCs with a history of gestational diabetes (*115*–*118*).
b. Nonvascular disease				**Evidence:** Among women with insulin-dependent or non–insulin-dependent diabetes, limited evidence on use of POCs (POPs, DMPA, and LNG implant) suggests that these methods have little effect on short-term or long-term diabetes control (e.g., glycosylated hemoglobin levels), hemostatic markers, or lipid profile (*119*–*122*).
i. Non-insulin dependent	2	2	2	
ii. Insulin dependent	2	2	2	
c. Nephropathy, retinopathy, or neuropathy	2	3	2	**Comment:** Concern exists about hypoestrogenic effects and reduced HDL levels, particularly among users of DMPA. The effects of DMPA might persist for some time after discontinuation. Some POCs might increase the risk for thrombosis, although this increase is substantially less than with COCs.
d. Other vascular disease or diabetes of >20 years' duration	2	3	2	**Comment:** Concern exists about hypoestrogenic effects and reduced HDL levels, particularly among users of DMPA. The effects of DMPA might persist for some time after discontinuation. Some POCs might increase the risk for thrombosis, although this increase is substantially less than with COCs.
Thyroid disorders				
a. Simple goiter	1	1	1	—
b. Hyperthyroid	1	1	1	—
c. Hypothyroid	1	1	1	—
Gastrointestinal Conditions				
Inflammatory bowel disease (ulcerative colitis or Crohn's disease)	1	2	2	**Evidence:** Risk for disease relapse among women with IBD using oral contraceptives (most studies did not specify formulation) did not increase significantly from that for nonusers (*123*). **Comment:** Absorption of POPs among women with IBD might be reduced if the woman has substantial malabsorption caused by severe disease or small bowel surgery. Women with IBD have a higher prevalence of osteoporosis and osteopenia than the general population. Use of DMPA, which has been associated with small changes in BMD, might be of concern.
Gallbladder disease				
a. Symptomatic				
i. Treated by cholecystectomy	2	2	2	—
ii. Medically treated	2	2	2	—
iii. Current	2	2	2	—
b. Asymptomatic	2	2	2	—
History of cholestasis				
a. Pregnancy related	1	1	1	—
b. Past COC related	2	2	2	**Comment:** Theoretical concern exists that a history of COC-related cholestasis might predict subsequent cholestasis with POC use. However, this has not been documented.
Viral hepatitis				
a. Acute or flare	1	1	1	—
b. Carrier	1	1	1	—
c. Chronic	1	1	1	—
Cirrhosis Severe cirrhosis is associated with increased risk for adverse health events as a result of pregnancy (Box 2).				
a. Mild (compensated)	1	1	1	—
b. Severe (decompensated)	3	3	3	—

See table footnotes on page 49.

TABLE C1. (*Continued*) Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category			Clarifications/Evidence/Comments
	Implants	DMPA	POPs	
Liver tumors Hepatocellular adenoma and malignant liver tumors are associated with increased risk for adverse health events as a result of pregnancy (Box 2).				
a. Benign				
i. Focal nodular hyperplasia	2	2	2	**Evidence:** Limited direct evidence suggests that hormonal contraceptive use does not influence either progression or regression of liver lesions among women with focal nodular hyperplasia (*124*).
ii. Hepatocellular adenoma	3	3	3	**Comment:** No evidence is available about hormonal contraceptive use among women with hepatocellular adenoma. COC use in healthy women is associated with development and growth of hepatocellular adenoma; whether other hormonal contraceptives have similar effects is not known.
b. Malignant (hepatoma)	3	3	3	—
Respiratory Conditions				
Cystic fibrosis This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	1	2	1	**Clarification:** Persons with cystic fibrosis are at increased risk for diabetes, liver disease, gallbladder disease, and VTE (particularly related to use of central venous catheters) and are frequently prescribed antibiotics. Categories assigned to such conditions in U.S. MEC should be the same for women with cystic fibrosis who have these conditions. For cystic fibrosis, classifications are based on the assumption that no other conditions are present; these classifications must be modified in the presence of such conditions. **Clarification:** Certain drugs to treat cystic fibrosis (e.g., lumacaftor) might reduce effectiveness of hormonal contraceptives, including oral, injectable, transdermal, and implantable contraceptives. **Evidence:** Limited evidence suggests that use of COCs or oral contraceptives (type not specified) among women with cystic fibrosis is not associated with worsening of disease severity. Very limited evidence suggests that cystic fibrosis does not impair the effectiveness of hormonal contraception (*125*). **Comment:** Women with cystic fibrosis have a higher prevalence of osteopenia, osteoporosis, and fragility fractures than the general population. Use of DMPA, which has been associated with small changes in BMD, might be of concern.
Anemias				
Thalassemia	1	1	1	—
Sickle cell disease This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	1	1	1	**Evidence:** Among women with sickle cell disease, POC use did not have adverse effects on hematologic parameters and, in some studies, was beneficial with respect to clinical symptoms (*126–133*).
Iron deficiency anemia	1	1	1	**Comment:** Changes in the menstrual pattern associated with POC use have little effect on hemoglobin levels.
Solid Organ Transplantation				
Solid organ transplantation This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).				
a. Complicated: graft failure (acute or chronic), rejection, or cardiac allograft vasculopathy	2	2	2	—
b. Uncomplicated	2	2	2	—

See table footnotes on page 49.

TABLE C1. (*Continued*) Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category			Clarifications/Evidence/Comments
	Implants	DMPA	POPs	
Drug Interactions				
Antiretroviral therapy				**Comment:** These recommendations generally are for ARV agents used alone. However, most women receiving ARV therapy are using multiple drugs in combination. In general, whether interactions between ARVs and hormonal contraceptives differ when ARVs are given alone or in combination is unknown.
a. Nucleoside reverse transcriptase inhibitors (NRTIs)				
i. Abacavir (ABC)	1	1	1	**Evidence:** NRTIs do not appear to have significant risk for interactions with hormonal contraceptive methods (*134–139*).
ii. Tenofovir (TDF)	1	1	1	
iii. Zidovudine (AZT)	1	1	1	
iv. Lamivudine (3TC)	1	1	1	
v. Didanosine (DDI)	1	1	1	
vi. Emtricitabine (FTC)	1	1	1	
vii. Stavudine (D4T)	1	1	1	
b. Nonnucleoside reverse transcriptase inhibitors (NNRTIs)				
i. Efavirenz (EFV)	2	1	2	**Clarification:** Evidence suggests drug interactions between EFV and certain hormonal contraceptives. These interactions might reduce the effectiveness of the hormonal contraceptive. **Evidence:** One study found that women using etonogestrel implants with EFV had a higher pregnancy rate than women not using ARVs, although confidence intervals overlapped and absolute pregnancy rates were still lower than for other hormonal methods; another study found that etonogestrel levels were decreased and 5% of women had presumptive ovulation while using etonogestrel implants with EFV (*140*,*141*). Three studies of women using LNG implants showed increased pregnancy rates for women using EFV-containing ARV therapy compared with no ARV use, although absolute pregnancy rates were still lower than for other hormonal methods in one study (*141–143*); another study of LNG implant users found no difference in pregnancy rates with EFV compared with no EFV (*144*).No significant effects were found on pregnancy rates, DMPA levels, EFV levels, or HIV disease progression in women using DMPA and EFV compared with DMPA alone (*141*,*144–148*). No significant effects were found on HIV disease progression in women using LNG implants and EFV compared with no ARVs (*143*). No data have assessed effectiveness of contraceptive implants during later years of use when progestin concentrations are lower and risk for failure from drug interactions might be greater.
ii. Etravirine (ETR)	1	1	1	—
iii. Nevirapine (NVP)	1	1	1	**Evidence:** Five studies found no significant increase in pregnancy rates among women using implants and NVP compared with implants alone (*141–144*,*149*). Four studies found no significant increase in pregnancy rates among women using DMPA or other contraceptive injectables and NVP compared with DMPA or other contraceptive injectables alone (*141*,*144*,*147*,*150*). One study found no ovulations or changes in DMPA concentrations (*145*). No effect was found on HIV disease progression with use of NVP and DMPA or LNG implants (*143*,*145*,*147–149*,*151*). No data have assessed effectiveness of contraceptive implants during later years of use when progestin concentrations are lower and risk for failure from drug interactions might be greater.
iv. Rilpivirine (RPV)	1	1	1	—

See table footnotes on page 49.

TABLE C1. (*Continued*) Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category			Clarifications/Evidence/Comments
	Implants	DMPA	POPs	
c. Ritonavir-boosted protease inhibitors				
i. Ritonavir-boosted atazanavir (ATV/r)	2	1	2	**Clarification:** Theoretically, drug interactions might occur between certain ritonavir-boosted protease inhibitors and certain hormonal contraceptives that might reduce the effectiveness of the hormonal contraceptive. Any potential effect on contraceptive effectiveness is likely to be lower with DMPA than with other POCs because of the higher dose of DMPA. **Evidence:** One pharmacokinetic study demonstrated increased progestin concentrations with use of POPs and ATV/r compared with POPs alone (*152*).
ii. Ritonavir-boosted darunavir (DRV/r)	2	1	2	**Clarification:** Theoretically, drug interactions might occur between certain ritonavir-boosted protease inhibitors and certain hormonal contraceptives that might reduce the effectiveness of the hormonal contraceptive. Any potential effect on contraceptive effectiveness is likely to be lower with DMPA than with other POCs because of the higher dose of DMPA.
iii. Ritonavir-boosted fosamprenavir (FPV/r)	2	1	2	**Clarification:** Theoretically, drug interactions might occur between certain ritonavir-boosted protease inhibitors and certain hormonal contraceptives that might reduce the effectiveness of the hormonal contraceptive. Any potential effect on contraceptive effectiveness is likely to be lower with DMPA than with other POCs because of the higher dose of DMPA.
iv. Ritonavir-boosted lopinavir (LPV/r)	1	1	1	**Evidence:** One study demonstrated no pregnancies, no ovulations, no change in LPV/r level, and no change in HIV disease progression in women using DMPA (*153*); another study found a small increase in pregnancy rate in women using DMPA with LPV/r compared with no ARV therapy, however confidence intervals overlapped (*141*). Two studies found no increased risk for pregnancy in women using implants (*141*,*142*). Two studies found contraceptive hormones increased in women using LPV/r with DMPA or etonogestrel implants (*140*,*153*).
v. Ritonavir-boosted saquinavir (SQV/r)	2	1	2	**Clarification:** Theoretically, drug interactions might occur between certain ritonavir-boosted protease inhibitors and certain hormonal contraceptives that might reduce the effectiveness of the hormonal contraceptive. Any potential effect on contraceptive effectiveness is likely to be lower with DMPA than with other POCs because of the higher dose of DMPA.
vi. Ritonavir-boosted tipranavir (TPV/r)	2	1	2	**Clarification:** Theoretically, drug interactions might occur between certain ritonavir-boosted protease inhibitors and certain hormonal contraceptives that might reduce the effectiveness of the hormonal contraceptive. Any potential effect on contraceptive effectiveness is likely to be lower with DMPA than with other POCs because of the higher dose of DMPA.
d. Protease inhibitors without ritonavir				
i. Atazanavir (ATV)	1	1	1	**Comment:** When ATV is administered with Cobicistat, theoretical concern exists for a drug interaction with hormonal contraceptives. Cobicistat is an inhibitor of CYP3A and CYP2D6 and could theoretically increase contraceptive hormone levels. However, its effects on CYP enzymes and drug levels might vary when combined with other ARVs.
ii. Fosamprenavir (FPV)	2	2	2	**Clarification:** Theoretical concern exists that interactions between FPV and hormonal contraceptives leading to decreased levels of FPV might diminish effectiveness of the ARV drug. The drug interaction likely involves CYP3A4 pathways; POCs have less effect on CYP3A4 enzymes than CHCs.
iii. Indinavir (IDV)	1	1	1	—

See table footnotes on page 49.

TABLE C1. (*Continued*) Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category			Clarifications/Evidence/Comments
	Implants	DMPA	POPs	
iv. Nelfinavir (NFV)	2	1	2	**Clarification:** Theoretically, drug interactions might occur between certain protease inhibitors and certain hormonal contraceptives that might reduce the effectiveness of the hormonal contraceptive. Any potential effect on contraceptive effectiveness is likely to be lower with DMPA than with other POCs because of the higher dose of DMPA. Concern exists that interactions between NFV and POCs might decrease NFV levels. **Evidence:** One study found no pregnancies, no ovulations, no change in DMPA concentrations and no change in HIV disease progression with use of DMPA and NFV compared with DMPA alone; NFV concentrations were decreased with concomitant DMPA use (*145*,*147*).
e. CCR5 co-receptor antagonists				
i. Maraviroc (MVC)	1	1	1	
f. HIV integrase strand transfer inhibitors				
i. Raltegravir (RAL)	1	1	1	—
ii. Dolutegravir (DTG)	1	1	1	—
iii. Elvitegravir (EVG)	1	1	1	**Comment:** When EVG is administered with Cobicistat, theoretical concern exists for a drug interaction with hormonal contraceptives. Cobicistat is an inhibitor of CYP3A and CYP2D6 and could theoretically increase contraceptive hormone levels. However, its effects on CYP enzymes and drug levels may vary when combined with other ARVs.
g. Fusion inhibitors				
i. Enfuvirtide	1	1	1	—
Anticonvulsant therapy				—
a. Certain anticonvulsants (phenytoin, carbamazepine, barbiturates, primidone, topiramate, and oxcarbazepine)	2	1	3	**Clarification:** Although the interaction of certain anticonvulsants with POPs and etonogestrel implants is not harmful to women, it is likely to reduce the effectiveness of POPs and etonogestrel implants. Whether increasing the hormone dose of POPs alleviates this concern remains unclear. Use of other contraceptives should be encouraged for women who are long-term users of any of these drugs. Use of DMPA is a category 1 because its effectiveness is not decreased by use of certain anticonvulsants. **Evidence:** Use of certain anticonvulsants might decrease the effectiveness of POCs (*154*–*156*).
b. Lamotrigine	1	1	1	**Evidence:** No drug interactions have been reported among women with epilepsy receiving lamotrigine and POCs (*157*).
Antimicrobial therapy				
a. Broad-spectrum antibiotics	1	1	1	—
b. Antifungals	1	1	1	—
c. Antiparasitics	1	1	1	—
d. Rifampin or rifabutin therapy	2	1	3	**Clarification:** Although the interaction of rifampin or rifabutin with POPs and etonogestrel implants is not harmful to women, it is likely to reduce the effectiveness of POPs and etonogestrel implants. Use of other contraceptives should be encouraged for women who are long-term users of any of these drugs. Use of DMPA is a category 1 because its effectiveness is not decreased by use of rifampin or rifabutin. Whether increasing the hormone dose of POPs alleviates this concern remains unclear.

See table footnotes on page 49.

TABLE C1. (*Continued*) Classifications for progestin-only contraceptives, including implants, depot medroxyprogesterone acetate, and progestin-only pills

Condition	Category			Clarifications/Evidence/Comments
	Implants	DMPA	POPs	
Psychotropic medications				**Comment:** For many common psychotropic agents, limited or no theoretical concern exits for clinically significant drug interactions when co-administered with hormonal contraceptives. However, either no or very limited data exist examining potential interactions for these classes of medications.
a. SSRIs	1	1	1	**Evidence:** No evidence specifically examined the use of POCs with SSRIs. Limited clinical and pharmacokinetic data do not demonstrate concern for SSRIs decreasing the effectiveness of oral contraceptives. Limited evidence suggests that for women taking SSRIs, the use of hormonal contraceptives was not associated with differences in effectiveness of the SSRI for treatment or in adverse events when compared with women not taking hormonal contraceptives (*158*).
				Comment: Drugs that are inhibitors of CYP3A4 or CYP2C9 theoretically have the potential to increase levels of contraceptive steroid, which might increase adverse events. Fluvoxamine is an SSRI known to be a moderate inhibitor of both 3A4 and 2C9; however, no clinical or pharmacokinetic studies were identified to explore potential drug-drug interactions.
St. John's wort	2	1	2	**Evidence:** No evidence specifically examined the use of POCs with St John's wort. Although clinical data are limited, studies with pharmacokinetic and pharmacodynamics outcomes raise concern that St. John's wort might decrease effectiveness of hormonal contraceptives, including increased risk for breakthrough bleeding and ovulation and increased metabolism of estrogen and progestin. Any interactions might be dependent on the dose of St John's wort, and the concentration of active ingredients across types of St. John's wort preparations may vary (*159*).
				Comment: Any potential effect on contraceptive effectiveness is likely to be lower with DMPA than with other POCs because of the higher dose of DMPA.

Abbreviations: ARV = antiretroviral; BMD = bone mineral density; BMI = body mass index; COC = combined oral contraceptive; DMPA = depot medroxyprogesterone acetate; DVT = deep venous thrombosis; hCG = human chorionic gonadotropin; HDL = high-density lipoprotein; HIV = human immunodeficiency virus; IBD = inflammatory bowel disease; LDL = low-density lipoprotein; LNG = levonorgestrel; NA = not applicable; NET-EN = norethisterone enantate; PE = pulmonary embolism; PID = pelvic inflammatory disease; POC = progestin-only contraceptive; POP = progestin-only pill; SSRI = selective serotonin reuptake inhibitor; STD = sexually transmitted disease; VTE = venous thromboembolism.

References

1. Lanza LL, McQuay LJ, Rothman KJ, et al. Use of depot medroxyprogesterone acetate contraception and incidence of bone fracture. Obstet Gynecol 2013;121:593–600. http://dx.doi.org/10.1097/AOG.0b013e318283d1a1
2. Harel Z, Riggs S, Vaz R, Flanagan P, Harel D, Machan JT. Bone accretion in adolescents using the combined estrogen and progestin transdermal contraceptive method Ortho Evra: a pilot study. J Pediatr Adolesc Gynecol 2010;23:23–31. http://dx.doi.org/10.1016/j.jpag.2009.04.008
3. Kaunitz AM, Miller PD, Rice VM, Ross D, McClung MR. Bone mineral density in women aged 25–35 years receiving depot medroxyprogesterone acetate: recovery following discontinuation. Contraception 2006;74:90–9. http://dx.doi.org/10.1016/j.contraception.2006.03.010
4. Kaunitz AM, Arias R, McClung M. Bone density recovery after depot medroxyprogesterone acetate injectable contraception use. Contraception 2008;77:67–76. http://dx.doi.org/10.1016/j.contraception.2007.10.005
5. Kaunitz AM, Darney PD, Ross D, Wolter KD, Speroff L. Subcutaneous DMPA vs. intramuscular DMPA: a 2-year randomized study of contraceptive efficacy and bone mineral density. Contraception 2009;80:7–17. http://dx.doi.org/10.1016/j.contraception.2009.02.005
6. Lappe JM, Stegman MR, Recker RR. The impact of lifestyle factors on stress fractures in female Army recruits. Osteoporos Int 2001;12:35–42. http://dx.doi.org/10.1007/s001980170155
7. Lara-Torre E, Edwards CP, Perlman S, Hertweck SP. Bone mineral density in adolescent females using depot medroxyprogesterone acetate. J Pediatr Adolesc Gynecol 2004;17:17–21. http://dx.doi.org/10.1016/j.jpag.2003.11.017
8. Lopez LM, Chen M, Mullins S, Curtis KM, Helmerhorst FM. Steroidal contraceptives and bone fractures in women: evidence from observational studies. Cochrane Database Syst Rev 2012;8:CD009849.
9. Lopez LM, Grimes DA, Schulz KF, Curtis KM. Steroidal contraceptives: effect on bone fractures in women. Cochrane Database Syst Rev 2011;(7):CD006033.
10. Meier C, Brauchli YB, Jick SS, Kraenzlin ME, Meier CR. Use of depot medroxyprogesterone acetate and fracture risk. J Clin Endocrinol Metab 2010;95:4909–16. http://dx.doi.org/10.1210/jc.2010-0032
11. Merki-Feld GS, Neff M, Keller PJ. A 2-year prospective study on the effects of depot medroxyprogesterone acetate on bone mass-response to estrogen and calcium therapy in individual users. Contraception 2003;67:79–86. http://dx.doi.org/10.1016/S0010-7824(02)00460-2
12. Monteiro-Dantas C, Espejo-Arce X, Lui-Filho JF, Fernandes AM, Monteiro I, Bahamondes L. A three-year longitudinal evaluation of the forearm bone density of users of etonogestrel- and levonorgestrel-releasing contraceptive implants. Reprod Health 2007;4:11. http://dx.doi.org/10.1186/1742-4755-4-11
13. Naessen T, Olsson SE, Gudmundson J. Differential effects on bone density of progestogen-only methods for contraception in premenopausal women. Contraception 1995;52:35–9. http://dx.doi.org/10.1016/0010-7824(95)00121-P
14. Sanches L, Marchi NM, Castro S, Juliato CT, Villarroel M, Bahamondes L. Forearm bone mineral density in postmenopausal former users of depot medroxyprogesterone acetate. Contraception 2008;78:365–9. http://dx.doi.org/10.1016/j.contraception.2008.07.013
15. Scholes D, LaCroix AZ, Ichikawa LE, Barlow WE, Ott SM. Injectable hormone contraception and bone density: results from a prospective study. Epidemiology 2002;13:581–7. http://dx.doi.org/10.1097/00001648-200209000-00015

16. Scholes D, LaCroix AZ, Ichikawa LE, Barlow WE, Ott SM. Change in bone mineral density among adolescent women using and discontinuing depot medroxyprogesterone acetate contraception. Arch Pediatr Adolesc Med 2005;159:139–44. http://dx.doi.org/10.1001/archpedi.159.2.139
17. Segall-Gutierrez P, Agarwal R, Ge M, Lopez C, Hernandez G, Stanczyk FZ. A pilot study examining short-term changes in bone mineral density among class 3 obese users of depot-medroxyprogesterone acetate. Eur J Contracept Reprod Health Care 2013;18:199–205. http://dx.doi.org/10.3109/13625187.2013.774358
18. Tang OS, Tang G, Yip PS, Li B. Further evaluation on long-term depot-medroxyprogesterone acetate use and bone mineral density: a longitudinal cohort study. Contraception 2000;62:161–4. http://dx.doi.org/10.1016/S0010-7824(00)00168-2
19. Vestergaard P, Rejnmark L, Mosekilde L. The effects of depot medroxyprogesterone acetate and intrauterine device use on fracture risk in Danish women. Contraception 2008;78:459–64. http://dx.doi.org/10.1016/j.contraception.2008.07.014
20. Viola AS, Castro S, Marchi NM, Bahamondes MV, Viola CF, Bahamondes L. Long-term assessment of forearm bone mineral density in postmenopausal former users of depot medroxyprogesterone acetate. Contraception 2011;84:122–7. http://dx.doi.org/10.1016/j.contraception.2010.11.007
21. Walsh JS, Eastell R, Peel NF. Depot medroxyprogesterone acetate use after peak bone mass is associated with increased bone turnover but no decrease in bone mineral density. Fertil Steril 2010;93:697–701. http://dx.doi.org/10.1016/j.fertnstert.2008.10.004
22. Wetmore CM, Ichikawa L, LaCroix AZ, Ott SM, Scholes D. Association between caffeine intake and bone mass among young women: potential effect modification by depot medroxyprogesterone acetate use. Osteoporos Int 2008;19:519–27. http://dx.doi.org/10.1007/s00198-007-0473-2
23. Wong AY, Tang LC, Chin RK. Levonorgestrel-releasing intrauterine system (Mirena) and depot medroxyprogesterone acetate (Depoprovera) as long-term maintenance therapy for patients with moderate and severe endometriosis: a randomised controlled trial. Aust N Z J Obstet Gynaecol 2010;50:273–9. http://dx.doi.org/10.1111/j.1479-828X.2010.01152.x
24. Yang KY, Kim YS, Ji YI, Jung MH. Changes in bone mineral density of users of the levonorgestrel-releasing intrauterine system. J Nippon Med Sch 2012;79:190–4. http://dx.doi.org/10.1272/jnms.79.190
25. Zhang MH, Zhang W, Zhang AD, Yang Y, Gai L. Effect of depot medroxyprogesterone acetate on bone mineral density in adolescent women. Chin Med J (Engl) 2013;126:4043–7.
26. Bahamondes MV, Monteiro I, Castro S, Espejo-Arce X, Bahamondes L. Prospective study of the forearm bone mineral density of long-term users of the levonorgestrel-releasing intrauterine system. Hum Reprod 2010;25:1158–64. http://dx.doi.org/10.1093/humrep/deq043
27. Banks E, Berrington A, Casabonne D. Overview of the relationship between use of progestogen-only contraceptives and bone mineral density. BJOG 2001;108:1214–21. http://dx.doi.org/10.1111/j.1471-0528.2001.00296.x
28. Beerthuizen R, van Beek A, Massai R, Mäkäräinen L, Hout J, Bennink HC. Bone mineral density during long-term use of the progestagen contraceptive implant Implanon compared to a non-hormonal method of contraception. Hum Reprod 2000;15:118–22. http://dx.doi.org/10.1093/humrep/15.1.118
29. Beksinska ME, Kleinschmidt I, Smit JA, Farley TM. Bone mineral density in adolescents using norethisterone enanthate, depot-medroxyprogesterone acetate or combined oral contraceptives for contraception. Contraception 2007;75:438–43. http://dx.doi.org/10.1016/j.contraception.2007.02.001
30. Beksinska ME, Kleinschmidt I, Smit JA, Farley TM. Bone mineral density in a cohort of adolescents during use of norethisterone enanthate, depot-medroxyprogesterone acetate or combined oral contraceptives and after discontinuation of norethisterone enanthate. Contraception 2009;79:345–9. http://dx.doi.org/10.1016/j.contraception.2008.11.009
31. Berenson AB, Breitkopf CR, Grady JJ, Rickert VI, Thomas A. Effects of hormonal contraception on bone mineral density after 24 months of use. Obstet Gynecol 2004;103:899–906. http://dx.doi.org/10.1097/01.AOG.0000117082.49490.d5
32. Berenson AB, Rahman M, Breitkopf CR, Bi LX. Effects of depot medroxyprogesterone acetate and 20-microgram oral contraceptives on bone mineral density. Obstet Gynecol 2008;112:788–99. http://dx.doi.org/10.1097/AOG.0b013e3181875b78
33. Busen NH, Britt RB, Rianon N. Bone mineral density in a cohort of adolescent women using depot medroxyprogesterone acetate for one to two years. J Adolesc Health 2003;32:257–9. http://dx.doi.org/10.1016/S1054-139X(02)00567-0
34. Caird LE, Reid-Thomas V, Hannan WJ, Gow S, Glasier AF. Oral progestogen-only contraception may protect against loss of bone mass in breast-feeding women. Clin Endocrinol (Oxf) 1994;41:739–45. http://dx.doi.org/10.1111/j.1365-2265.1994.tb02788.x
35. Clark MK, Sowers M, Levy B, Nichols S. Bone mineral density loss and recovery during 48 months in first-time users of depot medroxyprogesterone acetate. Fertil Steril 2006;86:1466–74. http://dx.doi.org/10.1016/j.fertnstert.2006.05.024
36. Cromer BA, Lazebnik R, Rome E, et al. Double-blinded randomized controlled trial of estrogen supplementation in adolescent girls who receive depot medroxyprogesterone acetate for contraception. Am J Obstet Gynecol 2005;192:42–7. http://dx.doi.org/10.1016/j.ajog.2004.07.041
37. Cromer BA, Blair JM, Mahan JD, Zibners L, Naumovski Z. A prospective comparison of bone density in adolescent girls receiving depot medroxyprogesterone acetate (Depo-Provera), levonorgestrel (Norplant), or oral contraceptives. J Pediatr 1996;129:671–6. http://dx.doi.org/10.1016/S0022-3476(96)70148-8
38. Cromer BA, Bonny AE, Stager M, et al. Bone mineral density in adolescent females using injectable or oral contraceptives: a 24-month prospective study. Fertil Steril 2008;90:2060–7. http://dx.doi.org/10.1016/j.fertnstert.2007.10.070
39. Cromer BA, Stager M, Bonny A, et al. Depot medroxyprogesterone acetate, oral contraceptives and bone mineral density in a cohort of adolescent girls. J Adolesc Health 2004;35:434–41. http://dx.doi.org/10.1016/j.jadohealth.2004.07.005
40. Cundy T, Ames R, Horne A, et al. A randomized controlled trial of estrogen replacement therapy in long-term users of depot medroxyprogesterone acetate. J Clin Endocrinol Metab 2003;88:78–81. http://dx.doi.org/10.1210/jc.2002-020874
41. Cundy T, Cornish J, Evans MC, Roberts H, Reid IR. Recovery of bone density in women who stop using medroxyprogesterone acetate. BMJ 1994;308:247–8. http://dx.doi.org/10.1136/bmj.308.6923.247
42. Cundy T, Cornish J, Roberts H, Reid IR. Menopausal bone loss in long-term users of depot medroxyprogesterone acetate contraception. Am J Obstet Gynecol 2002;186:978–83. http://dx.doi.org/10.1067/mob.2002.122420
43. Di X, Li Y, Zhang C, Jiang J, Gu S. Effects of levonorgestrel-releasing subdermal contraceptive implants on bone density and bone metabolism. Contraception 1999;60:161–6. http://dx.doi.org/10.1016/S0010-7824(99)00080-3
44. Díaz S, Reyes MV, Zepeda A, et al. Norplant® implants and progesterone vaginal rings do not affect maternal bone turnover and density during lactation and after weaning. Hum Reprod 1999;14:2499–505. http://dx.doi.org/10.1093/humrep/14.10.2499
45. Gai L, Zhang J, Zhang H, Gai P, Zhou L, Liu Y. The effect of depot medroxyprogesterone acetate (DMPA) on bone mineral density (BMD) and evaluating changes in BMD after discontinuation of DMPA in Chinese women of reproductive age. Contraception 2011;83:218–22. http://dx.doi.org/10.1016/j.contraception.2010.07.027
46. Bahamondes L, Espejo-Arce X, Hidalgo MM, Hidalgo-Regina C, Teatin-Juliato C, Petta CA. A cross-sectional study of the forearm bone density of long-term users of levonorgestrel-releasing intrauterine system. Hum Reprod 2006;21:1316–9. http://dx.doi.org/10.1093/humrep/dei457
47. Bahamondes L, Monteiro-Dantas C, Espejo-Arce X, et al. A prospective study of the forearm bone density of users of etonorgestrel- and levonorgestrel-releasing contraceptive implants. Hum Reprod 2006;21:466–70. http://dx.doi.org/10.1093/humrep/dei358

48. Pitts SA, Feldman HA, Dorale A, Gordon CM. Bone mineral density, fracture, and vitamin D in adolescents and young women using depot medroxyprogesterone acetate. J Pediatr Adolesc Gynecol 2012;25:23–6. http://dx.doi.org/10.1016/j.jpag.2011.07.014
49. US Department of Health and Human Services. Healthy people 2020: maternal, infant, and child health objectives. Washington, DC: US Department of Health and Human Services; 2015. http://www.healthypeople.gov/2020/topics-objectives/topic/maternal-infant-and-child-health/objectives
50. Braga GC, Ferriolli E, Quintana SM, Ferriani RA, Pfrimer K, Vieira CS. Immediate postpartum initiation of etonogestrel-releasing implant: a randomized controlled trial on breastfeeding impact. Contraception 2015;92:536–42. http://dx.doi.org/10.1016/j.contraception.2015.07.009
51. Phillips SJ, Tepper NK, Kapp N, Nanda K, Temmerman M, Curtis KM. Progestogen-only contraceptive use among breastfeeding women: a systematic review. Contraception 2015;S0010-7824(15)00585-5.
52. Kurunmäki H. Contraception with levonorgestrel-releasing subdermal capsules, Norplant, after pregnancy termination. Contraception 1983;27:473–82. http://dx.doi.org/10.1016/0010-7824(83)90044-6
53. Kurunmäki H, Toivonen J, Lähteenmäki PL, Luukkainen T. Immediate postabortal contraception with Norplant: levonorgestrel, gonadotropin, estradiol, and progesterone levels over two postabortal months and return of fertility after removal of Norplant capsules. Contraception 1984;30:431–42. http://dx.doi.org/10.1016/0010-7824(84)90035-0.
54. Lähteenmäki P, Toivonen J, Lähteenmäki PL. Postabortal contraception with norethisterone enanthate injections. Contraception 1983;27:553–62 http://dx.doi.org/10.1016/0010-7824(83)90020-3
55. Ortayli N, Bulut A, Sahin T, Sivin I. Immediate postabortal contraception with the levonorgestrel intrauterine device, Norplant, and traditional methods. Contraception 2001;63:309–14. http://dx.doi.org/10.1016/S0010-7824(01)00212-8
56. Beksinska ME, Smit JA, Kleinschmidt I, Milford C, Farley TM. Prospective study of weight change in new adolescent users of DMPA, NET-EN, COCs, nonusers and discontinuers of hormonal contraception. Contraception 2010;81:30–4. http://dx.doi.org/10.1016/j.contraception.2009.07.007
57. Bender NM, Segall-Gutierrez P, Najera SO, Stanczyk FZ, Montoro M, Mishell DR Jr. Effects of progestin-only long-acting contraception on metabolic markers in obese women. Contraception 2013;88:418–25. http://dx.doi.org/10.1016/j.contraception.2012.12.007
58. Berenson AB, Rahman M. Changes in weight, total fat, percent body fat, and central-to-peripheral fat ratio associated with injectable and oral contraceptive use. Am J Obstet Gynecol 2009;200:329.e1–8. http://dx.doi.org/10.1016/j.ajog.2008.12.052
59. Bonny AE, Secic M, Cromer B. Early weight gain related to later weight gain in adolescents on depot medroxyprogesterone acetate. Obstet Gynecol 2011;117:793–7. http://dx.doi.org/10.1097/AOG.0b013e31820f387c.
60. Bonny AE, Ziegler J, Harvey R, Debanne SM, Secic M, Cromer BA. Weight gain in obese and nonobese adolescent girls initiating depot medroxyprogesterone, oral contraceptive pills, or no hormonal contraceptive method. Arch Pediatr Adolesc Med 2006;160:40–5 http://dx.doi.org/10.1001/archpedi.160.1.40
61. Clark MK, Dillon JS, Sowers M, Nichols S. Weight, fat mass, and central distribution of fat increase when women use depot-medroxyprogesterone acetate for contraception. Int J Obes 2005;29:1252–8. http://dx.doi.org/10.1038/sj.ijo.0803023
62. Gerlach LS, Saldaña SN, Wang Y, Nick TG, Spigarelli MG. Retrospective review of the relationship between weight change and demographic factors following initial depot medroxyprogesterone acetate injection in adolescents. Clin Ther 2011;33:182–7. http://dx.doi.org/10.1016/j.clinthera.2011.02.008
63. Jain J, Jakimiuk AJ, Bode FR, Ross D, Kaunitz AM. Contraceptive efficacy and safety of DMPA-SC. Contraception 2004;70:269–75. http://dx.doi.org/10.1016/j.contraception.2004.06.011
64. Kozlowski KJ, Rickert VI, Hendon A, Davis P. Adolescents and Norplant: preliminary findings of side effects. J Adolesc Health 1995;16:373–8. http://dx.doi.org/10.1016/S1054-139X(94)00029-E
65. Le YC, Rahman M, Berenson AB. Early weight gain predicting later weight gain among depot medroxyprogesterone acetate users. Obstet Gynecol 2009;114:279–84. http://dx.doi.org/10.1097/AOG.0b013e3181af68b2
66. Leiman G. Depo-medroxyprogesterone acetate as a contraceptive agent: its effect on weight and blood pressure. Am J Obstet Gynecol 1972;114:97–102. http://dx.doi.org/10.1016/0002-9378(72)90296-7.
67. Lopez LM, Grimes DA, Chen M, et al. Hormonal contraceptives for contraception in overweight or obese women. Cochrane Database Syst Rev 2013;4:CD008452.
68. Mangan SA, Larsen PG, Hudson S. Overweight teens at increased risk for weight gain while using depot medroxyprogesterone acetate. J Pediatr Adolesc Gynecol 2002;15:79–82. http://dx.doi.org/10.1016/S1083-3188(01)00147-4
69. Nyirati CM, Habash DL, Shaffer LE. Weight and body fat changes in postpartum depot-medroxyprogesterone acetate users. Contraception 2013;88:169–76. http://dx.doi.org/10.1016/j.contraception.2012.10.016
70. Pantoja M, Medeiros T, Baccarin MC, Morais SS, Bahamondes L, Fernandes AM. Variations in body mass index of users of depot-medroxyprogesterone acetate as a contraceptive. Contraception 2010;81:107–11. http://dx.doi.org/10.1016/j.contraception.2009.07.008
71. Risser WL, Gefter LR, Barratt MS, Risser JM. Weight change in adolescents who used hormonal contraception. J Adolesc Health 1999;24:433–6. http://dx.doi.org/10.1016/S1054-139X(98)00151-7
72. Segall-Gutierrez P, Xiang AH, Watanabe RM, et al. Deterioration in cardiometabolic risk markers in obese women during depot medroxyprogesterone acetate use. Contraception 2012;85:36–41. http://dx.doi.org/10.1016/j.contraception.2011.04.016
73. Westhoff C, Jain JK, Milsom I, Ray A. Changes in weight with depot medroxyprogesterone acetate subcutaneous injection 104 mg/0.65 mL. Contraception 2007;75:261–7. http://dx.doi.org/10.1016/j.contraception.2006.12.009
74. Paulen ME, Zapata LB, Cansino C, Curtis KM, Jamieson DJ. Contraceptive use among women with a history of bariatric surgery: a systematic review. Contraception 2010;82:86–94. http://dx.doi.org/10.1016/j.contraception.2010.02.008
75. World Health Organization Collaborative Study of Cardiovascular Disease and Steroid Hormone Contraception. Cardiovascular disease and use of oral and injectable progestogen-only contraceptives and combined injectable contraceptives. Results of an international, multicenter, case-control study. Contraception 1998;57:315–24 http://dx.doi.org/10.1016/S0010-7824(98)00041-9
76. Heinemann LA, Assmann A, DoMinh T, Garbe E. Oral progestogen-only contraceptives and cardiovascular risk: results from the Transnational Study on Oral Contraceptives and the Health of Young Women. Eur J Contracept Reprod Health Care 1999;4:67–73. http://dx.doi.org/10.3109/13625189909064007
77. Vasilakis C, Jick H, del Mar Melero-Montes M. Risk of idiopathic venous thromboembolism in users of progestagens alone. Lancet 1999;354:1610–1. http://dx.doi.org/10.1016/S0140-6736(99)04394-9
78. Sönmezer M, Atabekoğlu C, Cengiz B, Dökmeci F, Cengiz SD. Depot-medroxyprogesterone acetate in anticoagulated patients with previous hemorrhagic corpus luteum. Eur J Contracept Reprod Health Care 2005;10:9–14. http://dx.doi.org/10.1080/13625180400020952
79. Tepper NK, Paulen ME, Marchbanks PA, Curtis KM. Safety of contraceptive use among women with peripartum cardiomyopathy: a systematic review. Contraception 2010;82:95–101. http://dx.doi.org/10.1016/j.contraception.2010.02.004
80. The Criteria Committee of the New York Heart Association. Nomenclature and criteria for diagnosis of diseases of the heart and great vessels. 9th ed. Boston, MA: Little, Brown and Co; 1994.
81. Bernatsky S, Clarke A, Ramsey-Goldman R, et al. Hormonal exposures and breast cancer in a sample of women with systemic lupus erythematosus. Rheumatology (Oxford) 2004;43:1178–81. http://dx.doi.org/10.1093/rheumatology/keh282

82. Bernatsky S, Ramsey-Goldman R, Gordon C, et al. Factors associated with abnormal Pap results in systemic lupus erythematosus. Rheumatology (Oxford) 2004;43:1386–9. http://dx.doi.org/10.1093/rheumatology/keh331
83. Chopra N, Koren S, Greer WL, et al. Factor V Leiden, prothrombin gene mutation, and thrombosis risk in patients with antiphospholipid antibodies. J Rheumatol 2002;29:1683–8.
84. Esdaile JM, Abrahamowicz M, Grodzicky T, et al. Traditional Framingham risk factors fail to fully account for accelerated atherosclerosis in systemic lupus erythematosus. Arthritis Rheum 2001;44:2331–7. http://dx.doi.org/10.1002/1529-0131(200110)44:10<2331::AID-ART395≥3.0.CO;2-I
85. Julkunen HA. Oral contraceptives in systemic lupus erythematosus: side-effects and influence on the activity of SLE. Scand J Rheumatol 1991;20:427–33. http://dx.doi.org/10.3109/03009749109096822
86. Julkunen HA, Kaaja R, Friman C. Contraceptive practice in women with systemic lupus erythematosus. Br J Rheumatol 1993;32:227–30. http://dx.doi.org/10.1093/rheumatology/32.3.227
87. Jungers P, Dougados M, Pélissier C, et al. Influence of oral contraceptive therapy on the activity of systemic lupus erythematosus. Arthritis Rheum 1982;25:618–23. http://dx.doi.org/10.1002/art.1780250603
88. Manzi S, Meilahn EN, Rairie JE, et al. Age-specific incidence rates of myocardial infarction and angina in women with systemic lupus erythematosus: comparison with the Framingham Study. Am J Epidemiol 1997;145:408–15. http://dx.doi.org/10.1093/oxfordjournals.aje.a009122
89. McAlindon T, Giannotta L, Taub N, D'Cruz D, Hughes G. Environmental factors predicting nephritis in systemic lupus erythematosus. Ann Rheum Dis 1993;52:720–4. http://dx.doi.org/10.1136/ard.52.10.720
90. McDonald J, Stewart J, Urowitz MB, Gladman DD. Peripheral vascular disease in patients with systemic lupus erythematosus. Ann Rheum Dis 1992;51:56–60. http://dx.doi.org/10.1136/ard.51.1.56
91. Mintz G, Gutiérrez G, Delezé M, Rodríguez E. Contraception with progestagens in systemic lupus erythematosus. Contraception 1984;30:29–38. http://dx.doi.org/10.1016/0010-7824(84)90076-3
92. Petri M. Musculoskeletal complications of systemic lupus erythematosus in the Hopkins Lupus Cohort: an update. Arthritis Care Res 1995;8:137–45. http://dx.doi.org/10.1002/art.1790080305
93. Petri M. Lupus in Baltimore: evidence-based 'clinical pearls' from the Hopkins Lupus Cohort. Lupus 2005;14:970–3. http://dx.doi.org/10.1191/0961203305lu2230xx
94. Petri M, Kim MY, Kalunian KC, et al; OC-SELENA Trial. Combined oral contraceptives in women with systemic lupus erythematosus. N Engl J Med 2005;353:2550–8. http://dx.doi.org/10.1056/NEJMoa051135
95. Sánchez-Guerrero J, Uribe AG, Jiménez-Santana L, et al. A trial of contraceptive methods in women with systemic lupus erythematosus. N Engl J Med 2005;353:2539–49. http://dx.doi.org/10.1056/NEJMoa050817
96. Sarabi ZS, Chang E, Bobba R, et al. Incidence rates of arterial and venous thrombosis after diagnosis of systemic lupus erythematosus. Arthritis Rheum 2005;53:609–12. http://dx.doi.org/10.1002/art.21314
97. Schaedel ZE, Dolan G, Powell MC. The use of the levonorgestrel-releasing intrauterine system in the management of menorrhagia in women with hemostatic disorders. Am J Obstet Gynecol 2005;193:1361–3. http://dx.doi.org/10.1016/j.ajog.2005.05.002
98. Somers E, Magder LS, Petri M. Antiphospholipid antibodies and incidence of venous thrombosis in a cohort of patients with systemic lupus erythematosus. J Rheumatol 2002;29:2531–6.
99. Urowitz MB, Bookman AA, Koehler BE, Gordon DA, Smythe HA, Ogryzlo MA. The bimodal mortality pattern of systemic lupus erythematosus. Am J Med 1976;60:221–5. http://dx.doi.org/10.1016/0002-9343(76)90431-9
100. Choojitarom K, Verasertniyom O, Totemchokchyakarn K, Nantiruj K, Sumethkul V, Janwityanujit S. Lupus nephritis and Raynaud's phenomenon are significant risk factors for vascular thrombosis in SLE patients with positive antiphospholipid antibodies. Clin Rheumatol 2008;27:345–51. http://dx.doi.org/10.1007/s10067-007-0721-z
101. Wahl DG, Guillemin F, de Maistre E, Perret C, Lecompte T, Thibaut G. Risk for venous thrombosis related to antiphospholipid antibodies in systemic lupus erythematosus—a meta-analysis. Lupus 1997;6:467–73. http://dx.doi.org/10.1177/096120339700600510
102. Farr SL, Folger SG, Paulen ME, Curtis KM. Safety of contraceptive methods for women with rheumatoid arthritis: a systematic review. Contraception 2010;82:64–71. http://dx.doi.org/10.1016/j.contraception.2010.02.003
103. Tepper NK, Whiteman MK, Zapata LB, Marchbanks PA, Curtis KM. Safety of hormonal contraceptives among women with migraine: a systematic review. Contraception 2016. Epub May 3, 2016. http://dx.doi.org/10.1016/j.contraception.2016.04.016
104. Tepper NK, Whiteman MK, Marchbanks PA, James AH, Curtis KM. Progestin-only contraception and thromboembolism: a systematic review. Contraception 2016. Epub May 3, 2016. http://dx.doi.org/10.1016/j.contraception.2016.04.014
105. Zapata LB, Oduyebo T, Whiteman MK, Marchbanks PA, Curtis KM. Contraceptive use among women with multiple sclerosis: a systematic review. Contraception. In press 2016.
106. Pagano HP, Zapata LB, Berry-Bibee EN, Nanda K, Curtis KM. Safety of hormonal contraception and intrauterine devices among women with depressive and bipolar disorders: a systematic review. Contraception 2016. Epub June 27, 2016. http://dx.doi.org/10.1016/j.contraception.2016.06.012
107. Smith JS, Green J, Berrington de Gonzalez A, et al. Cervical cancer and use of hormonal contraceptives: a systematic review. Lancet 2003;361:1159–67. http://dx.doi.org/10.1016/S0140-6736(03)12949-2.
108. Polis CB, Phillips SJ, Curtis KM, et al. Hormonal contraceptive methods and risk of HIV acquisition in women: a systematic review of epidemiological evidence. Contraception 2014;90:360–90 http://dx.doi.org/10.1016/j.contraception.2014.07.009
109. Phillips SJ, Curtis KM, Polis CB. Effect of hormonal contraceptive methods on HIV disease progression: a systematic review. AIDS 2013;27:787–94. http://dx.doi.org/10.1097/QAD.0b013e32835bb672
110. Polis CB, Phillips SJ, Curtis KM. Hormonal contraceptive use and female-to-male HIV transmission: a systematic review of the epidemiologic evidence. AIDS 2013;27:493–505. http://dx.doi.org/10.1097/QAD.0b013e32835ad539
111. Phillips SJ, Polis CB, Curtis KM. The safety of hormonal contraceptives for women living with HIV and their sexual partners. Contraception 2016;93:11–6 http://dx.doi.org/10.1016/j.contraception.2015.10.002.
112. Tagy AH, Saker ME, Moussa AA, Kolgah A. The effect of low-dose combined oral contraceptive pills versus injectable contraceptive (Depot Provera) on liver function tests of women with compensated bilharzial liver fibrosis. Contraception 2001;64:173–6. http://dx.doi.org/10.1016/S0010-7824(01)00248-7
113. Pyörälä T, Vähäpassi J, Huhtala M. The effect of lynestrenol and norethindrone on the carbohydrate and lipid metabolism in subjects with gestational diabetes. Ann Chir Gynaecol 1979;68:69–74.
114. Rådberg T, Gustafson A, Skryten A, Karlsson K. Metabolic studies in gestational diabetic women during contraceptive treatment: effects on glucose tolerance and fatty acid composition of serum lipids. Gynecol Obstet Invest 1982;13:17–29. http://dx.doi.org/10.1159/000299480

115. Kjos SL, Peters RK, Xiang A, Thomas D, Schaefer U, Buchanan TA. Contraception and the risk of type 2 diabetes mellitus in Latina women with prior gestational diabetes mellitus. JAMA 1998;280:533–8. http://dx.doi.org/10.1001/jama.280.6.533
116. Nelson AL, Le MH, Musherraf Z, Vanberckelaer A. Intermediate-term glucose tolerance in women with a history of gestational diabetes: natural history and potential associations with breastfeeding and contraception. Am J Obstet Gynecol 2008;198:699.e1–8.
117. Xiang AH, Kawakubo M, Buchanan TA, Kjos SL. A longitudinal study of lipids and blood pressure in relation to method of contraception in Latino women with prior gestational diabetes mellitus. Diabetes Care 2007;30:1952–8. http://dx.doi.org/10.2337/dc07-0180
118. Xiang AH, Kawakubo M, Kjos SL, Buchanan TA. Long-acting injectable progestin contraception and risk of type 2 diabetes in Latino women with prior gestational diabetes mellitus. Diabetes Care 2006;29:613–7. http://dx.doi.org/10.2337/diacare.29.03.06.dc05-1940
119. Diab KM, Zaki MM. Contraception in diabetic women: comparative metabolic study of Norplant, depot medroxyprogesterone acetate, low dose oral contraceptive pill and CuT380A. J Obstet Gynaecol Res 2000;26:17–26. http://dx.doi.org/10.1111/j.1447-0756.2000.tb01195.x
120. Lunt H, Brown LJ. Self-reported changes in capillary glucose and insulin requirements during the menstrual cycle. Diabet Med 1996;13:525–30. http://dx.doi.org/10.1002/(SICI)1096-9136(199606)13:6<525::AID-DIA123≥3.0.CO;2-D
121. Rådberg T, Gustafson A, Skryten A, Karlsson K. Oral contraception in diabetic women. A cross-over study on serum and high density lipoprotein (HDL) lipids and diabetes control during progestogen and combined estrogen/progestogen contraception. Horm Metab Res 1982;14:61–5.
122. Skouby SO, Mølsted-Pedersen L, Kühl C, Bennet P. Oral contraceptives in diabetic women: metabolic effects of four compounds with different estrogen/progestogen profiles. Fertil Steril 1986;46:858–64 http://dx.doi.org/10.1016/S0015-0282(16)49825-0.
123. Zapata LB, Paulen ME, Cansino C, Marchbanks PA, Curtis KM. Contraceptive use among women with inflammatory bowel disease: a systematic review. Contraception 2010;82:72–85. http://dx.doi.org/10.1016/j.contraception.2010.02.012
124. Kapp N, Curtis KM. Hormonal contraceptive use among women with liver tumors: a systematic review. Contraception 2009;80:387–90. http://dx.doi.org/10.1016/j.contraception.2009.01.021
125. Whiteman MK, Oduyebo T, Zapata LB, Walker S, Curtis KM. Contraceptive safety among women with cystic fibrosis: a systematic review. Contraception 2016. Epub June 7, 2016. http://dx.doi.org/10.1016/j.contraception.2016.05.016
126. Adadevoh BK, Isaacs WA. The effect of megestrol acetate on sickling. Am J Med Sci 1973;265:367–70. http://dx.doi.org/10.1097/00000441-197305000-00002
127. Barbosa IC, Ladipo OA, Nascimento ML, et al. Carbohydrate metabolism in sickle cell patients using a subdermal implant containing nomegestrol acetate (Uniplant). Contraception 2001;63:263–5. http://dx.doi.org/10.1016/S0010-7824(01)00202-5
128. de Abood M, de Castillo Z, Guerrero F, Espino M, Austin KL. Effect of Depo-Provera or Microgynon on the painful crises of sickle cell anemia patients. Contraception 1997;56:313–6. http://dx.doi.org/10.1016/S0010-7824(97)00156-X
129. De Ceulaer K, Hayes R, Gruber C, Serjeant GR. Medroxyprogesterone acetate and homozygous sickle-cell disease. Lancet 1982;320:229–31. http://dx.doi.org/10.1016/S0140-6736(82)90320-8
130. Howard RJ, Lillis C, Tuck SM. Contraceptives, counselling, and pregnancy in women with sickle cell disease. BMJ 1993;306:1735–7. http://dx.doi.org/10.1136/bmj.306.6894.1735
131. Ladipo OA, Falusi AG, Feldblum PJ, Osotimehin BO, Otolorin EO, Ojengbede OA. Norplant use by women with sickle cell disease. Int J Gynaecol Obstet 1993;41:85–7. http://dx.doi.org/10.1016/0020-7292(93)90159-T
132. Nascimento ML, Ladipo OA, Coutinho EM. Nomegestrol acetate contraceptive implant use by women with sickle cell disease. Clin Pharmacol Ther 1998;64:433–8. http://dx.doi.org/10.1016/S0009-9236(98)90074-1
133. Yoong WC, Tuck SM, Yardumian A. Red cell deformability in oral contraceptive pill users with sickle cell anaemia. Br J Haematol 1999;104:868–70. http://dx.doi.org/10.1046/j.1365-2141.1999.01255.x
134. Aweeka FT, Rosenkranz SL, Segal Y, et al; NIAID AIDS Clinical Trials Group. The impact of sex and contraceptive therapy on the plasma and intracellular pharmacokinetics of zidovudine. AIDS 2006;20:1833–41. http://dx.doi.org/10.1097/01.aids.0000244202.18629.36
135. Kearney BP, Mathias A. Lack of effect of tenofovir disoproxil fumarate on pharmacokinetics of hormonal contraceptives. Pharmacotherapy 2009;29:924–9. http://dx.doi.org/10.1592/phco.29.8.924
136. Todd CS, Deese J, Wang M, et al; FEM-PrEP Study Group. Sino-implant (II)® continuation and effect of concomitant tenofovir disoproxil fumarate-emtricitabine use on plasma levonorgestrel concentrations among women in Bondo, Kenya. Contraception 2015;91:248–52. http://dx.doi.org/10.1016/j.contraception.2014.10.008
137. Murnane PM, Heffron R, Ronald A, et al; Partners PrEP Study Team. Pre-exposure prophylaxis for HIV-1 prevention does not diminish the pregnancy prevention effectiveness of hormonal contraception. AIDS 2014;28:1825–30. http://dx.doi.org/10.1097/QAD.0000000000000290
138. Kasonde M, Niska RW, Rose C, et al. Bone mineral density changes among HIV-uninfected young adults in a randomised trial of pre-exposure prophylaxis with tenofovir-emtricitabine or placebo in Botswana. PLoS One 2014;9:e90111. http://dx.doi.org/10.1371/journal.pone.0090111
139. Callahan R, Nanda K, Kapiga S, et al; FEM-PrEP Study Group. Pregnancy and contraceptive use among women participating in the FEM-PrEP trial. J Acquir Immune Defic Syndr 2015;68:196–203. http://dx.doi.org/10.1097/QAI.0000000000000413
140. Vieira CS, Bahamondes MV, de Souza RM, et al. Effect of antiretroviral therapy including lopinavir/ritonavir or efavirenz on etonogestrel-releasing implant pharmacokinetics in HIV-positive women. J Acquir Immune Defic Syndr 2014;66:378–85. http://dx.doi.org/10.1097/QAI.0000000000000189
141. Patel RC, Onono M, Gandhi M, et al. Pregnancy rates in HIV-positive women using contraceptives and efavirenz-based or nevirapine-based antiretroviral therapy in Kenya: a retrospective cohort study. Lancet HIV 2015;2:e474–82. http://dx.doi.org/10.1016/S2352-3018(15)00184-8
142. Perry SH, Swamy P, Preidis GA, Mwanyumba A, Motsa N, Sarero HN. Implementing the Jadelle implant for women living with HIV in a resource-limited setting: concerns for drug interactions leading to unintended pregnancies. AIDS 2014;28:791–3. http://dx.doi.org/10.1097/QAD.0000000000000177
143. Scarsi KK, Darin KM, Nakalema S, et al. Unintended pregnancies observed with combined use of the levonorgestrel contraceptive implant and efavirenz-based antiretroviral therapy: a three-arm pharmacokinetic evaluation over 48 weeks. Clin Infect Dis 2015;62:675–82. http://dx.doi.org/10.1093/cid/civ1001

144. Pyra M, Heffron R, Mugo NR, et al; Partners in Prevention HSVHIV Transmission Study and Partners PrEP Study Teams. Effectiveness of hormonal contraception in HIV-infected women using antiretroviral therapy. AIDS 2015;29:2353–9. http://dx.doi.org/10.1097/QAD.0000000000000827
145. Cohn SE, Park JG, Watts DH, et al; ACTG A5093 Protocol Team. Depo-medroxyprogesterone in women on antiretroviral therapy: effective contraception and lack of clinically significant interactions. Clin Pharmacol Ther 2007;81:222–7. http://dx.doi.org/10.1038/sj.clpt.6100040
146. Nanda K, Amaral E, Hays M, Viscola MA, Mehta N, Bahamondes L. Pharmacokinetic interactions between depot medroxyprogesterone acetate and combination antiretroviral therapy. Fertil Steril 2008;90:965–71. http://dx.doi.org/10.1016/j.fertnstert.2007.07.1348
147. Watts DH, Park JG, Cohn SE, et al. Safety and tolerability of depot medroxyprogesterone acetate among HIV-infected women on antiretroviral therapy: ACTG A5093. Contraception 2008;77:84–90. http://dx.doi.org/10.1016/j.contraception.2007.10.002
148. Polis CB, Nakigozi G, Ssempijja V, et al. Effect of injectable contraceptive use on response to antiretroviral therapy among women in Rakai, Uganda. Contraception 2012;86:725–30. http://dx.doi.org/10.1016/j.contraception.2012.05.001
149. Hubacher D, Liku J, Kiarie J, et al. Effect of concurrent use of antiretroviral therapy and levonorgestrel sub-dermal implant for contraception on CD4 counts: a prospective cohort study in Kenya. J Int AIDS Soc 2013;16:18448. http://dx.doi.org/10.7448/IAS.16.1.18448
150. Myer L, Carter RJ, Katyal M, Toro P, El-Sadr WM, Abrams EJ. Impact of antiretroviral therapy on incidence of pregnancy among HIV-infected women in Sub-Saharan Africa: a cohort study. PLoS Med 2010;7:e1000229. http://dx.doi.org/10.1371/journal.pmed.1000229
151. Day S, Graham SM, Masese LN, et al. A prospective cohort study of the effect of depot medroxyprogesterone acetate on detection of plasma and cervical HIV-1 in women initiating and continuing antiretroviral therapy. J Acquir Immune Defic Syndr 2014;66:452–6. http://dx.doi.org/10.1097/QAI.0000000000000187
152. DuBois BN, Atrio J, Stanczyk FZ, Cherala G. Increased exposure of norethindrone in HIV+ women treated with ritonavir-boosted atazanavir therapy. Contraception 2015;91:71–5. http://dx.doi.org/10.1016/j.contraception.2014.08.009
153. Luque AE, Cohn SE, Park JG, et al. Depot medroxyprogesterone acetate in combination with a twice-daily lopinavir-ritonavir-based regimen in HIV-infected women showed effective contraception and a lack of clinically significant interactions, with good safety and tolerability: results of the ACTG 5283 study. Antimicrob Agents Chemother 2015;59:2094–101. http://dx.doi.org/10.1128/AAC.04701-14
154. Odlind V, Olsson SE. Enhanced metabolism of levonorgestrel during phenytoin treatment in a woman with Norplant implants. Contraception 1986;33:257–61. http://dx.doi.org/10.1016/0010-7824(86)90018-1
155. Schindlbeck C, Janni W, Friese K. Failure of Implanon contraception in a patient taking carbamazepin for epilepsia. Arch Gynecol Obstet 2006;273:255–6. http://dx.doi.org/10.1007/s00404-005-0064-4
156. Shane-McWhorter L, Cerveny JD, MacFarlane LL, Osborn C. Enhanced metabolism of levonorgestrel during phenobarbital treatment and resultant pregnancy. Pharmacotherapy 1998;18:1360–4.
157. Reimers A, Helde G, Brodtkorb E. Ethinyl estradiol, not progestogens, reduces lamotrigine serum concentrations. Epilepsia 2005;46:1414–7. http://dx.doi.org/10.1111/j.1528-1167.2005.10105.x
158. Berry-Bibee E, Kim MJ, Simmons K, Pagano P, Curtis K. Drug interactions between hormonal contraceptives and psychotropic drugs: a systematic review. Contraception. In press 2016.
159. Berry-Bibee E, Kim MJ, Tepper N, Riley H, Curtis K. The safety of St. John's wort and hormonal contraceptives: a systematic review. Contraception. In press 2016.

Appendix D

Classifications for Combined Hormonal Contraceptives

Combined hormonal contraceptives (CHCs) include low-dose (containing ≤35 μg ethinyl estradiol) combined oral contraceptives (COCs), the combined hormonal patch, and the combined vaginal ring (Box D1) (Table D1). Limited information is available about the safety of the combined hormonal patch and combined vaginal ring among women with specific medical conditions. Evidence indicates that the combined hormonal patch and the combined vaginal ring provide comparable safety and pharmacokinetic profiles to COCs with similar hormone formulations *(1–33)*. Pending further studies, the evidence available for recommendations about COCs applies to the recommendations for the combined hormonal patch and vaginal ring. Therefore, the patch and ring should have the same categories as COCs, except where noted. Therefore, the assigned categories should be considered a preliminary best judgement, which will be reevaluated as new data become available.

BOX D1. Categories for classifying combined hormonal contraceptives

1 = A condition for which there is no restriction for the use of the contraceptive method.
2 = A condition for which the advantages of using the method generally outweigh the theoretical or proven risks.
3 = A condition for which the theoretical or proven risks usually outweigh the advantages of using the method.
4 = A condition that represents an unacceptable health risk if the contraceptive method is used.

COCs, the patch, and the ring do not protect against sexually transmitted diseases (STDs), including human immunodeficiency virus (HIV), and women using these methods should be counseled that consistent and correct use of the male latex condom reduces the risk for transmission of HIV and other STDs. Use of female condoms can provide protection from transmission of STDs, although data are limited

TABLE D1. Classifications for combined hormonal contraceptives, including pill, patch, and ring

Condition	Category CHCs	Clarifications/Evidence/Comments
Personal Characteristics and Reproductive History		
Pregnancy	NA	**Clarification:** Use of CHCs is not required. No known harm to the woman, the course of her pregnancy, or the fetus occurs if CHCs are inadvertently used during pregnancy.
Age		
a. Menarche to <40 years	1	**Evidence:** Evidence is inconsistent about whether CHC use affects fracture risk (*34*–*45*), although three recent studies show no effect (*34*,*35*,*45*). CHC use might decrease BMD in adolescents, especially in those choosing very low-dose formulations (COCs containing <30 μg ethinyl estradiol) (*46*–*59*). CHC use has little to no effect on BMD in premenopausal women (*60*–*74*) and might preserve bone mass in those who are perimenopausal (*75*–*83*). BMD is a surrogate marker for fracture risk that might not be valid for premenopausal women and therefore might not accurately predict current or future (postmenopausal) fracture risk (*84*–*86*).
b. ≥40 years	2	**Comment:** The risk for cardiovascular disease increases with age and might increase with CHC use. In the absence of other adverse clinical conditions, CHCs can be used until menopause.
Parity		
a. Nulliparous	1	—
b. Parous	1	—
Breastfeeding		
a. <21 days postpartum	4	**Clarification:** Breastfeeding provides important health benefits for mother and infant. The U.S. Department of Health and Human Services recommends increasing the proportion of infants initially breastfed, exclusively breastfed through 6 months of life, and continuing breastfeeding through at least 1 year of life as key public health goals (*87*). **Evidence:** Clinical studies demonstrate conflicting results regarding effects on breastfeeding continuation or exclusivity in women exposed to COCs during lactation. No consistent effects on infant growth or illness have been reported. Adverse health outcomes or manifestations of exogenous estrogen in infants exposed to CHCs through breast milk have not been demonstrated; however, studies have been inadequately designed to determine whether a risk for either serious or subtle long-term effects exists (*88*). **Evidence:** One study examined use of CHCs during the postpartum period and found that VTE rates were higher for CHC users compared with nonusers at all time points postpartum (*89*). Rates were significantly different only after 13 weeks postpartum; however, the numbers needed to harm were lowest in the first 6 weeks postpartum. VTE risk is increased during pregnancy and the postpartum period; this risk is most pronounced in the first 3 weeks after delivery, decreasing to near baseline levels by 42 days postpartum (*90*–*94*). **Comment:** Certain women might be at risk for breastfeeding difficulties, such as women with previous breastfeeding difficulties, certain medical conditions, or certain perinatal complications, and those who deliver preterm. For these women, as for all women, discussions about contraception for breastfeeding women should include information about risks, benefits, and alternatives.
b. 21 to <30 days postpartum		
i. With other risk factors for VTE (e.g., age ≥35 years, previous VTE, thrombophilia, immobility, transfusion at delivery, peripartum cardiomyopathy, BMI ≥30 kg/m^2, postpartum hemorrhage, postcesarean delivery, preeclampsia, or smoking)	3	**Clarification:** Breastfeeding provides important health benefits for mother and infant. The U.S. Department of Health and Human Services recommends increasing the proportion of infants initially breastfed, exclusively breastfed through 6 months of life, and continuing breastfeeding through at least 1 year of life as key public health goals (*87*). **Clarification:** For women with other risk factors for VTE, these risk factors might increase the classification to a category 4. **Evidence:** Clinical studies demonstrate conflicting results regarding effects on breastfeeding continuation or exclusivity in women exposed to COCs during lactation. No consistent effects on infant growth or illness have been reported. Adverse health outcomes or manifestations of exogenous estrogen in infants exposed to CHCs through breast milk have not been demonstrated; however, studies have been inadequately designed to determine whether a risk for either serious or subtle long-term effects exists (*88*). **Evidence:** One study examined use of CHCs during the postpartum period and found that VTE rates were higher for CHC users compared with nonusers at all time points postpartum (*89*). Rates were significantly different only after 13 weeks postpartum; however, the numbers needed to harm were lowest in the first 6 weeks postpartum. VTE risk is increased during pregnancy and the postpartum period; this risk is most pronounced in the first 3 weeks after delivery, decreasing to near baseline levels by 42 days postpartum (*90*–*94*). **Comment:** Certain women might be at risk for breastfeeding difficulties, such as women with previous breastfeeding difficulties, certain medical conditions, or certain perinatal complications, and those who deliver preterm. For these women, as for all women, discussions about contraception for breastfeeding women should include information about risks, benefits, and alternatives.

See table footnotes on page 69.

TABLE D1. (*Continued*) Classifications for combined hormonal contraceptives, including pill, patch, and ring

Condition	Category CHCs	Clarifications/Evidence/Comments
ii. Without other risk factors for VTE	3	**Clarification:** Breastfeeding provides important health benefits for mother and infant. The U.S. Department of Health and Human Services recommends increasing the proportion of infants initially breastfed, exclusively breastfed through 6 months of life, and continuing breastfeeding through at least 1 year of life as key public health goals (*87*). **Evidence:** Clinical studies demonstrate conflicting results regarding effects on breastfeeding continuation or exclusivity in women exposed to COCs during lactation. No consistent effects on infant growth or illness have been reported. Adverse health outcomes or manifestations of exogenous estrogen in infants exposed to CHCs through breast milk have not been demonstrated; however, studies have been inadequately designed to determine whether a risk for either serious or subtle long-term effects exists (*88*). **Evidence:** One study examined use of CHCs during the postpartum period and found that VTE rates were higher for CHC users compared with nonusers at all time points postpartum (*89*). Rates were significantly different only after 13 weeks postpartum; however, the numbers needed to harm were lowest in the first 6 weeks postpartum. VTE risk is increased during pregnancy and the postpartum period; this risk is most pronounced in the first 3 weeks after delivery, decreasing to near baseline levels by 42 days postpartum (*90–94*). **Comment:** Certain women might be at risk for breastfeeding difficulties, such as women with previous breastfeeding difficulties, certain medical conditions, or certain perinatal complications, and those who deliver preterm. For these women, as for all women, discussions about contraception for breastfeeding women should include information about risks, benefits, and alternatives.
c. 30–42 days postpartum		
i. With other risk factors for VTE (e.g., age ≥35 years, previous VTE, thrombophilia, immobility, transfusion at delivery, peripartum cardiomyopathy, BMI ≥30 kg/m^2, postpartum hemorrhage, postcesarean delivery, preeclampsia, or smoking)	3	**Clarification:** Breastfeeding provides important health benefits for mother and infant. The U.S. Department of Health and Human Services recommends increasing the proportion of infants initially breastfed, exclusively breastfed through 6 months of life, and continuing breastfeeding through at least 1 year of life as key public health goals (*87*). **Clarification:** For women with other risk factors for VTE, these risk factors might increase the classification to a category 4. **Evidence:** Clinical studies demonstrate conflicting results regarding effects on breastfeeding continuation or exclusivity in women exposed to COCs during lactation. No consistent effects on infant growth or illness have been reported. Adverse health outcomes or manifestations of exogenous estrogen in infants exposed to CHCs through breast milk have not been demonstrated; however, studies have been inadequately designed to determine whether a risk for either serious or subtle long-term effects exists (*88*). **Evidence:** One study examined use of CHCs during the postpartum period and found that VTE rates were higher for CHC users compared with nonusers at all time points postpartum (*89*). Rates were significantly different only after 13 weeks postpartum; however, the numbers needed to harm were lowest in the first 6 weeks postpartum. VTE risk is increased during pregnancy and the postpartum period; this risk is most pronounced in the first 3 weeks after delivery, decreasing to near baseline levels by 42 days postpartum (*90–94*). **Comment:** Certain women might be at risk for breastfeeding difficulties, such as women with previous breastfeeding difficulties, certain medical conditions, or certain perinatal complications, and those who deliver preterm. For these women, as for all women, discussions about contraception for breastfeeding women should include information about risks, benefits, and alternatives.

See table footnotes on page 69.

TABLE D1. (*Continued*) Classifications for combined hormonal contraceptives, including pill, patch, and ring

Condition	Category CHCs	Clarifications/Evidence/Comments
ii. Without other risk factors for VTE	2	**Clarification:** Breastfeeding provides important health benefits for mother and infant. The U.S. Department of Health and Human Services recommends increasing the proportion of infants initially breastfed, exclusively breastfed through 6 months of life, and continuing breastfeeding through at least 1 year of life as key public health goals (*87*). **Evidence:** Clinical studies demonstrate conflicting results regarding effects on breastfeeding continuation or exclusivity in women exposed to COCs during lactation. No consistent effects on infant growth or illness have been reported. Adverse health outcomes or manifestations of exogenous estrogen in infants exposed to CHCs through breast milk have not been demonstrated; however, studies have been inadequately designed to determine whether a risk for either serious or subtle long-term effects exists (*88*). **Evidence:** One study examined use of CHCs during the postpartum period and found that VTE rates were higher for CHC users compared with nonusers at all time points postpartum (*89*). Rates were significantly different only after 13 weeks postpartum; however, the numbers needed to harm were lowest in the first 6 weeks postpartum. VTE risk is increased during pregnancy and the postpartum period; this risk is most pronounced in the first 3 weeks after delivery, decreasing to near baseline levels by 42 days postpartum (*90–94*). **Comment:** Certain women might be at risk for breastfeeding difficulties, such as women with previous breastfeeding difficulties, certain medical conditions, or certain perinatal complications, and those who deliver preterm. For these women, as for all women, discussions about contraception for breastfeeding women should include information about risks, benefits, and alternatives.
d. >42 days postpartum	2	**Clarification:** Breastfeeding provides important health benefits for mother and infant. The U.S. Department of Health and Human Services recommends increasing the proportion of infants initially breastfed, exclusively breastfed through 6 months of life, and continuing breastfeeding through at least 1 year of life as key public health goals (*87*). **Evidence:** Clinical studies demonstrate conflicting results regarding effects on breastfeeding continuation or exclusivity in women exposed to COCs during lactation. No consistent effects on infant growth or illness have been reported. Adverse health outcomes or manifestations of exogenous estrogen in infants exposed to CHCs through breast milk have not been demonstrated; however, studies have been inadequately designed to determine whether a risk for either serious or subtle long-term effects exists (*88*). **Comment:** Certain women might be at risk for breastfeeding difficulties, such as women with previous breastfeeding difficulties, certain medical conditions, or certain perinatal complications, and those who deliver preterm. For these women, as for all women, discussions about contraception for breastfeeding women should include information about risks, benefits, and alternatives.
Postpartum (nonbreastfeeding women)		
a. <21 days postpartum	4	**Evidence:** One study examined use of CHCs during the postpartum period and found that VTE rates were higher for CHC users compared with nonusers at all time points postpartum (*89*). Rates were significantly different only after 13 weeks postpartum; however, the numbers needed to harm were lowest in the first 6 weeks postpartum. VTE risk is increased during pregnancy and the postpartum period; this risk is most pronounced in the first 3 weeks after delivery, decreasing to near baseline levels by 42 days postpartum (*90–94*). Risk for pregnancy during the first 21 days postpartum is very low but increases after that point; ovulation before first menses is common (*95*).
b. 21–42 days postpartum		
i. With other risk factors for VTE (e.g., age ≥35 years, previous VTE, thrombophilia, immobility, transfusion at delivery, peripartum cardiomyopathy, BMI ≥30 kg/m^2 postpartum hemorrhage, postcesarean delivery, preeclampsia, or smoking)	3	**Clarification:** For women with other risk factors for VTE, these risk factors might increase the classification to a category 4. **Evidence:** One study examined use of CHCs during the postpartum period and found that VTE rates were higher for CHC users compared with nonusers at all time points postpartum (*89*). Rates were significantly different only after 13 weeks postpartum; however, the numbers needed to harm were lowest in the first 6 weeks postpartum. VTE risk is increased during pregnancy and the postpartum period; this risk is most pronounced in the first 3 weeks after delivery, decreasing to near baseline levels by 42 days postpartum (*90–94*).
ii. Without other risk factors for VTE	2	**Evidence:** One study examined use of CHCs during the postpartum period and found that VTE rates were higher for CHC users compared with nonusers at all time points postpartum (*89*). Rates were significantly different only after 13 weeks postpartum; however, the numbers needed to harm were lowest in the first 6 weeks postpartum. VTE risk is increased during pregnancy and the postpartum period; this risk is most pronounced in the first 3 weeks after delivery, decreasing to near baseline levels by 42 days postpartum (*90–94*).
c. >42 days postpartum	1	—

See table footnotes on page 69.

TABLE D1. (*Continued*) Classifications for combined hormonal contraceptives, including pill, patch, and ring

Condition	Category CHCs	Clarifications/Evidence/Comments
Postabortion		**Clarification:** CHCs may be started immediately postabortion.
a. First trimester	1	**Evidence:** Women who started taking COCs immediately after first trimester medical or surgical abortion did not experience more side effects or adverse vaginal bleeding outcomes or clinically significant changes in coagulation parameters than did women who used a placebo, an IUD, a nonhormonal contraceptive method, or delayed COC initiation (*96–102*). Limited evidence on women using the ring immediately after first trimester medical or surgical abortion found no serious adverse events and no infection related to use of the combined vaginal ring during 3 cycles of follow-up postabortion (*103*).
b. Second trimester	1	
c. Immediate postseptic abortion	1	
Past ectopic pregnancy	1	**Comment:** The risk for future ectopic pregnancy is increased among women who have had an ectopic pregnancy in the past. CHCs protect against pregnancy in general, including ectopic gestation.
History of pelvic surgery	1	—
Smoking		**Evidence:** COC users who smoked were at increased risk for cardiovascular diseases, especially myocardial infarction, compared with those who did not smoke. Studies also showed an increased risk for myocardial infarction with increasing number of cigarettes smoked per day (*104–116*).
a. Age <35 years	2	
b. Age ≥35 years		
i. <15 cigarettes per day	3	
ii. ≥15 cigarettes per day	4	
Obesity		**Evidence:** Obese women who use COCs are more likely than obese women who do not use COCs to experience VTE. Research examining the interaction between COCs and BMI on VTE risk is limited, particularly for women in the highest BMI categories (BMI ≥35 kg/m^2). Although the absolute risk for VTE in otherwise healthy women of reproductive age is small, obese women are at 2–3 times higher risk for VTE than normal weight women regardless of COC use. Limited evidence suggests that obese women who use COCs do not have a higher risk for acute myocardial infarction or stroke than do obese nonusers (*117*). Limited evidence suggests that effectiveness of some COC formulations might decrease with increasing BMI, however the observed reductions in effectiveness are minimal and evidence is conflicting (*118–125*). Effectiveness of the patch might be reduced in women >90 kg (*126*). Limited evidence suggests obese women are no more likely to gain weight during COC or vaginal ring use than normal weight or overweight women (*117*,*127*).
a. BMI ≥30 kg/m^2	2	
b. Menarche to <18 years and BMI ≥30 kg/m^2	2	
History of bariatric surgery This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).		
a. Restrictive procedures: decrease storage capacity of the stomach (vertical banded gastroplasty, laparoscopic adjustable gastric band, or laparoscopic sleeve gastrectomy)	1	**Evidence:** Limited evidence demonstrated no substantial decrease in effectiveness of oral contraceptives among women who underwent laparoscopic placement of an adjustable gastric band (*128*).
b. Malabsorptive procedures: decrease absorption of nutrients and calories by shortening the functional length of the small intestine (Roux-en-Y gastric bypass or biliopancreatic diversion)	COCs: 3 Patch and ring: 1	**Evidence:** Limited evidence demonstrated no substantial decrease in effectiveness of oral contraceptives among women who underwent a biliopancreatic diversion; however, evidence from pharmacokinetic studies reported conflicting results of oral contraceptive effectiveness among women who underwent a jejunoileal bypass (*128*). **Comment:** Bariatric surgical procedures involving a malabsorptive component have the potential to decrease oral contraceptive effectiveness, perhaps further decreased by postoperative complications, such as long-term diarrhea or vomiting.
Cardiovascular Disease		
Multiple risk factors for atherosclerotic cardiovascular disease (e.g., older age, smoking, diabetes, hypertension, low HDL, high LDL, or high triglyceride levels)	3/4	**Clarification:** When a woman has multiple major risk factors, any of which alone would substantially increase her risk for cardiovascular disease, use of CHCs might increase her risk to an unacceptable level. However, a simple addition of categories for multiple risk factors is not intended; for example, a combination of two category 2 risk factors might not necessarily warrant a higher category. **Clarification:** The recommendations apply to known preexisting medical conditions or characteristics. Few if any screening tests are needed before initiation of contraception. See the *U.S. Selected Practice Recommendations for Contraceptive Use* (http://www.cdc.gov/reproductivehealth/unintendedpregnancy/usspr.htm).

See table footnotes on page 69.

TABLE D1. (*Continued*) Classifications for combined hormonal contraceptives, including pill, patch, and ring

Condition	Category CHCs	Clarifications/Evidence/Comments
Hypertension Systolic blood pressure ≥160 mm Hg or diastolic blood pressure ≥100 mm Hg are associated with increased risk for adverse health events as a result of pregnancy (Box 2).		
a. Adequately controlled hypertension	3	**Clarification:** For all categories of hypertension, classifications are based on the assumption that no other risk factors exist for cardiovascular disease. When multiple risk factors do exist, risk for cardiovascular disease might increase substantially. A single reading of blood pressure level is not sufficient to classify a woman as hypertensive. **Clarification:** Women adequately treated for hypertension are at reduced risk for acute myocardial infarction and stroke compared with untreated women. Although no data exist, CHC users with adequately controlled and monitored hypertension should be at reduced risk for acute myocardial infarction and stroke compared with untreated hypertensive CHC users. **Evidence:** Among women with hypertension, COC users were at higher risk than nonusers for stroke, acute myocardial infarction, and peripheral arterial disease (*104*,*106*,*113–116*,*129–143*). Discontinuation of COCs in women with hypertension might improve blood pressure control (*144*).
b. Elevated blood pressure levels (properly taken measurements)		**Clarification:** For all categories of hypertension, classifications are based on the assumption that no other risk factors exist for cardiovascular disease. When multiple risk factors do exist, risk for cardiovascular disease might increase substantially. A single reading of blood pressure level is not sufficient to classify a woman as hypertensive. **Evidence:** Among women with hypertension, COC users were at higher risk than nonusers for stroke, acute myocardial infarction, and peripheral arterial disease (*104*,*106*,*113–116*,*129–143*). Discontinuation of COCs in women with hypertension might improve blood pressure control (*144*).
i. Systolic 140–159 mm Hg or diastolic 90–99 mm Hg	3	
ii. Systolic ≥160 mm Hg or diastolic ≥100 mm Hg	4	
c. Vascular disease	4	
History of high blood pressure during pregnancy (when current blood pressure is measurable and normal)	2	**Evidence:** Women with a history of high blood pressure in pregnancy who also used COCs had a higher risk for myocardial infarction and VTE than did COC users who did not have a history of high blood pressure during pregnancy. The absolute risks for acute myocardial infarction and VTE in this population remained small (*115*,*130*,*142*,*143*,*145–151*).
Deep venous thrombosis/Pulmonary embolism		
a. History of DVT/PE, not receiving anticoagulant therapy		
i. Higher risk for recurrent DVT/PE (one or more risk factors) • History of estrogen-associated DVT/PE • Pregnancy-associated DVT/PE • Idiopathic DVT/PE • Known thrombophilia, including antiphospholipid syndrome • Active cancer (metastatic, receiving therapy, or within 6 months after clinical remission), excluding nonmelanoma skin cancer • History of recurrent DVT/PE	4	—
ii. Lower risk for recurrent DVT/PE (no risk factors)	3	—
b. Acute DVT/PE	4	—
c. DVT/PE and established anticoagulant therapy for at least 3 months		**Clarification:** Women using anticoagulant therapy are at risk for gynecologic complications of therapy, such as hemorrhagic ovarian cysts and severe menorrhagia. Hormonal contraceptive methods can be of benefit in preventing or treating these complications. When a contraceptive method is used as a therapy, rather than solely to prevent pregnancy, the risk/benefit ratio might differ and should be considered on a case-by-case basis.
i. Higher risk for recurrent DVT/PE (one or more risk factors) • Known thrombophilia, including antiphospholipid syndrome • Active cancer (metastatic, receiving therapy, or within 6 months after clinical remission), excluding nonmelanoma skin cancer • History of recurrent DTV/PE	4	
ii. Lower risk for recurrent DVT/PE (no risk factors)	3	
d. Family history (first-degree relatives)	2	**Comment:** Some conditions that increase the risk for DTV/PE are heritable.
e. Major surgery		
i. With prolonged immobilization	4	—
ii. Without prolonged immobilization	2	—
f. Minor surgery without immobilization	1	—
Known thrombogenic mutations (e.g., factor V Leiden; prothrombin mutation; and protein S, protein C, and antithrombin deficiencies) This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	4	**Clarification**: Routine screening is not appropriate because of the rarity of the conditions and the high cost of screening. **Evidence:** Among women with thrombogenic mutations, COC users had a twofold to twentyfold higher risk for thrombosis than did nonusers (*152–175*).
Superficial venous disorders		
a. Varicose veins	1	**Evidence:** One study suggested that among women with varicose veins, the rate of VTE and superficial venous thrombosis was higher in oral contraceptive users compared with nonusers; however, statistical significance was not reported and the number of events was small (*176*).

See table footnotes on page 69.

TABLE D1. (*Continued*) Classifications for combined hormonal contraceptives, including pill, patch, and ring

Condition	Category CHCs	Clarifications/Evidence/Comments
b. Superficial venous thrombosis (acute or history)	3	**Clarification:** Superficial venous thrombosis might be associated with an increased risk for VTE. If a woman has risk factors for concurrent DVT (e.g., known thrombophilia or cancer) or has current or history of DVT, see recommendations for DVT/PE. Superficial venous thrombosis associated with a peripheral intravenous catheter is less likely to be associated with additional thrombosis and use of CHCs may be considered. **Evidence:** One study demonstrated that among women with superficial venous thrombosis, the risk for VTE was higher in oral contraceptive users compared with nonusers (*176*).
Current and history of ischemic heart disease This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	4	—
Stroke (history of cerebrovascular accident) This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	4	—
Valvular heart disease Complicated valvular heart disease is associated with increased risk for adverse health events as a result of pregnancy (Box 2).		
a. Uncomplicated	2	—
b. Complicated (pulmonary hypertension, risk for atrial fibrillation, or history of subacute bacterial endocarditis)	4	**Comment:** Among women with valvular heart disease, CHC use may further increase the risk for arterial thrombosis; women with complicated valvular heart disease are at greatest risk.
Peripartum cardiomyopathy This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).		**Evidence:** No direct evidence exists about the safety of CHCs among women with peripartum cardiomyopathy. Limited indirect evidence from noncomparative studies of women with cardiac disease demonstrated few cases of hypertension and transient ischemic attack in women with cardiac disease using COCs. No cases of heart failure were reported (*177*).
a. Normal or mildly impaired cardiac function (New York Heart Association Functional Class I or II: patients with no limitation of activities or patients with slight, mild limitation of activity) (*178*)		
i. <6 months	4	**Comment:** COCs might increase fluid retention in healthy women; fluid retention may worsen heart failure in women with peripartum cardiomyopathy. COCs might induce cardiac arrhythmias in healthy women; women with peripartum cardiomyopathy have a high incidence of cardiac arrhythmias.
ii. ≥6 months	3	
b. Moderately or severely impaired cardiac function (New York Heart Association Functional Class III or IV: patients with marked limitation of activity or patients who should be at complete rest) (*178*)	4	
Rheumatic Diseases		
Systemic lupus erythematosus This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).		
a. Positive (or unknown) antiphospholipid antibodies	4	**Clarification:** Persons with SLE are at increased risk for ischemic heart disease, stroke, and VTE. Categories assigned to such conditions in U.S. MEC should be the same for women with SLE who have these conditions. For all subconditions of SLE, classifications are based on the assumption that no other risk factors for cardiovascular disease are present; these classifications must be modified in the presence of such risk factors. Many women with SLE can be considered good candidates for most contraceptive methods, including hormonal contraceptives (*179–197*). **Evidence:** Antiphospholipid antibodies are associated with a higher risk for both arterial and venous thrombosis (*198*,*199*).
b. Severe thrombocytopenia	2	**Clarification:** Persons with SLE are at increased risk for ischemic heart disease, stroke, and VTE. Categories assigned to such conditions in U.S. MEC should be the same for women with SLE who have these conditions. For all subconditions of SLE, classifications are based on the assumption that no other risk factors for cardiovascular disease are present; these classifications must be modified in the presence of such risk factors. Many women with SLE can be considered good candidates for most contraceptive methods, including hormonal contraceptives (*179–197*).
c. Immunosuppressive therapy	2	**Clarification:** Persons with SLE are at increased risk for ischemic heart disease, stroke, and VTE. Categories assigned to such conditions in U.S. MEC should be the same for women with SLE who have these conditions. For all subconditions of SLE, classifications are based on the assumption that no other risk factors for cardiovascular disease are present; these classifications must be modified in the presence of such risk factors. Many women with SLE can be considered good candidates for most contraceptive methods, including hormonal contraceptives (*179–197*).
d. None of the above	2	**Clarification:** Persons with SLE are at increased risk for ischemic heart disease, stroke, and VTE. Categories assigned to such conditions in U.S. MEC should be the same for women with SLE who have these conditions. For all subconditions of SLE, classifications are based on the assumption that no other risk factors for cardiovascular disease are present; these classifications must be modified in the presence of such risk factors. Many women with SLE can be considered good candidates for most contraceptive methods, including hormonal contraceptives (*179–197*).

See table footnotes on page 69.

TABLE D1. (*Continued*) Classifications for combined hormonal contraceptives, including pill, patch, and ring

Condition	Category CHCs	Clarifications/Evidence/Comments
Rheumatoid arthritis		**Evidence:** Limited evidence shows no consistent pattern of improvement or worsening of rheumatoid arthritis with use of oral contraceptives, progesterone, or estrogen (*200*).
a. Receiving immunosuppressive therapy	2	
b. Not receiving immunosuppressive therapy	2	
Neurologic Conditions		
Headaches		
a. Nonmigraine (mild or severe)	1	**Clarification:** Classification depends on accurate diagnosis of those severe headaches that are migraines and those headaches that are not, as well as diagnosis of ever experiencing aura. Aura is a specific focal neurologic symptom. For more information about headache classification see *The International Headache Society Classification, 3rd edition* (http://www.ihs-classification.org/_downloads/mixed/International-Headache-Classification-III-ICHD-III-2013-Beta.pdf). Any new headaches or marked changes in headaches should be evaluated.
b. Migraine		**Clarification:** Classification depends on accurate diagnosis of those severe headaches that are migraines and those headaches that are not, as well as diagnosis of ever experiencing aura. Aura is a specific focal neurologic symptom. For more information about headache classification see *The International Headache Society Classification, 3rd edition* (http://www.ihs-classification.org/_downloads/mixed/International-Headache-Classification-III-ICHD-III-2013-Beta.pdf). Any new headaches or marked changes in headaches should be evaluated.
i. Without aura (This category of migraine includes menstrual migraine.)	2	
ii. With aura	4	**Clarification:** Classification is for women without any other risk factors for stroke (e.g., age, hypertension, and smoking). **Evidence:** Among women with migraine, oral contraceptive use is associated with about a threefold increased risk for ischemic stroke compared with nonuse, although most studies did not specify migraine type or oral contraceptive formulation. The only study to examine migraine type found that the risk for ischemic stroke among women with migraine with aura was increased to a similar level among both oral contraceptive users and nonusers, compared with women without migraine (*201*). The risk for ischemic stroke is increased among women using COCs, compared with women not using COCs (*104*,*202*). The risk for ischemic stroke is also increased among women with migraine with aura, compared with women without migraine (*203*–*205*). One older meta-analysis found that migraine without aura was associated with an increased risk for ischemic stroke, while two more recent meta-analyses did not find such an association (*203*–*205*). **Comment:** Menstrual migraine is a subtype of migraine without aura. For more information, see *The International Headache Society Classification, 3rd edition* (http://www.ihs-classification.org/_downloads/mixed/International-Headache-Classification-III-ICHD-III-2013-Beta.pdf).
Epilepsy This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	1	**Clarification:** If a woman is taking anticonvulsants, see Drug Interactions section. Certain anticonvulsants lower COC effectiveness. The extent to which patch or ring use is similar to COC use in this regard remains unclear.
Multiple sclerosis		**Evidence:** Limited evidence suggests that use of COCs or oral contraceptives (type not specified) among women with multiple sclerosis does not worsen the clinical course of disease (*206*). **Comment:** No data exist that evaluate the increased risk for VTE among women with multiple sclerosis using CHCs. However, women with multiple sclerosis are at higher risk than unaffected women for VTE.
a. With prolonged immobility	3	
b. Without prolonged immobility	1	
Depressive Disorders		
Depressive disorders	1	**Clarification**: If a woman is receiving psychotropic medications or St. John's wort, see Drug Interactions section. **Evidence**: COC use was not associated with increased depressive symptoms in women with depression or scoring above threshold levels on a validated depression screening instrument compared with baseline or with nonusers with depression. One small study of women with bipolar disorder found that oral contraceptives did not significantly change mood across the menstrual cycle (*207*).
Reproductive Tract Infections and Disorders		
Vaginal bleeding patterns		
a. Irregular pattern without heavy bleeding	1	**Comment:** Irregular menstrual bleeding patterns are common among healthy women.
b. Heavy or prolonged bleeding (includes regular and irregular patterns)	1	**Clarification:** Unusually heavy bleeding should raise the suspicion of a serious underlying condition. **Evidence:** A Cochrane Collaboration Review identified one randomized controlled trial evaluating the effectiveness of COC use compared with naproxen and danazol in treating menorrhagia. Women with menorrhagia did not report worsening of the condition or any adverse events related to COC use (*208*).

See table footnotes on page 69.

TABLE D1. (*Continued*) Classifications for combined hormonal contraceptives, including pill, patch, and ring

Condition	Category CHCs	Clarifications/Evidence/Comments
Unexplained vaginal bleeding (suspicious for serious condition) before evaluation	2	**Clarification:** If pregnancy or an underlying pathological condition (e.g., pelvic malignancy) is suspected, it must be evaluated and the category adjusted after evaluation.
		Comment: No conditions that cause vaginal bleeding will be worsened in the short-term by use of CHCs.
Endometriosis	1	**Evidence:** A Cochrane Collaboration Review identified one randomized controlled trial evaluating the effectiveness of COC use compared with a gonadotropin-releasing hormone analog in treating the symptoms of endometriosis. Women with endometriosis did not report worsening of the condition or any adverse events related to COC use (*209*).
Benign ovarian tumors (including cysts)	1	
Severe dysmenorrhea	1	**Evidence:** Risk for side effects with COC use was not higher among women with dysmenorrhea than among women not using COCs. Some COC users had a reduction in pain and bleeding (*210*,*211*).
Gestational trophoblastic disease This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).		**Clarification:** For all subconditions of gestational trophoblastic disease, classifications are based on the assumption that women are under close medical supervision because of the need for monitoring of β-hCG levels for appropriate disease surveillance.
a. Suspected gestational trophoblastic disease (immediate postevacuation)		**Evidence:** After molar pregnancy evacuation, the balance of evidence found COC use did not increase the risk for postmolar trophoblastic disease, and β–hCG levels regressed more rapidly in some COC users than in nonusers (*212*). Limited evidence suggests that use of COCs during chemotherapy does not significantly affect the regression or treatment of postmolar trophoblastic disease compared with women who used a nonhormonal contraceptive method or DMPA during chemotherapy (*212*).
i. Uterine size first trimester	1	
ii. Uterine size second trimester	1	
b. Confirmed gestational trophoblastic disease (after initial evacuation and during monitoring)		
i. Undetectable/nonpregnant β-hCG levels	1	
ii. Decreasing β-hCG levels	1	
iii. Persistently elevated β-hCG levels or malignant disease, with no evidence or suspicion of intrauterine disease	1	
iv. Persistently elevated β-hCG levels or malignant disease, with evidence or suspicion of intrauterine disease	1	
Cervical ectropion	1	**Comment:** Cervical ectropion is not a risk factor for cervical cancer, and restriction of CHC use is unnecessary.
Cervical intraepithelial neoplasia	2	**Evidence:** Among women with persistent human papillomavirus infection, long-term COC use (≥5 years) might increase the risk for carcinoma in situ and invasive carcinoma (*213*). Limited evidence on women with low-grade squamous intraepithelial lesions found use of the vaginal ring did not worsen the condition (*9*).
Cervical cancer (awaiting treatment)	2	**Comment:** Theoretical concern exists that CHC use might affect prognosis of the existing disease. While awaiting treatment, women may use CHCs. In general, treatment of this condition can render a woman sterile.
Breast disease Breast cancer is associated with increased risk for adverse health events as a result of pregnancy (Box 2).		
a. Undiagnosed mass	2	**Clarification:** The woman should be evaluated as early as possible.
b. Benign breast disease	1	—
c. Family history of cancer	1	**Evidence:** Women with breast cancer susceptibility genes (e.g., *BRCA1* and *BRCA2*) have a higher baseline risk for breast cancer than women without these genes. The baseline risk for breast cancer is also higher among women with a family history of breast cancer than among those who do not have such a history. However, evidence does not suggest that the increased risk for breast cancer among women with either a family history of breast cancer or breast cancer susceptibility genes is modified by the use of COCs (*214–231*).
d. Breast cancer		**Comment:** Breast cancer is a hormonally sensitive tumor, and the prognosis for women with current or recent breast cancer might worsen with CHC use.
i. Current	4	
ii. Past and no evidence of current disease for 5 years	3	
Endometrial hyperplasia	1	—
Endometrial cancer This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	1	**Comment:** COC use reduces the risk for endometrial cancer; whether patch or ring use reduces the risk for endometrial cancer is not known. While awaiting treatment, women may use CHCs. In general, treatment of this condition renders a woman sterile.
Ovarian cancer This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	1	**Comment:** COC use reduces the risk for ovarian cancer; whether patch or ring use reduces the risk for ovarian cancer is not known. While awaiting treatment, women may use CHCs. In general, treatment of this condition can render a woman sterile.
Uterine fibroids	1	**Comment:** COCs do not appear to cause growth of uterine fibroids, and patch and ring also are not expected to cause growth.

See table footnotes on page 69.

TABLE D1. (*Continued*) Classifications for combined hormonal contraceptives, including pill, patch, and ring

Condition	Category CHCs	Clarifications/Evidence/Comments
Pelvic inflammatory disease		**Comment:** COCs might reduce the risk for PID among women with STDs but do not protect against HIV or lower genital tract STDs. Whether use of patch or ring reduces the risk for PID among women with STDs is unknown; however, they do not protect against HIV or lower genital tract STDs.
a. Past PID		
i. With subsequent pregnancy	1	
ii. Without subsequent pregnancy	1	
b. Current PID	1	
Sexually transmitted diseases		
a. Current purulent cervicitis or chlamydial infection or gonococcal infection	1	—
b. Vaginitis (including *Trichomonas vaginalis* and bacterial vaginosis)	1	—
c. Other factors related to STDs	1	—
HIV		
High risk for HIV	1	**Evidence:** Overall, evidence does not support an association between oral contraceptives and risk for HIV acquisition (*232*).
HIV infection For women with HIV infection who are not clinically well or not receiving ARV therapy, this condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	1	**Clarification:** Drug interactions might exist between hormonal contraceptives and ARV drugs; see Drug Interactions section. **Evidence:** Overall, evidence does not support an association between COC use and progression of HIV. Limited direct evidence does not support an association between COC use and transmission of HIV to noninfected partners; studies measuring genital viral shedding as a proxy for infectivity have had mixed results. Studies measuring whether hormonal contraceptive methods affect plasma HIV viral load generally have found no effect (*233–235*).
Other Infections		
Schistosomiasis Schistosomiasis with fibrosis of the liver is associated with increased risk for adverse health events as a result of pregnancy (Box 2).		
a. Uncomplicated	1	**Evidence:** Among women with uncomplicated schistosomiasis, COC use had no adverse effects on liver function (*236–242*).
b. Fibrosis of the liver (if severe, see Cirrhosis section)	1	—
Tuberculosis This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).		**Clarification:** If a woman is taking rifampin, see Drug Interactions section. Rifampin is likely to decrease COC effectiveness. The extent to which patch or ring use is similar to COC use in this regard remains unclear.
a. Nonpelvic	1	
b. Pelvic	1	
Malaria	1	—
Endocrine Conditions		
Diabetes Insulin-dependent diabetes; diabetes with nephropathy, retinopathy, or neuropathy; diabetes with other vascular disease; or diabetes of >20 years' duration are associated with increased risk for adverse health events as a result of pregnancy (Box 2).		
a. History of gestational disease	1	**Evidence:** The development of non–insulin-dependent diabetes in women with a history of gestational diabetes is not increased by use of COCs (*243–250*). Likewise, lipid levels appear to be unaffected by COC use (*251–253*).
b. Nonvascular disease		**Evidence:** Among women with insulin-dependent or non–insulin-dependent diabetes, COC use had limited effect on daily insulin requirements and no effect on long-term diabetes control (e.g., glycosylated hemoglobin levels) or progression to retinopathy. Changes in lipid profile and hemostatic markers were limited, and most changes remained within normal values (*254–263*).
i. Non-insulin dependent	2	
ii. Insulin dependent	2	
c. Nephropathy, retinopathy, or neuropathy	3/4	**Clarification:** The category should be assessed according to the severity of the condition.
d. Other vascular disease or diabetes of >20 years' duration	3/4	**Clarification:** The category should be assessed according to the severity of the condition.
Thyroid disorders		
a. Simple goiter	1	—
b. Hyperthyroid	1	—
c. Hypothyroid	1	—
Gastrointestinal Conditions		
Inflammatory bowel disease (ulcerative colitis or Crohn's disease)	2/3	**Clarification:** For women with mild IBD and with no other risk factor for VTE, the benefits of CHC use generally outweigh the risks (category 2). However, for women with IBD who are at increased risk for VTE (e.g., those with active or extensive disease, surgery, immobilization, corticosteroid use, vitamin deficiencies, or fluid depletion), the risks of CHC use generally outweigh the benefits (category 3). **Evidence:** Risk for disease relapse was not significantly higher among women with IBD using oral contraceptives (most studies did not specify type) than among nonusers (*264*). Absorption of COCs among women with mild ulcerative colitis and no or small ileal resections was similar to the absorption among healthy women (*264*). Findings might not apply to women with Crohn's disease or more extensive bowel resections. No data exist that evaluate the increased risk for VTE among women with IBD using CHCs. However, women with IBD are at higher risk than unaffected women for VTE (*264*).

See table footnotes on page 69.

TABLE D1. (*Continued*) Classifications for combined hormonal contraceptives, including pill, patch, and ring

Condition	Category CHCs		Clarifications/Evidence/Comments
Gallbladder disease			**Comment:** CHCs might cause a small increased risk for gallbladder disease. CHCs might worsen existing gallbladder disease.
a. Symptomatic			
i. Treated by cholecystectomy	2		
ii. Medically treated	3		
iii. Current	3		
b. Asymptomatic	2		
History of cholestasis			
a. Pregnancy related	2		**Comment:** History of pregnancy-related cholestasis might predict an increased risk for COC-related cholestasis.
b. Past COC related	3		**Comment:** History of COC-related cholestasis predicts an increased risk with subsequent COC use.
Viral hepatitis	Initiation	Continuation	
a. Acute or flare	3/4	2	**Clarification (initiation):** The category should be assessed according to the severity of the condition. **Evidence:** Data suggest that in women with chronic hepatitis, COC use does not increase the rate or severity of cirrhotic fibrosis, nor does it increase the risk for hepatocellular carcinoma. For women who are carriers, COC use does not appear to trigger liver failure or severe dysfunction. Evidence is limited for COC use during active hepatitis (*265*).
b. Carrier	1	1	**Evidence:** Data suggest that in women with chronic hepatitis, COC use does not increase the rate or severity of cirrhotic fibrosis, nor does it increase the risk for hepatocellular carcinoma. For women who are carriers, COC use does not appear to trigger liver failure or severe dysfunction. Evidence is limited for COC use during active hepatitis (*265*).
c. Chronic	1	1	**Evidence:** Data suggest that in women with chronic hepatitis, COC use does not increase the rate or severity of cirrhotic fibrosis, nor does it increase the risk for hepatocellular carcinoma. For women who are carriers, COC use does not appear to trigger liver failure or severe dysfunction. Evidence is limited for COC use during active hepatitis (*265*).
Cirrhosis Severe cirrhosis is associated with increased risk for adverse health events as a result of pregnancy (Box 2).			
a. Mild (compensated)	1		—
b. Severe (decompensated)	4		—
Liver tumors Hepatocellular adenoma and malignant liver tumors are associated with increased risk for adverse health events as a result of pregnancy (Box 2).			
a. Benign			
i. Focal nodular hyperplasia	2		**Evidence:** Limited direct evidence suggests that hormonal contraceptive use does not influence either progression or regression of liver lesions among women with focal nodular hyperplasia (*266*).
ii. Hepatocellular adenoma	4		—
b. Malignant (hepatoma)	4		—
Respiratory Conditions			
Cystic fibrosis This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	1		**Clarification:** Persons with cystic fibrosis are at increased risk for diabetes, liver disease, gallbladder disease, and VTE (particularly related to use of central venous catheters) and are frequently prescribed antibiotics. Categories assigned to such conditions in U.S. MEC should be the same for women with cystic fibrosis who have these conditions. For cystic fibrosis, classifications are based on the assumption that no other conditions are present; these classifications must be modified in the presence of such conditions. **Clarification:** Certain drugs to treat cystic fibrosis (e.g., lumacaftor) might reduce effectiveness of hormonal contraceptives, including oral, injectable, transdermal, and implantable contraceptives. **Evidence:** Limited evidence suggests that use of COCs or oral contraceptives (type not specified) among women with cystic fibrosis is not associated with worsening of disease severity. Very limited evidence suggests that cystic fibrosis does not impair the effectiveness of hormonal contraception (*267*).
Anemias			
Thalassemia	1		**Comment:** Anecdotal evidence from countries where thalassemia is prevalent indicates that COC use does not worsen the condition.
Sickle cell disease This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	2		—
Iron deficiency anemia	1		**Comment:** CHC use might decrease menstrual blood loss.
Solid Organ Transplantation			
Solid organ transplantation This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).			

See table footnotes on page 69.

TABLE D1. (*Continued*) Classifications for combined hormonal contraceptives, including pill, patch, and ring

Condition	Category CHCs	Clarifications/Evidence/Comments
a. Complicated: graft failure (acute or chronic), rejection, cardiac allograft vasculopathy	4	**Evidence:** Limited evidence of COC and patch users indicated no overall changes in biochemical measures. However, one study reported discontinuations of COC use in two (8%) of 26 women as a result of serious medical complications, and in one case report, a woman developed cholestasis associated with high-dose COC use (*268*).
b. Uncomplicated	2	**Clarification:** Women with Budd-Chiari syndrome should not use CHCs because of the increased risk for thrombosis. **Evidence:** Limited evidence of COC and patch users indicated no overall changes in biochemical measures. However, one study reported discontinuations of COC use in two (8%) of 26 women as a result of serious medical complications, and in one case report, a woman developed cholestasis associated with high-dose COC use (*268*).
Drug Interactions		
Antiretroviral therapy		**Comment:** These recommendations generally are for ARV agents used alone. However, most women receiving ARV therapy are using multiple drugs in combination. In general, whether interactions between ARVs and hormonal contraceptives differ when ARVs are given alone or in combination is unknown.
a. Nucleoside reverse transcriptase inhibitors (NRTIs)		
i. Abacavir (ABC)	1	**Evidence:** NRTIs do not appear to have significant risk for interactions with hormonal contraceptive methods (*269–274*).
ii. Tenofovir (TDF)	1	
iii. Zidovudine (AZT)	1	
iv. Lamivudine (3TC)	1	
v. Didanosine (DDI)	1	
vi. Emtricitabine (FTC)	1	
vii. Stavudine (D4T)	1	
b. Nonnucleoside reverse transcriptase inhibitors (NNRTIs)		
i. Efavirenz (EFV)	2	**Clarification:** Evidence suggests drug interactions between EFV and certain hormonal contraceptives. These interactions might reduce the effectiveness of the hormonal contraceptive. **Evidence:** Two studies suggested that pregnancy rates might be higher among women using COCs and EFV compared with COCs alone, although one study found no difference in pregnancy rates (*275–277*) Two studies found conflicting results on ovulations in women receiving COCs and EFV compared with EFV alone (*278*,*279*). Two pharmacokinetic studies demonstrated decreases in ethinyl estradiol and progestin concentrations in women receiving COCs and EFV compared with COCs alone (*279*,*280*). Pharmacokinetic studies demonstrated generally no changes in EFV concentrations with concomitant COC use (*279*,*280*).
ii. Etravirine (ETR)	1	**Evidence:** One study demonstrated no clinically relevant pharmacokinetic or pharmacodynamic changes in women using COCs and ETR compared with COCs alone (*281*).
iii. Nevirapine (NVP)	1	**Evidence:** Five studies found no significant differences in pregnancy rates among women using COCs and NVP compared with women using COCs alone (*275–277*,*282*,*283*). Three studies reported no ovulations among women receiving COCs and NVP (*278*,*283*,*284*). Two pharmacokinetic studies demonstrated decreased concentrations of ethinyl estradiol and progestin among women using COCs and NVP compared with COCs alone, and one study found no change in contraceptive hormone concentrations (*278*,*284*,*285*). Pharmacokinetic studies demonstrated generally no changes in NVP concentrations with concomitant COC use (*278*,*285*,*286*).
iv. Rilpivirine (RPV)	1	**Evidence:** One study demonstrated no clinical significant pharmacokinetic changes or adverse events in women using COCs and RPV compared with COCs alone (*287*).
c. Ritonavir-boosted protease inhibitors		
i. Ritonavir-boosted atazanavir (ATV/r)	2	**Clarification:** Theoretically, drug interactions might occur between certain ritonavir-boosted protease inhibitors and certain hormonal contraceptives that might reduce the effectiveness of the hormonal contraceptive. **Evidence:** One pharmacokinetic study demonstrated decreased estrogen but increased progestin concentrations in women using COCs and ATV/r compared with COCs alone (*288*).
ii. Ritonavir-boosted darunavir (DRV/r)	2	**Clarification:** Theoretically, drug interactions might occur between certain ritonavir-boosted protease inhibitors and certain hormonal contraceptives that might reduce the effectiveness of the hormonal contraceptive. **Evidence:** One pharmacokinetic study demonstrated no change in follicle-stimulating hormone or luteinizing hormone but decreases in ethinyl estradiol and norethindrone in women using COCs with DRV/r compared with COCs alone (*289*).

See table footnotes on page 69.

TABLE D1. (*Continued*) Classifications for combined hormonal contraceptives, including pill, patch, and ring

Condition	Category CHCs	Clarifications/Evidence/Comments
iii. Ritonavir-boosted fosamprenavir (FPV/r)	2	**Clarification:** Theoretically, drug interactions might occur between certain ritonavir-boosted protease inhibitors and certain hormonal contraceptives that might reduce the effectiveness of the hormonal contraceptive. **Evidence:** Information from the package label states that both ethinyl estradiol and norethindrone concentrations decreased with concurrent administration of COCs and FPV/r (*290*).
iv. Ritonavir-boosted lopinavir (LPV/r)	1	**Evidence:** One study demonstrated a non-significant increase in pregnancy rates among women using COCs and LPV/r compared with COCs alone (*275*). One study demonstrated no ovulations in women using the combined hormonal patch and LPV/r compared with combined hormonal patch alone; ethinyl estradiol concentrations for COC and patch users decreased but norelgestromin concentrations increased with use of the patch (*291*).
v. Ritonavir-boosted saquinavir (SQV/r)	2	**Clarification:** Theoretically, drug interactions might occur between certain ritonavir-boosted protease inhibitors and certain hormonal contraceptives that might reduce the effectiveness of the hormonal contraceptive. **Evidence:** One pharmacokinetic study demonstrated no change in SQV concentrations in women using COC and SQV compared with COCs alone (*292*).
iv. Ritonavir-boosted tipranavir (TPV/r)	2	**Clarification:** Theoretically, drug interactions might occur between certain ritonavir-boosted protease inhibitors and certain hormonal contraceptives that might reduce the effectiveness of the hormonal contraceptive. **Evidence:** Information from the package label states that ethinyl estradiol concentrations decrease but norethindrone concentrations increased with concurrent administration of COCs and TPV/r (*293*).
d. Protease inhibitors without ritonavir		
i. Atazanavir (ATV)	2	**Clarification:** Theoretical concern exists that increased levels of ethinyl estradiol because of interactions with ATV might increase the risk for adverse events. **Evidence:** Information from the package label states that there are inconsistent changes in ethinyl estradiol concentrations and increases in progestin concentrations with concurrent administration of two different COCs and ATV (*294*). **Comment:** When ATV is administered with Cobicistat, theoretical concern exists for a drug interaction with hormonal contraceptives. Cobicistat is an inhibitor of CYP3A and CYP2D6 and could theoretically increase contraceptive hormone levels. However, its effects on CYP enzymes and drug levels may vary when combined with other ARVs.
ii. Fosamprenavir (FPV)	3	**Clarification:** Concern exists that interactions between FPV and hormonal contraceptives leading to decreased levels of FPV might diminish effectiveness of the ARV drug. **Evidence:** Information from the package label states that amprenavir concentrations decreased with concurrent administration of COCs and amprenavir. Norethindrone concentrations increased and ethinyl estradiol concentrations did not change (*290*).
iii. Indinavir (IDV)	1	**Evidence:** One small study found no pregnancies in women using COCs and IDV (*277*).
iv. Nelfinavir (NFV)	2	**Clarification:** Evidence suggests drug interactions between certain protease inhibitors and certain hormonal contraceptives. These interactions might reduce the effectiveness of the hormonal contraceptive. **Evidence:** One small study suggested that women using COCs and NFV may have had higher pregnancy rates than those using COCs alone (*277*).
e. CCR5 co-receptor antagonists		
i. Maraviroc (MVC)	1	**Evidence:** COC concentrations were not altered by co-administration with MVC (*295*).
f. HIV integrase strand transfer inhibitors		
i. Raltegravir (RAL)	1	**Evidence:** One pharmacokinetic study demonstrated increased concentrations of norgestimate and no change in ethinyl estradiol among women using COCs and RAL compared with COCs alone (*296*).
ii. Dolutegravir (DTG)	1	**Evidence:** One study demonstrated no clinically relevant pharmacokinetic or pharmacodynamic changes in women using COCs and DTG compared with COCs alone (*297*).

See table footnotes on page 69.

TABLE D1. (*Continued*) Classifications for combined hormonal contraceptives, including pill, patch, and ring

Condition	Category CHCs	Clarifications/Evidence/Comments
iii. Elvitegravir (EVG)	1	**Evidence:** Information from the package label states that ethinyl estradiol concentrations decreased and norgestimate concentrations increased with concurrent administration of COCs and EVG (*298*).
		Comment: When ATV is administered with Cobicistat, theoretical concern exists for a drug interaction with hormonal contraceptives. Cobicistat is an inhibitor of CYP3A and CYP2D6 and could theoretically increase contraceptive hormone levels. However, its effects on CYP enzymes and drug levels may vary when combined with other ARVs.
g. Fusion inhibitors		
i. Enfuvirtide	1	—
Anticonvulsant therapy		
a. Certain anticonvulsants (phenytoin, carbamazepine, barbiturates, primidone, topiramate, oxcarbazepine)	3	**Clarification:** Although the interaction of certain anticonvulsants with CHCs is not harmful to women, it is likely to reduce the effectiveness of CHCs. Use of other contraceptives should be encouraged for women who are long-term users of any of these drugs. When a COC is chosen, a preparation containing a minimum of 30 μg ethinyl estradiol should be used.
		Evidence: Use of certain anticonvulsants might decrease the effectiveness of COCs (*299–302*).
b. Lamotrigine	3	**Clarification:** The recommendation for lamotrigine applies only for situations where lamotrigine monotherapy is taken concurrently with COCs. Anticonvulsant treatment regimens that combine lamotrigine and non–enzyme-inducing antiepileptic drugs (e.g., sodium valproate) do not interact with COCs.
		Evidence: Pharmacokinetic studies show levels of lamotrigine decrease significantly during COC use (*303–307*). Some women who used both COCs and lamotrigine experienced increased seizure activity in one trial (*303*).
Antimicrobial therapy		
a. Broad-spectrum antibiotics	1	**Evidence:** Most broad-spectrum antibiotics do not affect the contraceptive effectiveness of COCs *(308–344)*, patch (*345*), or ring (*346*).
b. Antifungals	1	**Evidence:** Studies of antifungal agents have shown no clinically significant pharmacokinetic interactions with COCs (*347–356*), or ring (*357*).
c. Antiparasitics	1	**Evidence:** Studies of antiparasitic agents have shown no clinically significant pharmacokinetic interactions with COCs (*236*,*358–362*).
d. Rifampin or rifabutin therapy	3	**Clarification:** Although the interaction of rifampin or rifabutin therapy with CHCs is not harmful to women, it is likely to reduce the effectiveness of CHCs. Use of other contraceptives should be encouraged for women who are long-term users of either of these drugs. When a COC is chosen, a preparation containing a minimum of 30 μg ethinyl estradiol should be used.
		Evidence: The balance of the evidence suggests that rifampin reduces the effectiveness of COCs (*363–378*). Data on rifabutin are limited, but effects on metabolism of COCs are less than with rifampin, and small studies have not shown evidence of ovulation (*365*,*372*).
Psychotropic medications		**Comment:** For many common psychotropic agents, limited or no theoretical concern exists for clinically significant drug interactions when co-administered with hormonal contraceptives. However, either no or very limited data exist examining potential interactions for these classes of medications. For psychotropic agents that are CYP1A2 substrates, such as duloxetine, mirtazapine, ziprasidone, olanzapine, clomipramine, imipramine, and amitriptyline, co-administration with CHCs could theoretically yield increased concentrations of the psychotropic drug. For agents with narrow therapeutic windows, such as tricyclic antidepressants, increased drug concentrations might pose safety concerns that could necessitate closer monitoring.
a. SSRIs	1	**Evidence:** Limited clinical and pharmacokinetic data do not demonstrate concern for SSRIs decreasing the effectiveness of oral contraceptives. Limited evidence suggests that for women taking SSRIs, the use of hormonal contraceptives was not associated with differences in effectiveness of the SSRI for treatment or in adverse events when compared with women not taking hormonal contraceptives (*379*).
		Comment: Drugs that are inhibitors of CYP3A4 or CYP2C9 theoretically have the potential to increase levels of contraceptive steroids which might increase adverse events. Fluvoxamine is an SSRI known to be a moderate inhibitor of both CYP3A4 and CYP2C9; however, no clinical or pharmacokinetic studies were identified to explore potential drug-drug interactions.

See table footnotes on page 69.

TABLE D1. (*Continued*) Classifications for combined hormonal contraceptives, including pill, patch, and ring

Condition	Category CHCs	Clarifications/Evidence/Comments
St. John's wort	2	**Evidence:** Although clinical data are limited, studies with pharmacokinetic and pharmacodynamics outcomes raise concern that St. John's wort might decrease effectiveness of hormonal contraceptives, including increased risk for breakthrough bleeding and ovulation and increased metabolism of estrogen and progestins. Any interactions might be dependent on the dose of St John's wort, and the concentration of active ingredients across types of St. John's wort preparations may vary (*380*).

Abbreviations: ARV = antiretroviral; BMD = bone mineral density; BMI = body mass index; CHC = combined hormonal contraceptive; COC = combined oral contraceptive; DVT = deep venous thrombosis; hCG = human chorionic gonadotropin; HDL = high-density lipoprotein; HIV = human immunodeficiency virus; IBD = inflammatory bowel disease; LDL = low-density lipoprotein; PE = pulmonary embolism; PID = pelvic inflammatory disease; SLE = systemic lupus erythematosus; SSRI = selective serotonin reuptake inhibitor; STD = sexually transmitted infection; VTE = venous thromboembolism.

References

1. Abrams LS, Skee DM, Natarajan J, Wong FA, Lasseter KC. Multiple-dose pharmacokinetics of a contraceptive patch in healthy women participants. Contraception 2001;64:287–94. http://dx.doi.org/10.1016/S0010-7824(01)00273-6
2. Ahrendt HJ, Nisand I, Bastianelli C, et al. Efficacy, acceptability and tolerability of the combined contraceptive ring, NuvaRing, compared with an oral contraceptive containing 30 microg of ethinyl estradiol and 3 mg of drospirenone. Contraception 2006;74:451–7. http://dx.doi.org/10.1016/j.contraception.2006.07.004
3. Audet M-C, Moreau M, Koltun WD, et al; ORTHO EVRA/EVRA 004 Study Group. Evaluation of contraceptive efficacy and cycle control of a transdermal contraceptive patch vs an oral contraceptive: a randomized controlled trial. JAMA 2001;285:2347–54. http://dx.doi.org/10.1001/jama.285.18.2347
4. Bjarnadóttir RI, Tuppurainen M, Killick SR. Comparison of cycle control with a combined contraceptive vaginal ring and oral levonorgestrel/ethinyl estradiol. Am J Obstet Gynecol 2002;186:389–95. http://dx.doi.org/10.1067/mob.2002.121103
5. Boonyarangkul A, Taneepanichskul S. Comparison of cycle control and side effects between transdermal contraceptive patch and an oral contraceptive in women older than 35 years. J Med Assoc Thai 2007;90:1715–9.
6. Burkman RT. The transdermal contraceptive patch: a new approach to hormonal contraception. Int J Fertil Womens Med 2002;47:69–76.
7. Cole JA, Norman H, Doherty M, Walker AM. Venous thromboembolism, myocardial infarction, and stroke among transdermal contraceptive system users. Obstet Gynecol 2007;109:339–46. http://dx.doi.org/10.1097/01.AOG.0000250968.82370.04
8. Devineni D, Skee D, Vaccaro N, et al. Pharmacokinetics and pharmacodynamics of a transdermal contraceptive patch and an oral contraceptive. J Clin Pharmacol 2007;47:497–509. http://dx.doi.org/10.1177/0091270006297919.
9. Dieben TO, Roumen FJ, Apter D. Efficacy, cycle control, and user acceptability of a novel combined contraceptive vaginal ring. Obstet Gynecol 2002;100:585–93.
10. Dittrich R, Parker L, Rosen JB, Shangold G, Creasy GW, Fisher AC; Ortho Evra/Evra 001 Study Group. Transdermal contraception: evaluation of three transdermal norelgestromin/ethinyl estradiol doses in a randomized, multicenter, dose-response study. Am J Obstet Gynecol 2002;186:15–20. http://dx.doi.org/10.1067/mob.2002.118844
11. Duijkers I, Killick S, Bigrigg A, Dieben TO. A comparative study on the effects of a contraceptive vaginal ring NuvaRing and an oral contraceptive on carbohydrate metabolism and adrenal and thyroid function. Eur J Contracept Reprod Health Care 2004;9:131–40. http://dx.doi.org/10.1080/13625180400007199
12. Duijkers IJ, Klipping C, Verhoeven CH, Dieben TO. Ovarian function with the contraceptive vaginal ring or an oral contraceptive: a randomized study. Hum Reprod 2004;19:2668–73. http://dx.doi.org/10.1093/humrep/deh493
13. Elkind-Hirsch KE, Darensbourg C, Ogden B, Ogden LF, Hindelang P. Contraceptive vaginal ring use for women has less adverse metabolic effects than an oral contraceptive. Contraception 2007;76:348–56. http://dx.doi.org/10.1016/j.contraception.2007.08.001
14. Hedon B, Helmerhorst FM, Cronje HS, Shangold GA, Fisher AC, Creasy GW. Comparison of efficacy, cycle control, compliance, and safety in users of a contraceptive patch versus an oral contraceptive. Int J Gynaecol Obstet 2000;70(Suppl 2):B78. http://dx.doi.org/10.1016/S0020-7292(00)85161-9
15. Jick S, Kaye JA, Li L, Jick H. Further results on the risk of nonfatal venous thromboembolism in users of the contraceptive transdermal patch compared to users of oral contraceptives containing norgestimate and 35 microg of ethinyl estradiol. Contraception 2007;76:4–7. http://dx.doi.org/10.1016/j.contraception.2007.03.003
16. Jick SS, Jick H. Cerebral venous sinus thrombosis in users of four hormonal contraceptives: levonorgestrel-containing oral contraceptives, norgestimate-containing oral contraceptives, desogestrel-containing oral contraceptives and the contraceptive patch. Contraception 2006;74:290–2. http://dx.doi.org/10.1016/j.contraception.2006.05.071
17. Jick SS, Jick H. The contraceptive patch in relation to ischemic stroke and acute myocardial infarction. Pharmacotherapy 2007;27:218–20. http://dx.doi.org/10.1592/phco.27.2.218
18. Jick SS, Kaye JA, Russmann S, Jick H. Risk of nonfatal venous thromboembolism in women using a contraceptive transdermal patch and oral contraceptives containing norgestimate and 35 microg of ethinyl estradiol. Contraception 2006;73:223–8. http://dx.doi.org/10.1016/j.contraception.2006.01.001
19. Magnusdóttir EM, Bjarnadóttir RI, Onundarson PT, et al. The contraceptive vaginal ring (NuvaRing) and hemostasis: a comparative study. Contraception 2004;69:461–7. http://dx.doi.org/10.1016/j.contraception.2003.12.010
20. Massai R, Mäkäräinen L, Kuukankorpi A, Klipping C, Duijkers I, Dieben T. The combined contraceptive vaginal ring and bone mineral density in healthy pre-menopausal women. Hum Reprod 2005;20:2764–8. http://dx.doi.org/10.1093/humrep/dei117
21. Milsom I, Lete I, Bjertnaes A, et al. Effects on cycle control and bodyweight of the combined contraceptive ring, NuvaRing, versus an oral contraceptive containing 30 μg ethinyl estradiol and 3 mg drospirenone. Hum Reprod 2006;21:2304–11. http://dx.doi.org/10.1093/humrep/del162
22. Oddsson K, Leifels-Fischer B, de Melo NR, et al. Efficacy and safety of a contraceptive vaginal ring (NuvaRing) compared with a combined oral contraceptive: a 1-year randomized trial. Contraception 2005;71:176–82. http://dx.doi.org/10.1016/j.contraception.2004.09.001
23. Pierson RA, Archer DF, Moreau M, Shangold GA, Fisher AC, Creasy GW. Ortho Evra/Evra versus oral contraceptives: follicular development and ovulation in normal cycles and after an intentional dosing error. Fertil Steril 2003;80:34–42. http://dx.doi.org/10.1016/S0015-0282(03)00556-9
24. Radowicki S, Skórzewska K, Szlendak K. Safety evaluation of a transdermal contraceptive system with an oral contraceptive [Polish]. Ginekol Pol 2005;76:884–9.

25. Sabatini R, Cagiano R. Comparison profiles of cycle control, side effects and sexual satisfaction of three hormonal contraceptives. Contraception 2006;74:220–3. http://dx.doi.org/10.1016/j.contraception.2006.03.022
26. Smallwood GH, Meador ML, Lenihan JP, Shangold GA, Fisher AC, Creasy GW; ORTHO EVRA/EVRA 002 Study Group. Efficacy and safety of a transdermal contraceptive system. Obstet Gynecol 2001;98:799–805.
27. Timmer CJ, Mulders TM. Pharmacokinetics of etonogestrel and ethinylestradiol released from a combined contraceptive vaginal ring. Clin Pharmacokinet 2000;39:233–42. http://dx.doi.org/10.2165/00003088-200039030-00005
28. Tuppurainen M, Klimscheffskij R, Venhola M, Dieben TO. The combined contraceptive vaginal ring (NuvaRing) and lipid metabolism: a comparative study. Contraception 2004;69:389–94. http://dx.doi.org/10.1016/j.contraception.2004.01.004
29. Urdl W, Apter D, Alperstein A, et al; ORTHO EVRA/EVRA 003 Study Group. Contraceptive efficacy, compliance and beyond: factors related to satisfaction with once-weekly transdermal compared with oral contraception. Eur J Obstet Gynecol Reprod Biol 2005;121:202–10. http://dx.doi.org/10.1016/j.ejogrb.2005.01.021
30. van den Heuvel MW, van Bragt AJM, Alnabawy AKM, Kaptein MCJ. Comparison of ethinylestradiol pharmacokinetics in three hormonal contraceptive formulations: the vaginal ring, the transdermal patch and an oral contraceptive. Contraception 2005;72:168–74. http://dx.doi.org/10.1016/j.contraception.2005.03.005
31. Veres S, Miller L, Burington B. A comparison between the vaginal ring and oral contraceptives. Obstet Gynecol 2004;104:555–63. http://dx.doi.org/10.1097/01.AOG.0000136082.59644.13
32. White T, Ozel B, Jain JK, Stanczyk FZ. Effects of transdermal and oral contraceptives on estrogen-sensitive hepatic proteins. Contraception 2006;74:293–6. http://dx.doi.org/10.1016/j.contraception.2006.04.005
33. Zieman M, Guillebaud J, Weisberg E, Shangold GA, Fisher AC, Creasy GW. Contraceptive efficacy and cycle control with the Ortho Evra/Evra transdermal system: the analysis of pooled data. Fertil Steril 2002;77(Suppl 2):S13–8. http://dx.doi.org/10.1016/S0015-0282(01)03275-7
34. Memon S, Iversen L, Hannaford PC. Is the oral contraceptive pill associated with fracture in later life? New evidence from the Royal College of General Practitioners Oral Contraception Study. Contraception 2011;84:40–7. http://dx.doi.org/10.1016/j.contraception.2010.11.019
35. Vestergaard P, Rejnmark L, Mosekilde L. Fracture risk in very young women using combined oral contraceptives. Contraception 2008;78:358–64. http://dx.doi.org/10.1016/j.contraception.2008.06.010
36. Vestergaard P, Rejnmark L, Mosekilde L. Oral contraceptive use and risk of fractures. Contraception 2006;73:571–6. http://dx.doi.org/10.1016/j.contraception.2006.01.006
37. Barad D, Kooperberg C, Wactawski-Wende J, Liu J, Hendrix SL, Watts NB. Prior oral contraception and postmenopausal fracture: a Women's Health Initiative observational cohort study. Fertil Steril 2005;84:374–83. http://dx.doi.org/10.1016/j.fertnstert.2005.01.132
38. Michaëlsson K, Baron JA, Farahmand BY, Ljunghall S. Influence of parity and lactation on hip fracture risk. Am J Epidemiol 2001;153:1166–72. http://dx.doi.org/10.1093/aje/153.12.1166
39. Michaëlsson K, Baron JA, Farahmand BY, Persson I, Ljunghall S. Oral-contraceptive use and risk of hip fracture: a case-control study. Lancet 1999;353:1481–4. http://dx.doi.org/10.1016/S0140-6736(98)09044-8
40. La Vecchia C, Tavani A, Gallus S. Oral contraceptives and risk of hip fractures. Lancet 1999;354:335–6. http://dx.doi.org/10.1016/S0140-6736(05)75239-9
41. Vessey M, Mant J, Painter R. Oral contraception and other factors in relation to hospital referral for fracture. Findings in a large cohort study. Contraception 1998;57:231–5. http://dx.doi.org/10.1016/S0010-7824(98)00026-2
42. O'Neill TW, Marsden D, Adams JE, Silman AJ. Risk factors, falls, and fracture of the distal forearm in Manchester, UK. J Epidemiol Community Health 1996;50:288–92. http://dx.doi.org/10.1136/jech.50.3.288
43. Mallmin H, Ljunghall S, Persson I, Bergström R. Risk factors for fractures of the distal forearm: a population-based case-control study. Osteoporos Int 1994;4:298–304. http://dx.doi.org/10.1007/BF01622186
44. Cooper C, Hannaford P, Croft P, Kay CR. Oral contraceptive pill use and fractures in women: a prospective study. Bone 1993;14:41–5. http://dx.doi.org/10.1016/8756-3282(93)90254-8
45. Meier C, Brauchli YB, Jick SS, Kraenzlin ME, Meier CR. Use of depot medroxyprogesterone acetate and fracture risk. J Clin Endocrinol Metab 2010;95:4909–16. http://dx.doi.org/10.1210/jc.2010-0032
46. Cibula D, Skrenkova J, Hill M, Stepan JJ. Low-dose estrogen combined oral contraceptives may negatively influence physiological bone mineral density acquisition during adolescence. Eur J Endocrinol 2012;166:1003–11. http://dx.doi.org/10.1530/EJE-11-1047
47. Gai L, Jia Y, Zhang M, et al. Effect of two kinds of different combined oral contraceptives use on bone mineral density in adolescent women. Contraception 2012;86:332–6. http://dx.doi.org/10.1016/j.contraception.2012.01.009
48. Scholes D, Hubbard RA, Ichikawa LE, et al. Oral contraceptive use and bone density change in adolescent and young adult women: a prospective study of age, hormone dose, and discontinuation. J Clin Endocrinol Metab 2011;96:E1380–7. http://dx.doi.org/10.1210/jc.2010-3027
49. Lattakova M, Borovsky M, Payer J, Killinger Z. Oral contraception usage in relation to bone mineral density and bone turnover in adolescent girls. Eur J Contracept Reprod Health Care 2009;14:207–14. http://dx.doi.org/10.1080/13625180902838828
50. Beksinska ME, Kleinschmidt I, Smit JA, Farley TM. Bone mineral density in a cohort of adolescents during use of norethisterone enanthate, depot-medroxyprogesterone acetate or combined oral contraceptives and after discontinuation of norethisterone enanthate. Contraception 2009;79:345–9. http://dx.doi.org/10.1016/j.contraception.2008.11.009
51. Pikkarainen E, Lehtonen-Veromaa M, Möttönen T, Kautiainen H, Viikari J. Estrogen-progestin contraceptive use during adolescence prevents bone mass acquisition: a 4-year follow-up study. Contraception 2008;78:226–31. http://dx.doi.org/10.1016/j.contraception.2008.05.002
52. Cromer BA, Bonny AE, Stager M, et al. Bone mineral density in adolescent females using injectable or oral contraceptives: a 24-month prospective study. Fertil Steril 2008;90:2060–7. http://dx.doi.org/10.1016/j.fertnstert.2007.10.070
53. Berenson AB, Rahman M, Breitkopf CR, Bi LX. Effects of depot medroxyprogesterone acetate and 20-μg oral contraceptives on bone mineral density. Obstet Gynecol 2008;112:788–99. http://dx.doi.org/10.1097/AOG.0b013e3181875b78
54. Harel Z, Riggs S, Vaz R, Flanagan P, Harel D, Machan JT. Bone accretion in adolescents using the combined estrogen and progestin transdermal contraceptive method Ortho Evra: a pilot study. J Pediatr Adolesc Gynecol 2010;23:23–31. http://dx.doi.org/10.1016/j.jpag.2009.04.008
55. Cobb KL, Bachrach LK, Sowers M, et al. The effect of oral contraceptives on bone mass and stress fractures in female runners. Med Sci Sports Exerc 2007;39:1464–73. http://dx.doi.org/10.1249/mss.0b013e318074e532
56. Beksinska ME, Kleinschmidt I, Smit JA, Farley TM. Bone mineral density in adolescents using norethisterone enanthate, depot-medroxyprogesterone acetate or combined oral contraceptives for contraception. Contraception 2007;75:438–43. http://dx.doi.org/10.1016/j.contraception.2007.02.001
57. Lara-Torre E, Edwards CP, Perlman S, Hertweck SP. Bone mineral density in adolescent females using depot medroxyprogesterone acetate. J Pediatr Adolesc Gynecol 2004;17:17–21. http://dx.doi.org/10.1016/j.jpag.2003.11.017
58. Cromer BA, Blair JM, Mahan JD, Zibners L, Naumovski Z. A prospective comparison of bone density in adolescent girls receiving depot medroxyprogesterone acetate (Depo-Provera), levonorgestrel (Norplant), or oral contraceptives. J Pediatr 1996;129:671–6. http://dx.doi.org/10.1016/S0022-3476(96)70148-8
59. Polatti F, Perotti F, Filippa N, Gallina D, Nappi RE. Bone mass and long-term monophasic oral contraceptive treatment in young women. Contraception 1995;51:221–4. http://dx.doi.org/10.1016/0010-7824(95)00036-A
60. Sørdal T, Grob P, Verhoeven C. Effects on bone mineral density of a monophasic combined oral contraceptive containing nomegestrol acetate/17ß-estradiol in comparison to levonorgestrel/ethinylestradiol. Acta Obstet Gynecol Scand 2012;91:1279–85. http://dx.doi.org/10.1111/j.1600-0412.2012.01498.x

61. Gargano V, Massaro M, Morra I, Formisano C, Di Carlo C, Nappi C. Effects of two low-dose combined oral contraceptives containing drospirenone on bone turnover and bone mineral density in young fertile women: a prospective controlled randomized study. Contraception 2008;78:10–5. http://dx.doi.org/10.1016/j.contraception.2008.01.016
62. Nappi C, Di Spiezio Sardo A, Greco E, Tommaselli GA, Giordano E, Guida M. Effects of an oral contraceptive containing drospirenone on bone turnover and bone mineral density. Obstet Gynecol 2005;105:53–60. http://dx.doi.org/10.1097/01.AOG.0000148344.26475.fc
63. Berenson AB, Radecki CM, Grady JJ, Rickert VI, Thomas A. A prospective, controlled study of the effects of hormonal contraception on bone mineral density. Obstet Gynecol 2001;98:576–82.
64. Berenson AB, Breitkopf CR, Grady JJ, Rickert VI, Thomas A. Effects of hormonal contraception on bone mineral density after 24 months of use. Obstet Gynecol 2004;103:899–906. http://dx.doi.org/10.1097/01.AOG.0000117082.49490.d5
65. Elgán C, Samsioe G, Dykes AK. Influence of smoking and oral contraceptives on bone mineral density and bone remodeling in young women: a 2-year study. Contraception 2003;67:439–47. http://dx.doi.org/10.1016/S0010-7824(03)00048-9
66. Elgán C, Dykes AK, Samsioe G. Bone mineral density changes in young women: a two year study. Gynecol Endocrinol 2004;19:169–77. http://dx.doi.org/10.1080/09513590400012119
67. Endrikat J, Mih E, Düsterberg B, et al. A 3-year double-blind, randomized, controlled study on the influence of two oral contraceptives containing either 20 μg or 30 μg ethinylestradiol in combination with levonorgestrel on bone mineral density. Contraception 2004;69:179–87. http://dx.doi.org/10.1016/j.contraception.2003.10.002
68. Paoletti AM, Orrù M, Lello S, et al. Short-term variations in bone remodeling markers of an oral contraception formulation containing 3 mg of drospirenone plus 30 microg of ethinyl estradiol: observational study in young postadolescent women. Contraception 2004;70:293–8. http://dx.doi.org/10.1016/j.contraception.2004.04.004
69. Nappi C, Di Spiezio Sardo A, Acunzo G, et al. Effects of a low-dose and ultra-low-dose combined oral contraceptive use on bone turnover and bone mineral density in young fertile women: a prospective controlled randomized study. Contraception 2003;67:355–9. http://dx.doi.org/10.1016/S0010-7824(03)00025-8
70. Reed SD, Scholes D, LaCroix AZ, Ichikawa LE, Barlow WE, Ott SM. Longitudinal changes in bone density in relation to oral contraceptive use. Contraception 2003;68:177–82. http://dx.doi.org/10.1016/S0010-7824(03)00147-1
71. Cobb KL, Kelsey JL, Sidney S, Ettinger B, Lewis CE. Oral contraceptives and bone mineral density in white and black women in CARDIA. Coronary Risk Development in Young Adults. Osteoporos Int 2002;13:893–900. http://dx.doi.org/10.1007/s001980200123
72. Burr DB, Yoshikawa T, Teegarden D, et al. Exercise and oral contraceptive use suppress the normal age-related increase in bone mass and strength of the femoral neck in women 18–31 years of age. Bone 2000;27:855–63. http://dx.doi.org/10.1016/S8756-3282(00)00403-8
73. Recker RR, Davies KM, Hinders SM, Heaney RP, Stegman MR, Kimmel DB. Bone gain in young adult women. JAMA 1992;268:2403–8. http://dx.doi.org/10.1001/jama.1992.03490170075028
74. Mazess RB, Barden HS. Bone density in premenopausal women: effects of age, dietary intake, physical activity, smoking, and birth-control pills. Am J Clin Nutr 1991;53:132–42.
75. Gambacciani M, Cappagli B, Lazzarini V, Ciaponi M, Fruzzetti F, Genazzani AR. Longitudinal evaluation of perimenopausal bone loss: effects of different low dose oral contraceptive preparations on bone mineral density. Maturitas 2006;54:176–80. http://dx.doi.org/10.1016/j.maturitas.2005.10.007
76. Gambacciani M, Spinetti A, Taponeco F, Cappagli B, Piaggesi L, Fioretti P. Longitudinal evaluation of perimenopausal vertebral bone loss: effects of a low-dose oral contraceptive preparation on bone mineral density and metabolism. Obstet Gynecol 1994;83:392–6.
77. Gambacciani M, Spinetti A, Cappagli B, et al. Hormone replacement therapy in perimenopausal women with a low dose oral contraceptive preparation: effects on bone mineral density and metabolism. Maturitas 1994;19:125–31. http://dx.doi.org/10.1016/0378-5122(94)90062-0
78. Gambacciani M, Cappagli B, Ciaponi M, Benussi C, Genazzani AR. Hormone replacement therapy in perimenopause: effect of a low dose oral contraceptive preparation on bone quantitative ultrasound characteristics. Menopause 1999;6:43–8. http://dx.doi.org/10.1097/00042192-199906010-00009
79. Volpe A, Malmusi S, Zanni AL, Landi S, Cagnacci A. Oral contraceptives and bone metabolism. Eur J Contracept Reprod Health Care 1997;2:225–8. http://dx.doi.org/10.3109/13625189709165298
80. Hansen MA, Overgaard K, Riis BJ, Christiansen C. Potential risk factors for development of postmenopausal osteoporosis—examined over a 12-year period. Osteoporos Int 1991;1:95–102. http://dx.doi.org/10.1007/BF01880450
81. Shargil AA. Hormone replacement therapy in perimenopausal women with a triphasic contraceptive compound: a three-year prospective study. Int J Fertil 1985;30:15, 18–28.
82. Taechakraichana N, Limpaphayom K, Ninlagarn T, Panyakhamlerd K, Chaikittisilpa S, Dusitsin N. A randomized trial of oral contraceptive and hormone replacement therapy on bone mineral density and coronary heart disease risk factors in postmenopausal women. Obstet Gynecol 2000;95:87–94.
83. Taechakraichana N, Jaisamrarn U, Panyakhamlerd K, Chaikittisilpa S, Limpaphayom K. Difference in bone acquisition among hormonally treated postmenopausal women with normal and low bone mass. J Med Assoc Thai 2001;84(Suppl 2):S586–92.
84. Grimes DA, Schulz KF. Surrogate end points in clinical research: hazardous to your health. Obstet Gynecol 2005;105:1114–8. http://dx.doi.org/10.1097/01.AOG.0000157445.67309.19
85. Schönau E. The peak bone mass concept: is it still relevant? Pediatr Nephrol 2004;19:825–31. http://dx.doi.org/10.1007/s00467-004-1465-5
86. Cohen A, Shane E. Treatment of premenopausal women with low bone mineral density. Curr Osteoporos Rep 2008;6:39–46. http://dx.doi.org/10.1007/s11914-008-0007-7
87. US Department of Health and Human Services. Healthy people 2020: maternal, infant, and child health objectives. Washington, DC: US Department of Health and Human Services; 2015. http://www.healthypeople.gov/2020/topics-objectives/topic/maternal-infant-and-child-health/objectives.
88. Tepper NK, Phillips SJ, Kapp N, Gaffield ME, Curtis KM. Combined hormonal contraceptive use among breastfeeding women: an updated systematic review. Contraception 2015;S0010-7824(15)00218-8.
89. Petersen JF, Bergholt T, Nielsen AK, Paidas MJ, Løkkegaard EC. Combined hormonal contraception and risk of venous thromboembolism within the first year following pregnancy. Danish nationwide historical cohort 1995–2009. Thromb Haemost 2014;112:73–8. http://dx.doi.org/10.1160/TH13-09-0797
90. Jackson E, Curtis KM, Gaffield ME. Risk of venous thromboembolism during the postpartum period: a systematic review. Obstet Gynecol 2011;117:691–703. http://dx.doi.org/10.1097/AOG.0b013e31820ce2db
91. Kamel H, Navi BB, Sriram N, Hovsepian DA, Devereux RB, Elkind MS. Risk of a thrombotic event after the 6-week postpartum period. N Engl J Med 2014;370:1307–15. http://dx.doi.org/10.1056/NEJMoa1311485
92. Sultan AA, Tata LJ, West J, et al. Risk factors for first venous thromboembolism around pregnancy: a population-based cohort study from the United Kingdom. Blood 2013;121:3953–61. http://dx.doi.org/10.1182/blood-2012-11-469551
93. Sultan AA, West J, Tata LJ, Fleming KM, Nelson-Piercy C, Grainge MJ. Risk of first venous thromboembolism in and around pregnancy: a population-based cohort study. Br J Haematol 2012;156:366–73. http://dx.doi.org/10.1111/j.1365-2141.2011.08956.x

94. Tepper NK, Boulet SL, Whiteman MK, et al. Postpartum venous thromboembolism: incidence and risk factors. Obstet Gynecol 2014;123:987–96. http://dx.doi.org/10.1097/AOG.0000000000000230
95. Jackson E, Glasier A. Return of ovulation and menses in postpartum nonlactating women: a systematic review. Obstet Gynecol 2011;117:657–62. http://dx.doi.org/10.1097/AOG.0b013e31820ce18c
96. Lähteenmäki P. Influence of oral contraceptives on immediate postabortal pituitary-ovarian function. Acta Obstet Gynecol Scand Suppl 1978;76:1–43.
97. Lähteenmäki P, Rasi V, Luukkainen T, Myllyä G. Coagulation factors in women using oral contraceptives or intrauterine contraceptive devices immediately after abortion. Am J Obstet Gynecol 1981;141:175–9.
98. Martin CW, Brown AH, Baird DT. A pilot study of the effect of methotrexate or combined oral contraceptive on bleeding patterns after induction of abortion with mifepristone and a prostaglandin pessary. Contraception 1998;58:99–103. http://dx.doi.org/10.1016/S0010-7824(98)00072-9
99. Niswonger JW, London GD, Anderson GV, Wolfe L. Oral contraceptives during immediate postabortal period. Obstet Gynecol 1968;32:325–7.
100. Peterson WF. Contraceptive therapy following therapeutic abortion: an analysis. Obstet Gynecol 1974;44:853–7.
101. Tang OS, Gao PP, Cheng L, Lee SW, Ho PC. A randomized double-blind placebo-controlled study to assess the effect of oral contraceptive pills on the outcome of medical abortion with mifepristone and misoprostol. Hum Reprod 1999;14:722–5. http://dx.doi.org/10.1093/humrep/14.3.722
102. Tang OS, Xu J, Cheng L, Lee SW, Ho PC. The effect of contraceptive pills on the measured blood loss in medical termination of pregnancy by mifepristone and misoprostol: a randomized placebo controlled trial. Hum Reprod 2002;17:99–102. http://dx.doi.org/10.1093/humrep/17.1.99
103. Fine PM, Tryggestad J, Meyers NJ, Sangi-Haghpeykar H. Safety and acceptability with the use of a contraceptive vaginal ring after surgical or medical abortion. Contraception 2007;75:367–71. http://dx.doi.org/10.1016/j.contraception.2007.01.009
104. Gillum LA, Mamidipudi SK, Johnston SC. Ischemic stroke risk with oral contraceptives: a meta-analysis. JAMA 2000;284:72–8. http://dx.doi.org/10.1001/jama.284.1.72
105. Jick SS, Walker AM, Stergachis A, Jick H. Oral contraceptives and breast cancer. Br J Cancer 1989;59:618–21. http://dx.doi.org/10.1038/bjc.1989.125
106. Khader YS, Rice J, John L, Abueita O. Oral contraceptives use and the risk of myocardial infarction: a meta-analysis. Contraception 2003;68:11–7. http://dx.doi.org/10.1016/S0010-7824(03)00073-8
107. Lawson DH, Davidson JF, Jick H. Oral contraceptive use and venous thromboembolism: absence of an effect of smoking. BMJ 1977;2:729–30. http://dx.doi.org/10.1136/bmj.2.6089.729
108. Lidegaard O, Edström B, Kreiner S. Oral contraceptives and venous thromboembolism. A case-control study. Contraception 1998;57:291–301. http://dx.doi.org/10.1016/S0010-7824(98)00033-X
109. Nightingale AL, Lawrenson RA, Simpson EL, Williams TJ, MacRae KD, Farmer RD. The effects of age, body mass index, smoking and general health on the risk of venous thromboembolism in users of combined oral contraceptives. Eur J Contracept Reprod Health Care 2000;5:265–74. http://dx.doi.org/10.1080/13625180008500402
110. Petitti DB, Wingerd J, Pellegrin F, Ramcharan S. Risk of vascular disease in women. Smoking, oral contraceptives, noncontraceptive estrogens, and other factors. JAMA 1979;242:1150–4. http://dx.doi.org/10.1001/jama.1979.03300110022020
111. Rosenberg L, Palmer JR, Rao RS, Shapiro S. Low-dose oral contraceptive use and the risk of myocardial infarction. Arch Intern Med 2001;161:1065–70. http://dx.doi.org/10.1001/archinte.161.8.1065
112. Straneva P, Hinderliter A, Wells E, Lenahan H, Girdler S. Smoking, oral contraceptives, and cardiovascular reactivity to stress. Obstet Gynecol 2000;95:78–83.
113. Tanis BC, van den Bosch MA, Kemmeren JM, et al. Oral contraceptives and the risk of myocardial infarction. N Engl J Med 2001;345:1787–93. http://dx.doi.org/10.1056/NEJMoa003216
114. Van den Bosch MA, Kemmeren JM, Tanis BC, et al. The RATIO study: oral contraceptives and the risk of peripheral arterial disease in young women. J Thromb Hemost 2003;1:439–44.
115. World Health Organization Collaborative Study of Cardiovascular Disease and Steroid Hormone Contraception. Venous thromboembolic disease and combined oral contraceptives: results of international multicentre case-control study. Lancet 1995;346:1575–82. http://dx.doi.org/10.1016/S0140-6736(95)91926-0
116. Collaborative Group for the Study of Stroke in Young Women. Oral contraceptives and stroke in young women. Associated risk factors. JAMA 1975;231:718–22. http://dx.doi.org/10.1001/jama.1975.03240190022010
117. Horton LG, Simmons KB, Curtis KM. Combined hormonal contraceptive use among obese women and risk for cardiovascular events: a systematic review. Contraception 2016. Epub June 1, 2016. http://dx.doi.org/10.1016/j.contraception.2016.05.014
118. Brunner Huber LR, Hogue CJ, Stein AD, Drews C, Zieman M. Body mass index and risk for oral contraceptive failure: a case-cohort study in South Carolina. Ann Epidemiol 2006;16:637–43. http://dx.doi.org/10.1016/j.annepidem.2006.01.001
119. Brunner Huber LR, Toth JL. Obesity and oral contraceptive failure: findings from the 2002 National Survey of Family Growth. Am J Epidemiol 2007;166:1306–11. http://dx.doi.org/10.1093/aje/kwm221
120. Brunner LR, Hogue CJ. The role of body weight in oral contraceptive failure: results from the 1995 national survey of family growth. Ann Epidemiol 2005;15:492–9. http://dx.doi.org/10.1016/j.annepidem.2004.10.009
121. Holt VL, Cushing-Haugen KL, Daling JR. Body weight and risk of oral contraceptive failure. Obstet Gynecol 2002;99:820–7.
122. Holt VL, Scholes D, Wicklund KG, Cushing-Haugen KL, Daling JR. Body mass index, weight, and oral contraceptive failure risk. Obstet Gynecol 2005;105:46–52. http://dx.doi.org/10.1097/01.AOG.0000149155.11912.52
123. Trussell J, Schwarz EB, Guthrie K. Obesity and oral contraceptive pill failure. Contraception 2009;79:334–8. http://dx.doi.org/10.1016/j.contraception.2008.11.017
124. Vessey M. Oral contraceptive failures and body weight: findings in a large cohort study. J Fam Plann Reprod Health Care 2001;27:90–1. http://dx.doi.org/10.1783/147118901101195092
125. Yamazaki M, Dwyer K, Sobhan M, et al. Effect of obesity on the effectiveness of hormonal contraceptives: an individual participant data meta-analysis. Contraception 2015;92:445–52. http://dx.doi.org/10.1016/j.contraception.2015.07.016
126. Zieman M, Guillebaud J, Weisberg E, Shangold GA, Fisher AC, Creasy GW. Contraceptive efficacy and cycle control with the Ortho Evra/Evra transdermal system: the analysis of pooled data. Fertil Steril 2002;77(Suppl 2):S13–8. http://dx.doi.org/10.1016/S0015-0282(01)03275-7
127. O'Connell KJ, Osborne LM, Westhoff C. Measured and reported weight change for women using a vaginal contraceptive ring vs. a low-dose oral contraceptive. Contraception 2005;72:323–7. http://dx.doi.org/10.1016/j.contraception.2005.05.008

128. Paulen ME, Zapata LB, Cansino C, Curtis KM, Jamieson DJ. Contraceptive use among women with a history of bariatric surgery: a systematic review. Contraception 2010;82:86–94. http://dx.doi.org/10.1016/j.contraception.2010.02.008
129. Lidegaard Ø, Edström B, Kreiner S. Oral contraceptives and venous thromboembolism: a five-year national case-control study. Contraception 2002;65:187–96. http://dx.doi.org/10.1016/S0010-7824(01)00307-9
130. Croft P, Hannaford PC. Risk factors for acute myocardial infarction in women: evidence from the Royal College of General Practitioners' oral contraception study. BMJ 1989;298:165–8. http://dx.doi.org/10.1136/bmj.298.6667.165
131. D'Avanzo B, La Vecchia C, Negri E, Parazzini F, Franceschi S. Oral contraceptive use and risk of myocardial infarction: an Italian case-control study. J Epidemiol Community Health 1994;48:324–5. http://dx.doi.org/10.1136/jech.48.3.324
132. Dunn NR, Faragher B, Thorogood M, et al. Risk of myocardial infarction in young female smokers. Heart 1999;82:581–3. http://dx.doi.org/10.1136/hrt.82.5.581
133. Hannaford PC, Croft PR, Kay CR. Oral contraception and stroke. Evidence from the Royal College of General Practitioners' Oral Contraception Study. Stroke 1994;25:935–42. http://dx.doi.org/10.1161/01.STR.25.5.935
134. Heinemann LA, Lewis MA, Spitzer WO, Thorogood M, Guggenmoos-Holzmann I, Bruppacher R; Transnational Research Group on Oral Contraceptives and the Health of Young Women. Thromboembolic stroke in young women. A European case-control study on oral contraceptives. Contraception 1998;57:29–37. http://dx.doi.org/10.1016/S0010-7824(97)00204-7
135. Kemmeren JM, Tanis BC, van den Bosch MA, et al. Risk of Arterial Thrombosis in Relation to Oral Contraceptives (RATIO) study: oral contraceptives and the risk of ischemic stroke. Stroke 2002;33:1202–8. http://dx.doi.org/10.1161/01.STR.0000015345.61324.3F
136. Lewis MA, Heinemann LA, Spitzer WO, MacRae KD, Bruppacher R. The use of oral contraceptives and the occurrence of acute myocardial infarction in young women. Results from the Transnational Study on Oral Contraceptives and the Health of Young Women. Contraception 1997;56:129–40. http://dx.doi.org/10.1016/S0010-7824(97)00118-2
137. Lidegaard O. Oral contraception and risk of a cerebral thromboembolic attack: results of a case-control study. BMJ 1993;306:956–63. http://dx.doi.org/10.1136/bmj.306.6883.956
138. Lidegaard O. Oral contraceptives, pregnancy and the risk of cerebral thromboembolism: the influence of diabetes, hypertension, migraine and previous thrombotic disease. Br J Obstet Gynaecol 1995;102:153–9. http://dx.doi.org/10.1111/j.1471-0528.1995.tb09070.x
139. Lubianca JN, Faccin CS, Fuchs FD. Oral contraceptives: a risk factor for uncontrolled blood pressure among hypertensive women. Contraception 2003;67:19–24. http://dx.doi.org/10.1016/S0010-7824(02)00429-8
140. Narkiewicz K, Graniero GR, D'Este D, Mattarei M, Zonzin P, Palatini P. Ambulatory blood pressure in mild hypertensive women taking oral contraceptives. A case-control study. Am J Hypertens 1995;8:249–53. http://dx.doi.org/10.1016/0895-7061(95)96212-3
141. Siritho S, Thrift AG, McNeil JJ, You RX, Davis SM, Donnan GA; Melbourne Risk Factor Study (MERFS) Group. Risk of ischemic stroke among users of the oral contraceptive pill: The Melbourne Risk Factor Study (MERFS) Group. Stroke 2003;34:1575–80. http://dx.doi.org/10.1161/01.STR.0000077925.16041.6B
142. WHO Collaborative Study of Cardiovascular Disease and Steroid Hormone Contraception. Ischaemic stroke and combined oral contraceptives: results of an international, multicentre, case-control study. Lancet 1996;348:498–505. http://dx.doi.org/10.1016/S0140-6736(95)12393-8
143. WHO Collaborative Study of Cardiovascular Disease and Steroid Hormone Contraception. Acute myocardial infarction and combined oral contraceptives: results of an international multicentre case-control study. Lancet 1997;349:1202–9. http://dx.doi.org/10.1016/S0140-6736(97)02358-1
144. Lubianca JN, Moreira LB, Gus M, Fuchs FD. Stopping oral contraceptives: an effective blood pressure-lowering intervention in women with hypertension. J Hum Hypertens 2005;19:451–5. http://dx.doi.org/10.1038/sj.jhh.1001841
145. Aberg H, Karlsson L, Melander S. Studies on toxaemia of pregnancy with special reference to blood pressure. II. Results after 6–11 years' follow-up. Ups J Med Sci 1978;83:97–102. http://dx.doi.org/10.3109/03009737809179119
146. Carmichael SM, Taylor MM, Ayers CR. Oral contraceptives, hypertension, and toxemia. Obstet Gynecol 1970;35:371–6.
147. Meinel H, Ihle R, Laschinski M. [Effect of hormonal contraceptives on blood pressure following pregnancy-induced hypertension]. Zentralbl Gynakol 1987;109:527–31.
148. Pritchard JA, Pritchard SA. Blood pressure response to estrogen-progestin oral contraceptive after pregnancy-induced hypertension. Am J Obstet Gynecol 1977;129:733–9 http://dx.doi.org/10.1016/0002-9378(77)90390-8.
149. Sibai BM, Taslimi MM, el-Nazer A, Amon E, Mabie BC, Ryan GM. Maternal-perinatal outcome associated with the syndrome of hemolysis, elevated liver enzymes, and low platelets in severe preeclampsia-eclampsia. Am J Obstet Gynecol 1986;155:501–7. http://dx.doi.org/10.1016/0002-9378(86)90266-8
150. Sibai BM, Ramadan MK, Chari RS, Friedman SA. Pregnancies complicated by HELLP syndrome (hemolysis, elevated liver enzymes, and low platelets): subsequent pregnancy outcome and long-term prognosis. Am J Obstet Gynecol 1995;172:125–9. http://dx.doi.org/10.1016/0002-9378(95)90099-3
151. WHO Collaborative Study of Cardiovascular Disease and Steroid Hormone Contraception. Haemorrhagic stroke, overall stroke risk, and combined oral contraceptives: results of an international, multicentre, case-control study. Lancet 1996;348:505–10. http://dx.doi.org/10.1016/S0140-6736(95)12394-6
152. Sidney S, Petitti DB, Soff GA, Cundiff DL, Tolan KK, Quesenberry CP Jr. Venous thromboembolic disease in users of low-estrogen combined estrogen-progestin oral contraceptives. Contraception 2004;70:3–10. http://dx.doi.org/10.1016/j.contraception.2004.02.010
153. Andersen BS, Olsen J, Nielsen GL, et al. Third generation oral contraceptives and heritable thrombophilia as risk factors of non-fatal venous thromboembolism. Thromb Haemost 1998;79:28–31.
154. Aznar J, Mira Y, Vayá A, et al. Factor V Leiden and prothrombin G20210A mutations in young adults with cryptogenic ischemic stroke. Thromb Haemost 2004;91:1031–4.
155. Bennet L, Odeberg H. Resistance to activated protein C, highly prevalent amongst users of oral contraceptives with venous thromboembolism. J Intern Med 1998;243:27–32. http://dx.doi.org/10.1046/j.1365-2796.1998.00310.x
156. Bloemenkamp KW, Rosendaal FR, Helmerhorst FM, Vandenbroucke JP. Higher risk of venous thrombosis during early use of oral contraceptives in women with inherited clotting defects.[comment]. Arch Intern Med 2000;160:49–52. http://dx.doi.org/10.1001/archinte.160.1.49

157. Bloemenkamp KW, Helmerhorst FM, Rosendaal FR, Vandenbroucke JP, Büller HR. Enhancement by factor V Leiden mutation of risk of deep-vein thrombosis associated with oral contraceptives containing a third-generation progestagen. Lancet 1995;346:1593–6. http://dx.doi.org/10.1016/S0140-6736(95)91929-5
158. de Bruijn SF, Stam J, Koopman MM, Vandenbroucke JP; The Cerebral Venous Sinus Thrombosis Study Group. Case-control study of risk of cerebral sinus thrombosis in oral contraceptive users and in [correction of who are] carriers of hereditary prothrombotic conditions. BMJ 1998;316:589–92. http://dx.doi.org/10.1136/bmj.316.7131.589
159. Emmerich J, Rosendaal FR, Cattaneo M, et al; Study Group for Pooled-Analysis in Venous Thromboembolism. Combined effect of factor V Leiden and prothrombin 20210A on the risk of venous thromboembolism—pooled analysis of 8 case-control studies including 2310 cases and 3204 controls. Thromb Haemost 2001;86:809–16.
160. Gadelha T, André C, Jucá AA, Nucci M. Prothrombin 20210A and oral contraceptive use as risk factors for cerebral venous thrombosis. Cerebrovasc Dis 2005;19:49–52. http://dx.doi.org/10.1159/000081911
161. Legnani C, Palareti G, Guazzaloca G, et al. Venous thromboembolism in young women; role of thrombophilic mutations and oral contraceptive use. Eur Heart J 2002;23:984–90. http://dx.doi.org/10.1053/euhj.2001.3082
162. Martinelli I, Battaglioli T, Bucciarelli P, Passamonti SM, Mannucci PM. Risk factors and recurrence rate of primary deep vein thrombosis of the upper extremities. Circulation 2004;110:566–70. http://dx.doi.org/10.1161/01.CIR.0000137123.55051.9B
163. Martinelli I, Sacchi E, Landi G, Taioli E, Duca F, Mannucci PM. High risk of cerebral-vein thrombosis in carriers of a prothrombin-gene mutation and in users of oral contraceptives.[comment]. N Engl J Med 1998;338:1793–7. http://dx.doi.org/10.1056/NEJM199806183382502
164. Martinelli I, Taioli E, Bucciarelli P, Akhavan S, Mannucci PM. Interaction between the G20210A mutation of the prothrombin gene and oral contraceptive use in deep vein thrombosis. Arterioscler Thromb Vasc Biol 1999;19:700–3. http://dx.doi.org/10.1161/01.ATV.19.3.700
165. Middeldorp S, Meinardi JR, Koopman MM, et al. A prospective study of asymptomatic carriers of the factor V Leiden mutation to determine the incidence of venous thromboembolism. [comment]. Ann Intern Med 2001;135:322–7. http://dx.doi.org/10.7326/0003-4819-135-5-200109040-00008
166. Pabinger I, Schneider B; The GTH Study Group on Natural Inhibitors. Thrombotic risk of women with hereditary antithrombin III-, protein C- and protein S-deficiency taking oral contraceptive medication. Thromb Haemost 1994;71:548–52.
167. Pezzini A, Grassi M, Iacoviello L, et al. Inherited thrombophilia and stratification of ischaemic stroke risk among users of oral contraceptives. J Neurol Neurosurg Psychiatry 2007;78:271–6. http://dx.doi.org/10.1136/jnnp.2006.101675
168. Santamaría A, Mateo J, Oliver A, et al. Risk of thrombosis associated with oral contraceptives of women from 97 families with inherited thrombophilia: high risk of thrombosis in carriers of the G20210A mutation of the prothrombin gene. Haematologica 2001;86:965–71.
169. Slooter AJ, Rosendaal FR, Tanis BC, Kemmeren JM, van der Graaf Y, Algra A. Prothrombotic conditions, oral contraceptives, and the risk of ischemic stroke. J Thromb Haemost 2005;3:1213–7. http://dx.doi.org/10.1111/j.1538-7836.2005.01442.x
170. Spannagl M, Heinemann LA, Schramm W. Are factor V Leiden carriers who use oral contraceptives at extreme risk for venous thromboembolism? Eur J Contracept Reprod Health Care 2000;5:105–12. http://dx.doi.org/10.1080/13625180008500383
171. van Boven HH, Vandenbroucke JP, Briët E, Rosendaal FR. Gene-gene and gene-environment interactions determine risk of thrombosis in families with inherited antithrombin deficiency. Blood 1999;94:2590–4.
172. van Vlijmen EF, Brouwer JL, Veeger NJ, Eskes TK, de Graeff PA, van der Meer J. Oral contraceptives and the absolute risk of venous thromboembolism in women with single or multiple thrombophilic defects: results from a retrospective family cohort study. Arch Intern Med 2007;167:282–9. http://dx.doi.org/10.1001/archinte.167.3.282
173. Vandenbroucke JP, Koster T, Rosendaal FR, Briët E, Reitsma PH, Bertina RM. Increased risk of venous thrombosis in oral-contraceptive users who are carriers of factor V Leiden mutation.[comment]. Lancet 1994;344:1453–7. http://dx.doi.org/10.1016/S0140-6736(94)90286-0
174. Vayá A, Mira Y, Mateo J, et al. Prothrombin G20210A mutation and oral contraceptive use increase upper-extremity deep vein thrombotic risk. Thromb Haemost 2003;89:452–7.
175. Martinelli I, Battaglioli T, Burgo I, Di Domenico S, Mannucci PM. Oral contraceptive use, thrombophilia and their interaction in young women with ischemic stroke. Haematologica 2006;91:844–7.
176. Tepper NK, Marchbanks PA, Curtis KM. Superficial venous disease and combined hormonal contraceptives: a systematic review. Contraception 2015;S0010-7824(15)00128-6.
177. Tepper NK, Paulen ME, Marchbanks PA, Curtis KM. Safety of contraceptive use among women with peripartum cardiomyopathy: a systematic review. Contraception 2010;82:95–101. http://dx.doi.org/10.1016/j.contraception.2010.02.004
178. The Criteria Committee of the New York Heart Association. Nomenclature and criteria for diagnosis of diseases of the heart and great vessels. 9th ed. Boston, MA: Little, Brown and Co; 1994.
179. Bernatsky S, Clarke A, Ramsey-Goldman R, et al. Hormonal exposures and breast cancer in a sample of women with systemic lupus erythematosus. Rheumatology (Oxford) 2004;43:1178–81. http://dx.doi.org/10.1093/rheumatology/keh282
180. Bernatsky S, Ramsey-Goldman R, Gordon C, et al. Factors associated with abnormal Pap results in systemic lupus erythematosus. Rheumatology (Oxford) 2004;43:1386–9. http://dx.doi.org/10.1093/rheumatology/keh331
181. Chopra N, Koren S, Greer WL, et al. Factor V Leiden, prothrombin gene mutation, and thrombosis risk in patients with antiphospholipid antibodies. J Rheumatol 2002;29:1683–8.
182. Esdaile JM, Abrahamowicz M, Grodzicky T, et al. Traditional Framingham risk factors fail to fully account for accelerated atherosclerosis in systemic lupus erythematosus. Arthritis Rheum 2001;44:2331–7. http://dx.doi.org/10.1002/1529-0131(200110)44:10<2331::AID-ART395≥3.0.CO;2-I
183. Julkunen HA. Oral contraceptives in systemic lupus erythematosus: side-effects and influence on the activity of SLE. Scand J Rheumatol 1991;20:427–33. http://dx.doi.org/10.3109/03009749109096822
184. Julkunen HA, Kaaja R, Friman C. Contraceptive practice in women with systemic lupus erythematosus. Br J Rheumatol 1993;32:227–30. http://dx.doi.org/10.1093/rheumatology/32.3.227
185. Jungers P, Dougados M, Pélissier C, et al. Influence of oral contraceptive therapy on the activity of systemic lupus erythematosus. Arthritis Rheum 1982;25:618–23. http://dx.doi.org/10.1002/art.1780250603
186. Manzi S, Meilahn EN, Rairie JE, et al. Age-specific incidence rates of myocardial infarction and angina in women with systemic lupus erythematosus: comparison with the Framingham Study. Am J Epidemiol 1997;145:408–15. http://dx.doi.org/10.1093/oxfordjournals.aje.a009122

187. McAlindon T, Giannotta L, Taub N, D'Cruz D, Hughes G. Environmental factors predicting nephritis in systemic lupus erythematosus. Ann Rheum Dis 1993;52:720–4. http://dx.doi.org/10.1136/ard.52.10.720
188. McDonald J, Stewart J, Urowitz MB, Gladman DD. Peripheral vascular disease in patients with systemic lupus erythematosus. Ann Rheum Dis 1992;51:56–60. http://dx.doi.org/10.1136/ard.51.1.56
189. Mintz G, Gutiérrez G, Delezé M, Rodríguez E. Contraception with progestagens in systemic lupus erythematosus. Contraception 1984;30:29–38. http://dx.doi.org/10.1016/0010-7824(84)90076-3
190. Petri M. Musculoskeletal complications of systemic lupus erythematosus in the Hopkins Lupus Cohort: an update. Arthritis Care Res 1995;8:137–45. http://dx.doi.org/10.1002/art.1790080305
191. Petri M. Lupus in Baltimore: evidence-based 'clinical pearls' from the Hopkins Lupus Cohort. Lupus 2005;14:970–3. http://dx.doi.org/10.1191/0961203305lu2230xx
192. Petri M, Kim MY, Kalunian KC, et al; OC-SELENA Trial. Combined oral contraceptives in women with systemic lupus erythematosus. N Engl J Med 2005;353:2550–8. http://dx.doi.org/10.1056/NEJMoa051135
193. Sánchez-Guerrero J, Uribe AG, Jiménez-Santana L, et al. A trial of contraceptive methods in women with systemic lupus erythematosus. N Engl J Med 2005;353:2539–49. http://dx.doi.org/10.1056/NEJMoa050817
194. Sarabi ZS, Chang E, Bobba R, et al. Incidence rates of arterial and venous thrombosis after diagnosis of systemic lupus erythematosus. Arthritis Rheum 2005;53:609–12. http://dx.doi.org/10.1002/art.21314
195. Schaedel ZE, Dolan G, Powell MC. The use of the levonorgestrel-releasing intrauterine system in the management of menorrhagia in women with hemostatic disorders. Am J Obstet Gynecol 2005;193:1361–3. http://dx.doi.org/10.1016/j.ajog.2005.05.002
196. Somers E, Magder LS, Petri M. Antiphospholipid antibodies and incidence of venous thrombosis in a cohort of patients with systemic lupus erythematosus. J Rheumatol 2002;29:2531–6.
197. Urowitz MB, Bookman AA, Koehler BE, Gordon DA, Smythe HA, Ogryzlo MA. The bimodal mortality pattern of systemic lupus erythematosus. Am J Med 1976;60:221–5. http://dx.doi.org/10.1016/0002-9343(76)90431-9
198. Choojitarom K, Verasertniyom O, Totemchokchyakarn K, Nantiruj K, Sumethkul V, Janwityanujit S. Lupus nephritis and Raynaud's phenomenon are significant risk factors for vascular thrombosis in SLE patients with positive antiphospholipid antibodies. Clin Rheumatol 2008;27:345–51. http://dx.doi.org/10.1007/s10067-007-0721-z
199. Wahl DG, Guillemin F, de Maistre E, Perret C, Lecompte T, Thibaut G. Risk for venous thrombosis related to antiphospholipid antibodies in systemic lupus erythematosus—a meta-analysis. Lupus 1997;6:467–73. http://dx.doi.org/10.1177/096120339700600510
200. Farr SL, Folger SG, Paulen ME, Curtis KM. Safety of contraceptive methods for women with rheumatoid arthritis: a systematic review. Contraception 2010;82:64–71. http://dx.doi.org/10.1016/j.contraception.2010.02.003
201. Tepper NK, Whiteman MK, Zapata LB, Marchbanks PA, Curtis KM. Safety of hormonal contraceptives among women with migraine: a systematic review. Contraception 2016. Epub May 3, 2016. http:// dx.doi.org/10.1016/j.contraception.2016.04.016
202. Xu Z, Li Y, Tang S, Huang X, Chen T. Current use of oral contraceptives and the risk of first-ever ischemic stroke: A meta-analysis of observational studies. Thromb Res 2015;136:52–60. http://dx.doi.org/10.1016/j.thromres.2015.04.021
203. Etminan M, Takkouche B, Isorna FC, Samii A. Risk of ischaemic stroke in people with migraine: systematic review and meta-analysis of observational studies. BMJ 2005;330:63. http://dx.doi.org/10.1136/bmj.38302.504063.8F
204. Schürks M, Rist PM, Bigal ME, Buring JE, Lipton RB, Kurth T. Migraine and cardiovascular disease: systematic review and meta-analysis. BMJ 2009;339:b3914. http://dx.doi.org/10.1136/bmj.b3914
205. Spector JT, Kahn SR, Jones MR, Jayakumar M, Dalal D, Nazarian S. Migraine headache and ischemic stroke risk: an updated meta-analysis. Am J Med 2010;123:612–24. http://dx.doi.org/10.1016/j.amjmed.2009.12.021
206. Zapata LB, Oduyebo T, Whiteman MK, Marchbanks PA, Curtis KM. Contraceptive use among women with multiple sclerosis: a systematic review. Contraception. In press 2016.
207. Pagano HP, Zapata LB, Berry-Bibee EN, Nanda K, Curtis KM. Safety of hormonal contraception and intrauterine devices among women with depressive and bipolar disorders: a systematic review. Contraception 2016. Epub June 27, 2016. http://dx.doi.org/10.1016/j.contraception.2016.06.012
208. Iyer V, Farquhar C, Jepson R. Oral contraceptive pills for heavy menstrual bleeding. Cochrane Database Syst Rev 2000;(2):CD000154.
209. Davis L, Kennedy SS, Moore J, Prentice A. Oral contraceptives for pain associated with endometriosis. Cochrane Database Syst Rev 2007;(3):CD001019.
210. Hendrix SL, Alexander NJ. Primary dysmenorrhea treatment with a desogestrel-containing low-dose oral contraceptive. Contraception 2002;66:393–9. http://dx.doi.org/10.1016/S0010-7824(02)00414-6
211. Proctor ML, Roberts H, Farquhar CM. Combined oral contraceptive pill (OCP) as treatment for primary dysmenorrhoea. Cochrane Database Syst Rev 2001;(4):CD002120.
212. Gaffield ME, Kapp N, Curtis KM. Combined oral contraceptive and intrauterine device use among women with gestational trophoblastic disease. Contraception 2009;80:363–71. http://dx.doi.org/10.1016/j.contraception.2009.03.022
213. Smith JS, Green J, Berrington de Gonzalez A, et al. Cervical cancer and use of hormonal contraceptives: a systematic review. Lancet 2003;361:1159–67. http://dx.doi.org/10.1016/S0140-6736(03)12949-2
214. Black MM, Barclay THC, Polednak A, Kwon CS, Leis HP Jr, Pilnik S. Family history, oral contraceptive usage, and breast cancer. Cancer 1983;51:2147–51. http://dx.doi.org/10.1002/1097-0142(19830601)51:11<2147::AID-CNCR2820511133≥3.0.CO;2-X
215. Brinton LA, Hoover R, Szklo M, Fraumeni JF Jr. Oral contraceptives and breast cancer. Int J Epidemiol 1982;11:316–22. http://dx.doi.org/10.1093/ije/11.4.316
216. Brohet RM, Goldgar DE, Easton DF, et al. Oral contraceptives and breast cancer risk in the international BRCA1/2 carrier cohort study: a report from EMBRACE, GENEPSO, GEO-HEBON, and the IBCCS Collaborating Group. J Clin Oncol 2007;25:3831–6. http://dx.doi.org/10.1200/JCO.2007.11.1179
217. Claus EB, Stowe M, Carter D. Oral contraceptives and the risk of ductal breast carcinoma in situ. Breast Cancer Res Treat 2003;81:129–36. http://dx.doi.org/10.1023/A:1025728524310
218. Collaborative Group on Hormonal Factors in Breast Cancer. Familial breast cancer: collaborative reanalysis of individual data from 52 epidemiological studies including 58,209 women with breast cancer and 101,986 women without the disease. Lancet 2001;358:1389–99. http://dx.doi.org/10.1016/S0140-6736(01)06524-2
219. Grabrick DM, Hartmann LC, Cerhan JR, et al. Risk of breast cancer with oral contraceptive use in women with a family history of breast cancer.[comment]. JAMA 2000;284:1791–8. http://dx.doi.org/10.1001/jama.284.14.1791
220. Gronwald J, Byrski T, Huzarski T, et al. Influence of selected lifestyle factors on breast and ovarian cancer risk in BRCA1 mutation carriers from Poland. Breast Cancer Res Treat 2006;95:105–9. http://dx.doi.org/10.1007/s10549-005-9051-5

221. Haile RW, Thomas DC, McGuire V, et al; kConFab Investigators; Ontario Cancer Genetics Network Investigators. BRCA1 and BRCA2 mutation carriers, oral contraceptive use, and breast cancer before age 50. Cancer Epidemiol Biomarkers Prev 2006;15:1863–70. http://dx.doi.org/10.1158/1055-9965.EPI-06-0258
222. Harris NV, Weiss NS, Francis AM, Polissar L. Breast cancer in relation to patterns of oral contraceptive use. Am J Epidemiol 1982;116:643–51.
223. Hennekens CH, Speizer FE, Lipnick RJ, et al. A case-control study of oral contraceptive use and breast cancer. J Natl Cancer Inst 1984;72:39–42.
224. Jernström H, Loman N, Johannsson OT, Borg A, Olsson H. Impact of teenage oral contraceptive use in a population-based series of early-onset breast cancer cases who have undergone BRCA mutation testing. Eur J Cancer 2005;41:2312–20. http://dx.doi.org/10.1016/j.ejca.2005.03.035
225. Marchbanks PA, McDonald JA, Wilson HG, et al. Oral contraceptives and the risk of breast cancer. N Engl J Med 2002;346:2025–32. http://dx.doi.org/10.1056/NEJMoa013202
226. Milne RL, Knight JA, John EM, et al. Oral contraceptive use and risk of early-onset breast cancer in carriers and noncarriers of BRCA1 and BRCA2 mutations. Cancer Epidemiol Biomarkers Prev 2005;14:350–6. http://dx.doi.org/10.1158/1055-9965.EPI-04-0376
227. Narod SA, Dubé MP, Klijn J, et al. Oral contraceptives and the risk of breast cancer in BRCA1 and BRCA2 mutation carriers. J Natl Cancer Inst 2002;94:1773–9. http://dx.doi.org/10.1093/jnci/94.23.1773
228. Rosenberg L, Palmer JR, Rao RS, et al. Case-control study of oral contraceptive use and risk of breast cancer. Am J Epidemiol 1996;143:25–37. http://dx.doi.org/10.1093/oxfordjournals.aje.a008654
229. Silvera SA, Miller AB, Rohan TE. Oral contraceptive use and risk of breast cancer among women with a family history of breast cancer: a prospective cohort study. Cancer Causes Control 2005;16:1059–63. http://dx.doi.org/10.1007/s10552-005-0343-1
230. Ursin G, Henderson BE, Haile RW, et al. Does oral contraceptive use increase the risk of breast cancer in women with BRCA1/BRCA2 mutations more than in other women? Cancer Res 1997;57:3678–81.
231. Ursin G, Ross RK, Sullivan-Halley J, Hanisch R, Henderson B, Bernstein L. Use of oral contraceptives and risk of breast cancer in young women. Breast Cancer Res Treat 1998;50:175–84. http://dx.doi.org/10.1023/A:1006037823178
232. Polis CB, Phillips SJ, Curtis KM, et al. Hormonal contraceptive methods and risk of HIV acquisition in women: a systematic review of epidemiological evidence. Contraception 2014;90:360–90. http://dx.doi.org/10.1016/j.contraception.2014.07.009
233. Polis CB, Phillips SJ, Curtis KM. Hormonal contraceptive use and female-to-male HIV transmission: a systematic review of the epidemiologic evidence. AIDS 2013;27:493–505. http://dx.doi.org/10.1097/QAD.0b013e32835ad539
234. Phillips SJ, Polis CB, Curtis KM. The safety of hormonal contraceptives for women living with HIV and their sexual partners. Contraception 2016;93:11–6 http://dx.doi.org/10.1016/j.contraception.2015.10.002.
235. Phillips SJ, Curtis KM, Polis CB. Effect of hormonal contraceptive methods on HIV disease progression: a systematic review. AIDS 2013;27:787–94. http://dx.doi.org/10.1097/QAD.0b013e32835bb672
236. el-Raghy I, Back DJ, Osman F, Orme ML, Fathalla M. Contraceptive steroid concentrations in women with early active schistosomiasis: lack of effect of antischistosomal drugs. Contraception 1986;33:373–7. http://dx.doi.org/10.1016/0010-7824(86)90099-5
237. Gad-el-Mawla N, Abdallah A. Liver function in bilharzial females receiving contraceptive pills. Acta Hepatosplenol 1969;16:308–10.
238. Gad-el-Mawla N, el-Roubi O, Sabet S, Abdallah A. Plasma lipids and lipoproteins in bilharzial females during oral contraceptive therapy. J Egypt Med Assoc 1972;55:137–47.
239. Shaaban MM, Hammad WA, Falthalla MF, et al. Effects of oral contraception on liver function tests and serum proteins in women with active schistosomiasis. Contraception 1982;26:75–82. http://dx.doi.org/10.1016/0010-7824(82)90174-3
240. Shaaban MM, Ghaneimah SA, Mohamed MA, Abdel-Chani S, Mostafa SA. Effect of oral contraception on serum bile acid. Int J Gynaecol Obstet 1984;22:111–5. http://dx.doi.org/10.1016/0020-7292(84)90023-7
241. Sy FS, Osteria TS, Opiniano V, Gler S. Effect of oral contraceptive on liver function tests of women with schistosomiasis in the Philippines. Contraception 1986;34:283–94. http://dx.doi.org/10.1016/0010-7824(86)90009-0
242. Tagy AH, Saker ME, Moussa AA, Kolgah A. The effect of low-dose combined oral contraceptive pills versus injectable contraceptive (Depot Provera) on liver function tests of women with compensated bilharzial liver fibrosis. Contraception 2001;64:173–6. http://dx.doi.org/10.1016/S0010-7824(01)00248-7
243. Beck P, Wells SA. Comparison of the mechanisms underlying carbohydrate intolerance in subclinical diabetic women during pregnancy and during post-partum oral contraceptive steroid treatment. J Clin Endocrinol Metab 1969;29:807–18. http://dx.doi.org/10.1210/jcem-29-6-807
244. Kjos SL, Peters RK, Xiang A, Thomas D, Schaefer U, Buchanan TA. Contraception and the risk of type 2 diabetes mellitus in Latina women with prior gestational diabetes mellitus. JAMA 1998;280:533–8. http://dx.doi.org/10.1001/jama.280.6.533
245. Kung AW, Ma JT, Wong VC, et al. Glucose and lipid metabolism with triphasic oral contraceptives in women with history of gestational diabetes. Contraception 1987;35:257–69. http://dx.doi.org/10.1016/0010-7824(87)90027-8
246. Rådberg T, Gustafson A, Skryten A, Karlsson K. Metabolic studies in gestational diabetic women during contraceptive treatment: effects on glucose tolerance and fatty acid composition of serum lipids. Gynecol Obstet Invest 1982;13:17–29. http://dx.doi.org/10.1159/000299480
247. Skouby SO, Andersen O, Kühl C. Oral contraceptives and insulin receptor binding in normal women and those with previous gestational diabetes. Am J Obstet Gynecol 1986;155:802–7. http://dx.doi.org/10.1016/S0002-9378(86)80024-2
248. Skouby SO, Andersen O, Saurbrey N, Kühl C. Oral contraception and insulin sensitivity: in vivo assessment in normal women and women with previous gestational diabetes. J Clin Endocrinol Metab 1987;64:519–23. http://dx.doi.org/10.1210/jcem-64-3-519
249. Skouby SO, Mølsted-Pedersen L, Kühl C. Low dosage oral contraception in women with previous gestational diabetes. Obstet Gynecol 1982;59:325–8.
250. Xiang AH, Kawakubo M, Kjos SL, Buchanan TA. Long-acting injectable progestin contraception and risk of type 2 diabetes in Latino women with prior gestational diabetes mellitus. Diabetes Care 2006;29:613–7. http://dx.doi.org/10.2337/diacare.29.03.06.dc05-1940
251. Kjos SL, Shoupe D, Douyan S, et al. Effect of low-dose oral contraceptives on carbohydrate and lipid metabolism in women with recent gestational diabetes: results of a controlled, randomized, prospective study. Am J Obstet Gynecol 1990;163:1822–7. http://dx.doi.org/10.1016/0002-9378(90)90757-X
252. Rådberg T, Gustafson A, Skryten A, Karlsson K. Metabolic studies in women with previous gestational diabetes during contraceptive treatment: effects on serum lipids and high density lipoproteins. Acta Endocrinol (Copenh) 1982;101:134–9.

253. Skouby SO, Kühl C, Mølsted-Pedersen L, Petersen K, Christensen MS. Triphasic oral contraception: metabolic effects in normal women and those with previous gestational diabetes. Am J Obstet Gynecol 1985;153:495–500. http://dx.doi.org/10.1016/0002-9378(85)90460-0
254. Beck P, Arnett DM, Alsever RN, Eaton RP. Effect of contraceptive steroids on arginine- stimulated glucagon and insulin secretion in women. II. Carbohydrate and lipid physiology in insulin-dependent diabetics. Metabolism 1976;25:23–31 http://dx.doi.org/10.1016/0026-0495(76)90156-6.
255. Diab KM, Zaki MM. Contraception in diabetic women: comparative metabolic study of Norplant, depot medroxyprogesterone acetate, low dose oral contraceptive pill and CuT380A. J Obstet Gynaecol Res 2000;26:17–26. http://dx.doi.org/10.1111/j.1447-0756.2000.tb01195.x
256. Garg SK, Chase HP, Marshall G, Hoops SL, Holmes DL, Jackson WE. Oral contraceptives and renal and retinal complications in young women with insulin-dependent diabetes mellitus. JAMA 1994;271:1099–102. http://dx.doi.org/10.1001/jama.1994.03510380055037
257. Grigoryan OR, Grodnitskaya EE, Andreeva EN, Shestakova MV, Melnichenko GA, Dedov II. Contraception in perimenopausal women with diabetes mellitus. Gynecol Endocrinol 2006;22:198–206. http://dx.doi.org/10.1080/09513590600624317
258. Margolis KL, Adami H-O, Luo J, Ye W, Weiderpass E. A prospective study of oral contraceptive use and risk of myocardial infarction among Swedish women. Fertil Steril 2007;88:310–6. http://dx.doi.org/10.1016/j.fertnstert.2006.11.206
259. Petersen KR, Skouby SO, Jespersen J. Balance of coagulation activity with fibrinolysis during use of oral contraceptives in women with insulin-dependent diabetes mellitus. Int J Fertil Menopausal Stud 1995;40(Suppl 2):105–11.
260. Petersen KR, Skouby SO, Sidelmann J, Jespersen J. Assessment of endothelial function during oral contraception in women with insulin-dependent diabetes mellitus. Metabolism 1994;43:1379–83. http://dx.doi.org/10.1016/0026-0495(94)90031-0
261. Rådberg T, Gustafson A, Skryten A, Karlsson K. Oral contraception in diabetic women. A cross-over study on serum and high density lipoprotein (HDL) lipids and diabetes control during progestogen and combined estrogen/progestogen contraception. Horm Metab Res 1982;14:61–5.
262. Skouby SO, Jensen BM, Kühl C, Mølsted-Pedersen L, Svenstrup B, Nielsen J. Hormonal contraception in diabetic women: acceptability and influence on diabetes control and ovarian function of a nonalkylated estrogen/progestogen compound. Contraception 1985;32:23–31. http://dx.doi.org/10.1016/0010-7824(85)90113-1
263. Skouby SO, Mølsted-Pedersen L, Kühl C, Bennet P. Oral contraceptives in diabetic women: metabolic effects of four compounds with different estrogen/progestogen profiles. Fertil Steril 1986;46:858–64 http://dx.doi.org/10.1016/S0015-0282(16)49825-0.
264. Zapata LB, Paulen ME, Cansino C, Marchbanks PA, Curtis KM. Contraceptive use among women with inflammatory bowel disease: A systematic review. Contraception 2010;82:72–85. http://dx.doi.org/10.1016/j.contraception.2010.02.012
265. Kapp N, Tilley IB, Curtis KM. The effects of hormonal contraceptive use among women with viral hepatitis or cirrhosis of the liver: a systematic review. Contraception 2009;80:381–6. http://dx.doi.org/10.1016/j.contraception.2009.04.007
266. Kapp N, Curtis KM. Hormonal contraceptive use among women with liver tumors: a systematic review. Contraception 2009;80:387–90. http://dx.doi.org/10.1016/j.contraception.2009.01.021
267. Whiteman MK, Oduyebo T, Zapata LB, Walker S, Curtis KM. Contraceptive safety among women with cystic fibrosis: a systematic review. Contraception 2016. Epub June 7, 2016. http://dx.doi.org/10.1016/j.contraception.2016.05.016
268. Paulen ME, Folger SG, Curtis KM, Jamieson DJ. Contraceptive use among solid organ transplant patients: a systematic review. Contraception 2010;82:102–12. http://dx.doi.org/10.1016/j.contraception.2010.02.007
269. Aweeka FT, Rosenkranz SL, Segal Y, et al; NIAID AIDS Clinical Trials Group. The impact of sex and contraceptive therapy on the plasma and intracellular pharmacokinetics of zidovudine. AIDS 2006;20:1833–41. http://dx.doi.org/10.1097/01.aids.0000244202.18629.36
270. Kearney BP, Mathias A. Lack of effect of tenofovir disoproxil fumarate on pharmacokinetics of hormonal contraceptives. Pharmacotherapy 2009;29:924–9. http://dx.doi.org/10.1592/phco.29.8.924
271. Todd CS, Deese J, Wang M, et al; FEM-PrEP Study Group. Sino-implant (II)® continuation and effect of concomitant tenofovir disoproxil fumarate-emtricitabine use on plasma levonorgestrel concentrations among women in Bondo, Kenya. Contraception 2015;91:248–52. http://dx.doi.org/10.1016/j.contraception.2014.10.008
272. Murnane PM, Heffron R, Ronald A, et al; Partners PrEP Study Team. Pre-exposure prophylaxis for HIV-1 prevention does not diminish the pregnancy prevention effectiveness of hormonal contraception. AIDS 2014;28:1825–30. http://dx.doi.org/10.1097/QAD.0000000000000290
273. Kasonde M, Niska RW, Rose C, et al. Bone mineral density changes among HIV-uninfected young adults in a randomised trial of pre-exposure prophylaxis with tenofovir-emtricitabine or placebo in Botswana. PLoS One 2014;9:e90111. http://dx.doi.org/10.1371/journal.pone.0090111
274. Callahan R, Nanda K, Kapiga S, et al; FEM-PrEP Study Group. Pregnancy and contraceptive use among women participating in the FEM-PrEP trial. J Acquir Immune Defic Syndr 2015;68:196–203. http://dx.doi.org/10.1097/QAI.0000000000000413
275. Patel RC, Onono M, Gandhi M, et al. Pregnancy rates in HIV-positive women using contraceptives and efavirenz-based or nevirapine-based antiretroviral therapy in Kenya: a retrospective cohort study. Lancet HIV 2015;2:e474–82. http://dx.doi.org/10.1016/S2352-3018(15)00184-8
276. Pyra M, Heffron R, Mugo NR, et al; Partners in Prevention HSV/HIV Transmission Study and Partners PrEP Study Teams. Effectiveness of hormonal contraception in HIV-infected women using antiretroviral therapy. AIDS 2015;29:2353–9. http://dx.doi.org/10.1097/QAD.0000000000000827
277. Clark RA, Theall K. Population-based study evaluating association between selected antiretroviral therapies and potential oral contraceptive failure. J Acquir Immune Defic Syndr 2004;37:1219–20. http://dx.doi.org/10.1097/01.qai.0000136724.15758.ae
278. Landolt NK, Phanuphak N, Ubolyam S, et al. Efavirenz, in contrast to nevirapine, is associated with unfavorable progesterone and antiretroviral levels when coadministered with combined oral contraceptives. J Acquir Immune Defic Syndr 2013;62:534–9. http://dx.doi.org/10.1097/QAI.0b013e31827e8f98
279. Sevinsky H, Eley T, Persson A, et al. The effect of efavirenz on the pharmacokinetics of an oral contraceptive containing ethinyl estradiol and norgestimate in healthy HIV-negative women. Antivir Ther 2011;16:149–56. http://dx.doi.org/10.3851/IMP1725
280. Landolt NK, Phanuphak N, Ubolyam S, et al. Significant decrease of ethinylestradiol with nevirapine, and of etonogestrel with efavirenz in HIV-positive women. J Acquir Immune Defic Syndr 2014;66:e50–2.
281. Schöller-Gyüre M, Kakuda TN, Woodfall B, et al. Effect of steady-state etravirine on the pharmacokinetics and pharmacodynamics of ethinylestradiol and norethindrone. Contraception 2009;80:44–52. http://dx.doi.org/10.1016/j.contraception.2009.01.009

282. Myer L, Carter RJ, Katyal M, Toro P, El-Sadr WM, Abrams EJ. Impact of antiretroviral therapy on incidence of pregnancy among HIV-infected women in Sub-Saharan Africa: a cohort study. PLoS Med 2010;7:e1000229. http://dx.doi.org/10.1371/journal.pmed.1000229
283. Nanda K, Delany-Moretlwe S, Dubé K, et al. Nevirapine-based antiretroviral therapy does not reduce oral contraceptive effectiveness. AIDS 2013;27(Suppl 1):S17–25. http://dx.doi.org/10.1097/QAD.0000000000000050
284. Stuart GS, Moses A, Corbett A, et al. Combined oral contraceptives and antiretroviral PK/PD in Malawian women: pharmacokinetics and pharmacodynamics of a combined oral contraceptive and a generic combined formulation antiretroviral in Malawi. J Acquir Immune Defic Syndr 2011;58:e40–3. http://dx.doi.org/10.1097/QAI.0b013e31822b8bf8
285. Mildvan D, Yarrish R, Marshak A, et al. Pharmacokinetic interaction between nevirapine and ethinyl estradiol/norethindrone when administered concurrently to HIV-infected women. J Acquir Immune Defic Syndr 2002;29:471–7. http://dx.doi.org/10.1097/00042560-200204150-00007
286. Muro E, Droste JA, Hofstede HT, Bosch M, Dolmans W, Burger DM. Nevirapine plasma concentrations are still detectable after more than 2 weeks in the majority of women receiving single-dose nevirapine: implications for intervention studies. J Acquir Immune Defic Syndr 2005;39:419–2. http://dx.doi.org/10.1097/01.qai.0000167154.37357.f9
287. Crauwels HM, van Heeswijk RP, Buelens A, Stevens M, Hoetelmans RM. Lack of an effect of rilpivirine on the pharmacokinetics of ethinylestradiol and norethindrone in healthy volunteers. Int J Clin Pharmacol Ther 2014;52:118–28. http://dx.doi.org/10.5414/CP201943
288. Zhang J, Chung E, Yones C, et al. The effect of atazanavir/ritonavir on the pharmacokinetics of an oral contraceptive containing ethinyl estradiol and norgestimate in healthy women. Antivir Ther 2011;16:157–64. http://dx.doi.org/10.3851/IMP1724
289. Sekar VJ, Lefebvre E, Guzman SS, et al. Pharmacokinetic interaction between ethinyl estradiol, norethindrone and darunavir with low-dose ritonavir in healthy women. Antivir Ther 2008;13:563–9.
290. Glaxo Smith Kline. Lexiva (fosamprenavir calcium) [Package insert]. Research Triangle Park, NC: Glaxo Smith Kline; 2015.
291. Vogler MA, Patterson K, Kamemoto L, et al. Contraceptive efficacy of oral and transdermal hormones when co-administered with protease inhibitors in HIV-1-infected women: pharmacokinetic results of ACTG trial A5188. J Acquir Immune Defic Syndr 2010;55:473–82. http://dx.doi.org/10.1097/QAI.0b013e3181eb5ff5
292. Fröhlich M, Burhenne J, Martin-Facklam M, et al. Oral contraception does not alter single dose saquinavir pharmacokinetics in women. Br J Clin Pharmacol 2004;57:244–52 http://dx.doi.org/10.1111/j.1365-2125.2003.01983.x.
293. Boehringer Ingelheim Pharmaceuticals. Aptivus (tipranavir) [Package insert]. Ridgefield, CT; 2005.
294. Bristol-Myers Squibb. Reyataz (atazanavir sulfate) [Package insert]. Princeton, NJ; 2003.
295. Abel S, Russell D, Whitlock LA, Ridgway CE, Muirhead GJ. Effect of maraviroc on the pharmacokinetics of midazolam, lamivudine/zidovudine, and ethinyloestradiol/levonorgestrel in healthy volunteers. Br J Clin Pharmacol 2008;65(Suppl 1):19–26. http://dx.doi.org/10.1111/j.1365-2125.2008.03132.x
296. Anderson MS, Hanley WD, Moreau AR, et al. Effect of raltegravir on estradiol and norgestimate plasma pharmacokinetics following oral contraceptive administration in healthy women. Br J Clin Pharmacol 2011;71:616–20. http://dx.doi.org/10.1111/j.1365-2125.2010.03885.x
297. Song IH, Borland J, Chen S, Wajima T, Peppercorn AF, Piscitelli SC. Dolutegravir has no effect on the pharmacokinetics of oral contraceptives with norgestimate and ethinyl estradiol. Ann Pharmacother 2015;49:784–9. http://dx.doi.org/10.1177/1060028015580637
298. Gilead Sciences. Vitekta (elvitegravir) [Package insert]. Foster City, CA; 2012.
299. Back DJ, Bates M, Bowden A, et al. The interaction of phenobarbital and other anticonvulsants with oral contraceptive steroid therapy. Contraception 1980;22:495–503. http://dx.doi.org/10.1016/0010-7824(80)90102-X
300. Doose DR, Wang SS, Padmanabhan M, Schwabe S, Jacobs D, Bialer M. Effect of topiramate or carbamazepine on the pharmacokinetics of an oral contraceptive containing norethindrone and ethinyl estradiol in healthy obese and nonobese female subjects. Epilepsia 2003;44:540–9. http://dx.doi.org/10.1046/j.1528-1157.2003.55602.x
301. Fattore C, Cipolla G, Gatti G, et al. Induction of ethinylestradiol and levonorgestrel metabolism by oxcarbazepine in healthy women. Epilepsia 1999;40:783–7. http://dx.doi.org/10.1111/j.1528-1157.1999.tb00779.x
302. Rosenfeld WE, Doose DR, Walker SA, Nayak RK. Effect of topiramate on the pharmacokinetics of an oral contraceptive containing norethindrone and ethinyl estradiol in patients with epilepsy. Epilepsia 1997;38:317–23. http://dx.doi.org/10.1111/j.1528-1157.1997.tb01123.x
303. Christensen J, Petrenaite V, Atterman J, et al. Oral contraceptives induce lamotrigine metabolism: evidence from a double-blind, placebo-controlled trial. Epilepsia 2007;48:484–9. http://dx.doi.org/10.1111/j.1528-1167.2007.00997.x
304. Contin M, Albani F, Ambrosetto G, et al. Variation in lamotrigine plasma concentrations with hormonal contraceptive monthly cycles in patients with epilepsy. Epilepsia 2006;47:1573–5. http://dx.doi.org/10.1111/j.1528-1167.2006.00558.x
305. Reimers A, Helde G, Brodtkorb E. Ethinyl estradiol, not progestogens, reduces lamotrigine serum concentrations. Epilepsia 2005;46:1414–7. http://dx.doi.org/10.1111/j.1528-1167.2005.10105.x
306. Sabers A, Buchholt JM, Uldall P, Hansen EL. Lamotrigine plasma levels reduced by oral contraceptives. Epilepsy Res 2001;47:151–4. http://dx.doi.org/10.1016/S0920-1211(01)00305-9
307. Sabers A, Ohman I, Christensen J, Tomson T. Oral contraceptives reduce lamotrigine plasma levels. Neurology 2003;61:570–1. http://dx.doi.org/10.1212/01.WNL.0000076485.09353.7A
308. Back DJ, Breckenridge AM, MacIver M, et al. The effects of ampicillin on oral contraceptive steroids in women. Br J Clin Pharmacol 1982;14:43–8. http://dx.doi.org/10.1111/j.1365-2125.1982.tb04932.x
309. Back DJ, Grimmer SF, Orme ML, Proudlove C, Mann RD, Breckenridge AM. Evaluation of Committee on Safety of Medicines yellow card reports on oral contraceptive-drug interactions with anticonvulsants and antibiotics. Br J Clin Pharmacol 1988;25:527–32. http://dx.doi.org/10.1111/j.1365-2125.1988.tb03341.x
310. Back DJ, Tjia J, Martin C, et al. The lack of interaction between temafloxacin and combined oral contraceptive steroids. Contraception 1991;43:317–23. http://dx.doi.org/10.1016/0010-7824(91)90070-V
311. Bacon JF, Shenfield GM. Pregnancy attributable to interaction between tetracycline and oral contraceptives. BMJ 1980;280:293. http://dx.doi.org/10.1136/bmj.280.6210.293
312. Bainton R. Interaction between antibiotic therapy and contraceptive medication. Oral Surg Oral Med Oral Pathol 1986;61:453–5. http://dx.doi.org/10.1016/0030-4220(86)90385-3
313. Bollen M. Use of antibiotics when taking the oral contraceptive pill. [comment]. Aust Fam Physician 1995;24:928–9.
314. Bromham DR, Cartmill RS. Knowledge and use of secondary contraception among patients requesting termination of pregnancy. BMJ 1993;306:556–7. http://dx.doi.org/10.1136/bmj.306.6877.556

315. Côté J. Interaction of griseofulvin and oral contraceptives.[comment]. J Am Acad Dermatol 1990;22:124–5. http://dx.doi.org/10.1016/S0190-9622(08)80010-2
316. Csemiczky G, Alvendal C, Landgren BM. Risk for ovulation in women taking a low-dose oral contraceptive (Microgynon) when receiving antibacterial treatment with a fluoroquinolone (ofloxacin). Adv Contracept 1996;12:101–9. http://dx.doi.org/10.1007/BF01849631
317. de Groot AC, Eshuis H, Stricker BH. Inefficacy of oral contraception during use of minocycline [Dutch]. Ned Tijdschr Geneeskd 1990;134:1227–9.
318. DeSano EA Jr, Hurley SC. Possible interactions of antihistamines and antibiotics with oral contraceptive effectiveness. Fertil Steril 1982;37:853–4 http://dx.doi.org/10.1016/S0015-0282(16)46350-8.
319. Donley TG, Smith RF, Roy B. Reduced oral contraceptive effectiveness with concurrent antibiotic use: a protocol for prescribing antibiotics to women of childbearing age. Compendium 1990;11:392–6.
320. Friedman CI, Huneke AL, Kim MH, Powell J. The effect of ampicillin on oral contraceptive effectiveness. Obstet Gynecol 1980;55:33–7.
321. Grimmer SF, Allen WL, Back DJ, Breckenridge AM, Orme M, Tjia J. The effect of cotrimoxazole on oral contraceptive steroids in women. Contraception 1983;28:53–9. http://dx.doi.org/10.1016/S0010-7824(83)80005-5
322. Helms SE, Bredle DL, Zajic J, Jatjoura D, Brodell RT, Krishnarao I. Oral contraceptive failure rates and oral antibiotics. J Am Acad Dermatol 1997;36:705–10. http://dx.doi.org/10.1016/S0190-9622(97)80322-2
323. Hempel E, Böhm W, Carol W, Klinger G. [Enzyme induction by drugs and hormonal contraception]. Zentralbl Gynakol 1973;95:1451–7.
324. Hempel E, Zorn C, Graf K. [Effect of chemotherapy agents and antibiotics on hormonal contraception]. Z Arztl Fortbild (Jena) 1978;72:924–6.
325. Hetényi G. Possible interactions between antibiotics and oral contraceptives. Ther Hung 1989;37:86–9.
326. Hughes BR, Cunliffe WJ. Interactions between the oral contraceptive pill and antibiotics.[comment]. Br J Dermatol 1990;122:717–8. http://dx.doi.org/10.1111/j.1365-2133.1990.tb07299.x
327. Joshi JV, Joshi UM, Sankholi GM, et al. A study of interaction of low-dose combination oral contraceptive with Ampicillin and Metronidazole. Contraception 1980;22:643–52. http://dx.doi.org/10.1016/0010-7824(80)90089-X
328. Kakouris H, Kovacs GT. Pill failure and nonuse of secondary precautions. Br J Fam Plann 1992;18:41–4.
329. Kakouris H, Kovacs GT. How common are predisposing factors to pill failure among pill users? Br J Fam Plann 1994;20:33–5.
330. Kovacs GT, Riddoch G, Duncombe P, et al. Inadvertent pregnancies in oral contraceptive users. Med J Aust 1989;150:549–51.
331. Lequeux A. [Pregnancy under oral contraceptives after treatment with tetracycline]. Louv Med 1980;99:413–4.
332. London BM, Lookingbill DP. Frequency of pregnancy in acne patients taking oral antibiotics and oral contraceptives. Arch Dermatol 1994;130:392–3. http://dx.doi.org/10.1001/archderm.1994.01690030128027
333. Maggiolo F, Puricelli G, Dottorini M, Caprioli S, Bianchi W, Suter F. The effect of ciprofloxacin on oral contraceptive steroid treatments. Drugs Exp Clin Res 1991;17:451–4.
334. Murphy AA, Zacur HA, Charache P, Burkman RT. The effect of tetracycline on levels of oral contraceptives. Am J Obstet Gynecol 1991;164:28–33. http://dx.doi.org/10.1016/0002-9378(91)90617-Z
335. Neely JL, Abate M, Swinker M, D'Angio R. The effect of doxycycline on serum levels of ethinyl estradiol, norethindrone, and endogenous progesterone. Obstet Gynecol 1991;77:416–20.
336. Pillans PI, Sparrow MJ. Pregnancy associated with a combined oral contraceptive and itraconazole.[comment]. N Z Med J 1993;106:436.
337. Scholten PC, Droppert RM, Zwinkels MG, Moesker HL, Nauta JJ, Hoepelman IM. No interaction between ciprofloxacin and an oral contraceptive. Antimicrob Agents Chemother 1998;42:3266–8.
338. Silber TJ. Apparent oral contraceptive failure associated with antibiotic administration. J Adolesc Health Care 1983;4:287–9. http://dx.doi.org/10.1016/S0197-0070(83)80014-X
339. Sparrow MJ. Pill method failures. N Z Med J 1987;100:102–5.
340. Sparrow MJ. Pregnancies in reliable pill takers. N Z Med J 1989;102:575–7.
341. Sparrow MJ. Pill method failures in women seeking abortion: fourteen years experience. N Z Med J 1998;111:386–8.
342. van Dijke CP, Weber JC. Interaction between oral contraceptives and griseofulvin. Br Med J (Clin Res Ed) 1984;288:1125–6. http://dx.doi.org/10.1136/bmj.288.6424.1125-a
343. Wermeling DP, Chandler MH, Sides GD, Collins D, Muse KN. Dirithromycin increases ethinyl estradiol clearance without allowing ovulation. Obstet Gynecol 1995;86:78–84. http://dx.doi.org/10.1016/0029-7844(95)00075-3
344. Young LK, Farquhar CM, McCowan LM, Roberts HE, Taylor J. The contraceptive practices of women seeking termination of pregnancy in an Auckland clinic. N Z Med J 1994;107:189–92.
345. Abrams LS, Skee D, Natarajan J, Wong FA. Pharmacokinetic overview of Ortho Evra/Evra. Fertil Steril 2002;77(Suppl 2):S3–12. http://dx.doi.org/10.1016/S0015-0282(01)03261-7
346. Dogterom P, van den Heuvel MW, Thomsen T. Absence of pharmacokinetic interactions of the combined contraceptive vaginal ring NuvaRing with oral amoxicillin or doxycycline in two randomised trials. Clin Pharmacokinet 2005;44:429–38. http://dx.doi.org/10.2165/00003088-200544040-00007
347. Devenport MH, Crook D, Wynn V, Lees LJ. Metabolic effects of low-dose fluconazole in healthy female users and non-users of oral contraceptives. Br J Clin Pharmacol 1989;27:851–9. http://dx.doi.org/10.1111/j.1365-2125.1989.tb03449.x
348. Hilbert J, Messig M, Kuye O, Friedman H. Evaluation of interaction between fluconazole and an oral contraceptive in healthy women. Obstet Gynecol 2001;98:218–23.
349. Kovács I, Somos P, Hámori M. Examination of the potential interaction between ketoconazole (Nizoral) and oral contraceptives with special regard to products of low hormone content (Rigevidon, anteovin). Ther Hung 1986;34:167–70.
350. Lunell NO, Pschera H, Zador G, Carlström K. Evaluation of the possible interaction of the antifungal triazole SCH 39304 with oral contraceptives in normal healthy women. Gynecol Obstet Invest 1991;32:91–7. http://dx.doi.org/10.1159/000293003
351. McDaniel PA, Caldroney RD. Oral contraceptives and griseofulvin interactions. Drug Intell Clin Pharm 1986;20:384.
352. Meyboom RH, van Puijenbroek EP, Vinks MH, Lastdrager CJ. Disturbance of withdrawal bleeding during concomitant use of itraconazole and oral contraceptives. N Z Med J 1997;110:300.
353. Rieth H, Sauerbrey N. [Interaction studies with fluconazole, a new triazole antifungal drug]. Wien Med Wochenschr 1989;139:370–4.
354. Sinofsky FE, Pasquale SA. The effect of fluconazole on circulating ethinyl estradiol levels in women taking oral contraceptives. Am J Obstet Gynecol 1998;178:300–4. http://dx.doi.org/10.1016/S0002-9378(98)80016-1

355. van Puijenbroek EP, Feenstra J, Meyboom RH. [Pill cycle disturbance in simultaneous use of itraconazole and oral contraceptives]. Ned Tijdschr Geneeskd 1998;142:146–9.
356. Van Puijenbroek EP, Egberts AC, Meyboom RH, Leufkens HG. Signalling possible drug-drug interactions in a spontaneous reporting system: delay of withdrawal bleeding during concomitant use of oral contraceptives and itraconazole. Br J Clin Pharmacol 1999;47:689–93. http://dx.doi.org/10.1046/j.1365-2125.1999.00957.x
357. Verhoeven CH, van den Heuvel MW, Mulders TM, Dieben TO. The contraceptive vaginal ring, NuvaRing, and antimycotic co-medication. Contraception 2004;69:129–32. http://dx.doi.org/10.1016/j.contraception.2003.10.001
358. Back DJ, Breckenridge AM, Grimmer SF, Orme ML, Purba HS. Pharmacokinetics of oral contraceptive steroids following the administration of the antimalarial drugs primaquine and chloroquine. Contraception 1984;30:289–95. http://dx.doi.org/10.1016/0010-7824(84)90092-1
359. Croft AM, Herxheimer A. Adverse effects of the antimalaria drug, mefloquine: due to primary liver damage with secondary thyroid involvement? BMC Public Health 2002;2:6. http://dx.doi.org/10.1186/1471-2458-2-6
360. Karbwang J, Looareesuwan S, Back DJ, Migasana S, Bunnag D, Breckenridge AM. Effect of oral contraceptive steroids on the clinical course of malaria infection and on the pharmacokinetics of mefloquine in Thai women. Bull World Health Organ 1988;66:763–7.
361. McGready R, Stepniewska K, Seaton E, et al. Pregnancy and use of oral contraceptives reduces the biotransformation of proguanil to cycloguanil. Eur J Clin Pharmacol 2003;59:553–7. http://dx.doi.org/10.1007/s00228-003-0651-x
362. Wanwimolruk S, Kaewvichit S, Tanthayaphinant O, Suwannarach C, Oranratnachai A. Lack of effect of oral contraceptive use on the pharmacokinetics of quinine. Br J Clin Pharmacol 1991;31:179–81. http://dx.doi.org/10.1111/j.1365-2125.1991.tb05509.x
363. Back DJ, Breckenridge AM, Crawford F, et al. The effect of rifampicin on norethisterone pharmacokinetics. Eur J Clin Pharmacol 1979;15:193–7. http://dx.doi.org/10.1007/BF00563105
364. Back DJ, Breckenridge AM, Crawford FE, et al. The effect of rifampicin on the pharmacokinetics of ethynylestradiol in women. Contraception 1980;21:135–43. http://dx.doi.org/10.1016/0010-7824(80)90125-0
365. Barditch-Crovo P, Trapnell CB, Ette E, et al. The effects of rifampin and rifabutin on the pharmacokinetics and pharmacodynamics of a combination oral contraceptive. Clin Pharmacol Ther 1999;65:428–38. http://dx.doi.org/10.1016/S0009-9236(99)70138-4
366. Bolt HM, Bolt M, Kappus H. Interaction of rifampicin treatment with pharmacokinetics and metabolism of ethinyloestradiol in man. Acta Endocrinol (Copenh) 1977;85:189–97.
367. Gupta KC, Ali MY. Failure of oral contraceptive with rifampicin. Med J Zambia 1980;15:23.
368. Hirsch A. [Letter: Sleeping pills]. Nouv Presse Med 1973;2:2957.
369. Hirsch A, Tillement JP, Chretien J. Effets contrariants de la rifampicine sur les contraceptifs oraux: a propos de trois grossesses non desiree chez trois malades. Rev Fr Mal Respir 1975;2:174–82.
370. Joshi JV, Joshi UM, Sankolli GM, et al. A study of interaction of a low-dose combination oral contraceptive with anti-tubercular drugs. Contraception 1980;21:617–29. http://dx.doi.org/10.1016/0010-7824(80)90034-7
371. Kropp R. [Rifampicin and oral contraceptives (author's transl)]. Prax Pneumol 1974;28:270–2.
372. LeBel M, Masson E, Guilbert E, et al. Effects of rifabutin and rifampicin on the pharmacokinetics of ethinylestradiol and norethindrone. J Clin Pharmacol 1998;38:1042–50. http://dx.doi.org/10.1177/009127009803801109
373. Meyer B, Müller F, Wessels P, Maree J. A model to detect interactions between roxithromycin and oral contraceptives. Clin Pharmacol Ther 1990;47:671–4. http://dx.doi.org/10.1038/clpt.1990.92
374. Nocke-Finck L, Breuer H, Reimers D. Dtsch Med Wochenschr 1973;98:1521–3. http://dx.doi.org/10.1055/s-0028-1107071
375. Piguet B, Muglioni JF, Chaline G. [Letter: Oral contraception and rifampicin]. Nouv Presse Med 1975;04:115–6.
376. Reimers D, Jezek A. [The simultaneous use of rifampicin and other antitubercular agents with oral contraceptives]. Prax Pneumol 1971;25:255–62.
377. Skolnick JL, Stoler BS, Katz DB, Anderson WH. Rifampin, oral contraceptives, and pregnancy. JAMA 1976;236:1382. http://dx.doi.org/10.1001/jama.1976.03270130044027
378. Szoka PR, Edgren RA. Drug interactions with oral contraceptives: compilation and analysis of an adverse experience report database. Fertil Steril 1988;49(Suppl 2):31S–8S.
379. Berry-Bibee E, Kim MJ, Simmons K, Pagano P, Curtis K. Drug interactions between hormonal contraceptives and psychotropic drugs: a systematic review. Contraception. In press 2016.
380. Berry-Bibee E, Kim MJ, Tepper N, Riley H, Curtis K. The safety of St John's wort and hormonal contraceptives: a systematic review. Contraception. In press 2016.

Appendix E

Classifications for Barrier Methods

Classifications for barrier contraceptive methods include those for condoms, which include male latex condoms, male polyurethane condoms, and female condoms; spermicides; and diaphragm with spermicide or cervical cap (Box E1) (Table E1).

Women with conditions that make pregnancy an unacceptable risk should be advised that barrier methods for pregnancy prevention might not be appropriate for those who cannot use them consistently and correctly because of the relatively higher typical-use failure rates of these methods. Women should be counseled that consistent and correct use of the male latex condom reduces the risk for transmission of human immunodeficiency virus (HIV) and other sexually transmitted diseases (STDs). Use of female condoms can provide protection from transmission of STDs, although data are limited.

BOX E1. Categories for classifying barrier methods

1 = A condition for which there is no restriction for the use of the contraceptive method.
2 = A condition for which the advantages of using the method generally outweigh the theoretical or proven risks.
3 = A condition for which the theoretical or proven risks usually outweigh the advantages of using the method.
4 = A condition that represents an unacceptable health risk if the contraceptive method is used.

TABLE E1. Classifications for barrier methods, including condoms, spermicides, diaphragms (with spermicide), and cap

Condition	Category			Clarifications/Evidence/Comments
	Condom	Spermicide	Diaphragm (with spermicide)/Cap	
Personal Characteristics and Reproductive History				
Pregnancy	NA	NA	NA	**Clarification:** None of these methods are relevant for contraception during known pregnancy. However, for women who remain at risk for STDs/HIV during pregnancy, the correct and consistent use of condoms is recommended.
Age				
a. Menarche to <40 years	1	1	1	—
b. ≥40 years	1	1	1	—
Parity				
a. Nulliparous	1	1	1	—
b. Parous	1	1	2	**Clarification:** Risk for cervical cap failure is higher in parous women than in nulliparous women.
Postpartum (breastfeeding and nonbreastfeeding)				
a. <6 weeks postpartum	1	1	NA	**Clarification:** Diaphragm and cap are unsuitable until uterine involution is complete.
b. ≥6 weeks postpartum	1	1	1	—
Postabortion				
a. First trimester	1	1	1	—
b. Second trimester	1	1	1	**Clarification:** Diaphragm and cap are unsuitable until 6 weeks after second trimester abortion.
c. Immediate postseptic abortion	1	1	1	—
Past ectopic pregnancy	1	1	1	—
History of pelvic surgery	1	1	1	—
Smoking				
a. Age <35 years	1	1	1	—
b. Age ≥35 years				
i. <15 cigarettes per day	1	1	1	—
ii. ≥15 cigarettes per day	1	1	1	—
Obesity				**Comment:** Severe obesity might make diaphragm and cap placement difficult.
a. BMI ≥30 kg/m^2	1	1	1	
b. Menarche to <18 years and BMI ≥30 kg/m^2	1	1	1	

See table footnotes on page 87.

TABLE E1. (*Continued*) Classifications for barrier methods, including condoms, spermicides, diaphragms (with spermicide), and cap

Condition	Category			Clarifications/Evidence/Comments
	Condom	Spermicide	Diaphragm (with spermicide)/Cap	
History of bariatric surgery This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).				
a. Restrictive procedures: decrease storage capacity of the stomach (vertical banded gastroplasty, laparoscopic adjustable gastric band, or laparoscopic sleeve gastrectomy)	1	1	1	—
b. Malabsorptive procedures: decrease absorption of nutrients and calories by shortening the functional length of the small intestine (Roux-en-Y gastric bypass or biliopancreatic diversion)	1	1	1	—
Cardiovascular Disease				
Multiple risk factors for atherosclerotic cardiovascular disease (e.g., older age, smoking, diabetes, hypertension, low HDL, high LDL, or high triglyceride levels)	1	1	1	—
Hypertension Systolic blood pressure ≥160 mm Hg or diastolic blood pressure ≥100 mm Hg are associated with increased risk for adverse health events as a result of pregnancy (Box 2).				
a. Adequately controlled hypertension	1	1	1	—
b. Elevated blood pressure levels (properly taken measurements)				
i. Systolic 140–159 mm Hg or diastolic 90–99 mm Hg	1	1	1	—
ii. Systolic ≥160 mm Hg or diastolic ≥100 mm Hg	1	1	1	—
c. Vascular disease	1	1	1	—
History of high blood pressure during pregnancy (when current blood pressure is measurable and normal)	1	1	1	—
Deep venous thrombosis/Pulmonary embolism				
a. History of DVT/PE, not receiving anticoagulant therapy				
i. Higher risk for recurrent DVT/PE (one or more risk factors) • History of estrogen-associated DVT/PE • Pregnancy-associated DVT/PE • Idiopathic DVT/PE • Known thrombophilia, including antiphospholipid syndrome • Active cancer (metastatic, receiving therapy, or within 6 months after clinical remission), excluding nonmelanoma skin cancer • History of recurrent DVT/PE	1	1	1	—
ii. Lower risk for recurrent DVT/PE (no risk factors)	1	1	1	—
b. Acute DVT/PE	1	1	1	—
c. DVT/PE and established anticoagulant therapy for at least 3 months				
i. Higher risk for recurrent DVT/PE (one or more risk factors) • Known thrombophilia, including antiphospholipid syndrome • Active cancer (metastatic, receiving therapy, or within 6 months after clinical remission), excluding nonmelanoma skin cancer • History of recurrent DVT/PE	1	1	1	
ii. Lower risk for recurrent DVT/PE (no risk factors)	1	1	1	—
d. Family history (first-degree relatives)	1	1	1	—
e. Major surgery				
i. With prolonged immobilization	1	1	1	—
ii. Without prolonged immobilization	1	1	1	—
f. Minor surgery without immobilization	1	1	1	—

See table footnotes on page 87.

TABLE E1. (*Continued*) Classifications for barrier methods, including condoms, spermicides, diaphragms (with spermicide), and cap

Condition	Category			Clarifications/Evidence/Comments
	Condom	Spermicide	Diaphragm (with spermicide)/Cap	
Known thrombogenic mutations (e.g., factor V Leiden; prothrombin mutation; or protein S, protein C, and antithrombin deficiencies) This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	1	1	1	**Clarification:** Routine screening is not appropriate because of the rarity of the conditions and the high cost of screening.
Superficial venous disorders				
a. Varicose veins	1	1	1	—
b. Superficial venous thrombosis (acute or history)	1	1	1	—
Current and history of ischemic heart disease This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	1	1	1	—
Stroke (history of cerebrovascular accident) This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	1	1	1	—
Valvular heart disease Complicated valvular heart disease is associated with increased risk for adverse health events as a result of pregnancy (Box 2).				
a. Uncomplicated	1	1	1	—
b. Complicated (pulmonary hypertension, risk for atrial fibrillation, or history of subacute bacterial endocarditis)	1	1	2	—
Peripartum cardiomyopathy This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).				
a. Normal or mildly impaired cardiac function (New York Heart Association Functional Class I or II: patients with no limitation of activities or patients with slight, mild limitation of activity) (*1*)				
i. <6 months	1	1	1	—
ii. ≥6 months	1	1	1	—
b. Moderately or severely impaired cardiac function (New York Heart Association Functional Class III or IV: patients with marked limitation of activity or patients who should be at complete rest) (*1*)	1	1	1	—
Rheumatic Diseases				
Systemic lupus erythematosus This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).				
a. Positive (or unknown) antiphospholipid antibodies	1	1	1	—
b. Severe thrombocytopenia	1	1	1	—
c. Immunosuppressive therapy	1	1	1	—
d. None of the above	1	1	1	—
Rheumatoid arthritis				
a. Receiving immunosuppressive therapy	1	1	1	—
b. Not receiving immunosuppressive therapy	1	1	1	—
Neurologic Conditions				
Headaches				—
a. Nonmigraine (mild or severe)	1	1	1	—
b. Migraine				
i. Without aura (This category of migraine includes menstrual migraine.)	1	1	1	**Comment:** Menstrual migraine is a subtype of migraine without aura. For more information see *The International Headache Society Classification, 3rd edition* (http://www.ihs-classification.org/_downloads/mixed/International-Headache-Classification-III-ICHD-III-2013-Beta.pdf).
ii. With aura	1	1	1	—
Epilepsy This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	1	1	1	—
Multiple sclerosis				
a. With prolonged immobility	1	1	1	—
b. Without prolonged immobility	1	1	1	—
Depressive Disorders				
Depressive disorders	1	1	1	—
Reproductive Tract Infections and Disorders				
Unexplained vaginal bleeding (suspicious for serious condition) before evaluation	1	1	1	**Clarification:** If pregnancy or an underlying pathological condition (e.g., pelvic malignancy) is suspected, it must be evaluated and the category adjusted after evaluation.

See table footnotes on page 87.

TABLE E1. (*Continued*) Classifications for barrier methods, including condoms, spermicides, diaphragms (with spermicide), and cap

Condition	Category			Clarifications/Evidence/Comments
	Condom	Spermicide	Diaphragm (with spermicide)/Cap	
Endometriosis	1	1	1	—
Benign ovarian tumors (including cysts)	1	1	1	—
Severe dysmenorrhea	1	1	1	—
Gestational trophoblastic disease This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).				
a. Suspected gestational trophoblastic disease (immediate postevacuation)				
i. Uterine size first trimester	1	1	1	—
ii. Uterine size second trimester	1	1	1	—
b. Confirmed gestational trophoblastic disease (after initial evacuation and during monitoring)				
i. Undetectable/nonpregnant β–hCG levels	1	1	1	—
ii. Decreasing β–hCG levels	1	1	1	—
iii. Persistently elevated β-hCG levels or malignant disease, with no evidence or suspicion of intrauterine disease	1	1	1	—
iv. Persistently elevated β-hCG levels or malignant disease, with evidence or suspicion of intrauterine disease	1	1	1	—
Cervical ectropion	1	1	1	—
Cervical intraepithelial neoplasia	1	1	1	**Clarification:** The cap should not be used. Diaphragm use has no restrictions.
Cervical cancer (awaiting treatment)	1	2	1	**Clarification:** The cap should not be used. Diaphragm use has no restrictions. **Comment:** Repeated and high-dose use of the spermicide nonoxynol-9 can cause vaginal and cervical irritation or abrasions.
Breast disease Breast cancer is associated with increased risk for adverse health events as a result of pregnancy (Box 2).				
a. Undiagnosed mass	1	1	1	—
b. Benign breast disease	1	1	1	—
c. Family history of cancer	1	1	1	—
d. Breast cancer				
i. Current	1	1	1	—
ii. Past and no evidence of current disease for 5 years	1	1	1	—
Endometrial hyperplasia	1	1	1	—
Endometrial cancer This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	1	1	1	—
Ovarian cancer This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	1	1	1	—
Uterine fibroids	1	1	1	—
Anatomical abnormalities	1	1	NA	**Clarification:** The diaphragm cannot be used in certain cases of prolapse. Cap use is not appropriate for a woman with markedly distorted cervical anatomy.
Pelvic inflammatory disease				
a. Past PID				
i. With subsequent pregnancy	1	1	1	—
ii. Without subsequent pregnancy	1	1	1	—
b. Current PID	1	1	1	—
Sexually transmitted diseases				
a. Current purulent cervicitis or chlamydial infection or gonococcal infection	1	1	1	—
b. Vaginitis (including *Trichomonas vaginalis* and bacterial vaginosis)	1	1	1	—
c. Other factors related to STDs	1	1	1	—
HIV				
High risk for HIV	1	4	4	**Evidence:** Repeated and high-dose use of the spermicide nonoxynol-9 was associated with increased risk for genital lesions, which might increase the risk for HIV infection (*2*). **Comment:** Diaphragm use is assigned category 4 because of concerns about the spermicide, not the diaphragm.

See table footnotes on page 87.

TABLE E1. (*Continued*) Classifications for barrier methods, including condoms, spermicides, diaphragms (with spermicide), and cap

Condition	Category			Clarifications/Evidence/Comments
	Condom	Spermicide	Diaphragm (with spermicide)/Cap	
HIV infection For women with HIV infection who are not clinically well or not receiving ARV therapy, this condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	1	3	3	**Comment:** Use of spermicides or diaphragms (with spermicide) can disrupt the cervical mucosa, which might increase viral shedding and HIV transmission to noninfected sex partners.
Other Infections				
Schistosomiasis Schistosomiasis with fibrosis of the liver is associated with increased risk for adverse health events as a result of pregnancy (Box 2).				
a. Uncomplicated	1	1	1	—
b. Fibrosis of the liver	1	1	1	—
Tuberculosis This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).				
a. Nonpelvic	1	1	1	—
b. Pelvic	1	1	1	—
Malaria	1	1	1	—
History of toxic shock syndrome	1	1	3	**Comment:** Toxic shock syndrome has been reported in association with contraceptive sponge and diaphragm use.
Urinary tract infection	1	1	2	**Comment:** Use of diaphragms and spermicides might increase risk for urinary tract infection.
Endocrine Conditions				
Diabetes Insulin-dependent diabetes; diabetes with nephropathy, retinopathy, or neuropathy; diabetes with other vascular disease; or diabetes of >20 years' duration are associated with increased risk for adverse health events as a result of pregnancy (Box 2).				
a. History of gestational disease	1	1	1	—
b. Nonvascular disease				
i. Non-insulin dependent	1	1	1	—
ii. Insulin dependent	1	1	1	—
c. Nephropathy, retinopathy, or neuropathy	1	1	1	—
d. Other vascular disease or diabetes of >20 years' duration	1	1	1	—
Thyroid disorders				
a. Simple goiter	1	1	1	—
b. Hyperthyroid	1	1	1	—
c. Hypothyroid	1	1	1	—
Gastrointestinal Conditions				
Inflammatory bowel disease (ulcerative colitis or Crohn's disease)	1	1	1	—
Gallbladder disease				
a. Symptomatic				
i. Treated by cholecystectomy	1	1	1	—
ii. Medically treated	1	1	1	—
iii. Current	1	1	1	—
b. Asymptomatic	1	1	1	—
History of cholestasis				
a. Pregnancy related	1	1	1	—
b. Past COC related	1	1	1	—
Viral hepatitis				
a. Acute or flare	1	1	1	—
b. Carrier	1	1	1	—
c. Chronic	1	1	1	—
Cirrhosis Severe cirrhosis is associated with increased risk for adverse health events as a result of pregnancy (Box 2).				
a. Mild (compensated)	1	1	1	—
b. Severe (decompensated)	1	1	1	—

See table footnotes on page 87.

TABLE E1. (*Continued*) Classifications for barrier methods, including condoms, spermicides, diaphragms (with spermicide), and cap

Condition	Category			Clarifications/Evidence/Comments
	Condom	Spermicide	Diaphragm (with spermicide)/Cap	
Liver tumors				
Hepatocellular adenoma and malignant liver tumors are associated with increased risk for adverse health events as a result of pregnancy (Box 2).				
a. Benign				
i. Focal nodular hyperplasia	1	1	1	—
ii. Hepatocellular adenoma	1	1	1	—
b. Malignant (hepatoma)	1	1	1	—
Respiratory Conditions				
Cystic fibrosis	1	1	1	—
This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).				
Anemias				
Thalassemia	1	1	1	—
Sickle cell disease	1	1	1	—
This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).				
Iron deficiency anemia	1	1	1	—
Solid Organ Transplantation				
Solid organ transplantation				
This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).				
a. Complicated: graft failure (acute or chronic), rejection, or cardiac allograft vasculopathy	1	1	1	—
b. Uncomplicated	1	1	1	—
Drug Interactions				
Antiretroviral therapy				**Clarification:** No drug interaction between ARV therapy and barrier method use is known. However, HIV infection is classified as category 3 for spermicides and diaphragms (see HIV section).
a. Nucleoside reverse transcriptase inhibitors (NRTIs)				
i. Abacavir (ABC)	1	3	3	
ii. Tenofovir (TDF)	1	3	3	
iii. Zidovudine (AZT)	1	3	3	
iv. Lamivudine (3TC)	1	3	3	
v. Didanosine (DDI)	1	3	3	
vi. Emtricitabine (FTC)	1	3	3	
vii. Stavudine (D4T)	1	3	3	
b. Nonnucleoside reverse transcriptase inhibitors (NNRTIs)				
i. Efavirenz (EFV)	1	3	3	
ii. Etravirine (ETR)	1	3	3	
iii. Nevirapine (NVP)	1	3	3	
iv. Rilpivirine (RPV)	1	3	3	
c. Ritonavir-boosted protease inhibitors				
i. Ritonavir-boosted atazanavir (ATV/r)	1	3	3	
ii. Ritonavir-boosted darunavir (DRV/r)	1	3	3	
iii. Ritonavir-boosted fosamprenavir (FPV/r)	1	3	3	
iv. Ritonavir-boosted lopinavir (LPV/r)	1	3	3	
v. Ritonavir-boosted saquinavir (SQV/r)	1	3	3	
vi. Ritonavir-boosted tipranavir (TPV/r)	1	3	3	
d. Protease inhibitors without ritonavir				
i. Atazanavir (ATV)	1	3	3	
ii. Fosamprenavir (FPV)	1	3	3	
iii. Indinavir (IDV)	1	3	3	
iv. Nelfinavir (NFV)	1	3	3	
e. CCR5 co-receptor antagonists				
i. Maraviroc (MVC)	1	3	3	
f. HIV integrase strand transfer inhibitors				
i. Raltegravir (RAL)	1	3	3	
ii. Dolutegravir (DTG)	1	3	3	
iii. Elvitegravir (EVG)	1	3	3	
g. Fusion inhibitors				
i. Enfuvirtide	1	3	3	
Anticonvulsant therapy				
a. Certain anticonvulsants (phenytoin, carbamazepine, barbiturates, primidone, topiramate, or oxcarbazepine)	1	1	1	—
b. Lamotrigine	1	1	1	—

See table footnotes on page 87.

TABLE E1. (*Continued*) Classifications for barrier methods, including condoms, spermicides, diaphragms (with spermicide), and cap

Condition	Category			Clarifications/Evidence/Comments
	Condom	Spermicide	Diaphragm (with spermicide)/Cap	
Antimicrobial therapy				
a. Broad-spectrum antibiotics	1	1	1	—
b. Antifungals	1	1	1	—
c. Antiparasitics	1	1	1	—
d. Rifampin or rifabutin therapy	1	1	1	—
Psychotropic medications				
a. SSRIs	1	1	1	—
St. John's wort	1	1	1	—
Allergy to latex	3	1	3	**Clarification:** The condition of allergy to latex does not apply to plastic condoms/diaphragms.

Abbreviations: ARV = antiretroviral; BMI = body mass index; COC = combined oral contraceptive; DVT = deep venous thrombosis; hCG = human chorionic gonadotropin; HDL = high-density lipoprotein; HIV = human immunodeficiency virus; LDL = low-density lipoprotein; NA = not applicable; PE = pulmonary embolism; PID = pelvic inflammatory disease; SSRI = selective serotonin reuptake inhibitor; STD = sexually transmitted disease.

References

1. The Criteria Committee of the New York Heart Association. Nomenclature and criteria for diagnosis of diseases of the heart and great vessels. 9th ed. Boston, MA: Little, Brown and Co; 1994.
2. Wilkinson D, Ramjee G, Tholandi M, Rutherford G. Nonoxynol-9 for preventing vaginal acquisition of HIV infection by women from men. Cochrane Database Syst Rev 2002;4(CD003936):CD003936.

Appendix F

Classifications for Fertility Awareness–Based Methods

Fertility awareness–based (FAB) methods of family planning involve identifying the fertile days of the menstrual cycle, whether by observing fertility signs such as cervical secretions and basal body temperature or by monitoring cycle days (Box F1) (Table F1). FAB methods can be used in combination with abstinence or barrier methods during the fertile time. If barrier methods are used, see the Classifications for Barrier Methods (Appendix E).

No medical conditions worsen because of FAB methods. In general, FAB methods can be used without concern for health effects in persons who choose them. However, several conditions make their use more complex. The existence of these conditions suggests that 1) use of these methods should be delayed until the condition is corrected or resolved, or 2) persons using FAB methods need special counseling, and a provider with particular training in use of these methods is generally necessary to ensure correct use.

Women with conditions that make pregnancy an unacceptable risk should be advised that FAB methods might not be appropriate for them because of the relatively higher typical-use failure rates of these methods. Symptoms-based and calendar-based methods do not protect against sexually transmitted diseases (STDs), including human immunodeficiency virus (HIV), and women using these methods should be counseled that consistent and correct use of the male latex condom reduces the risk for transmission of HIV and other STDs. Use of female condoms can provide protection from transmission of STDs, although data are limited.

BOX F1. Definitions for terms associated with fertility awareness–based methods

- **Symptoms-based methods:** FAB methods based on observation of fertility signs (e.g., cervical secretions or basal body temperature) such as the cervical mucus method, the symptothermal method, and the TwoDay method.
- **Calendar-based methods:** FAB methods based on calendar calculations such as the calendar rhythm method and the standard days method.
- **Accept:** No medical reason exists to deny the particular FAB method to a woman in this circumstance.
- **Caution:** The method normally is provided in a routine setting but with extra preparation and precautions. For FAB methods, this usually means that special counseling might be needed to ensure correct use of the method by a woman in this circumstance.
- **Delay:** Use of this method should be delayed until the condition is evaluated or corrected. Alternative temporary methods of contraception should be offered.

Abbreviation: FAB = fertility awareness–based.

TABLE F1. Fertility awareness–based methods, including symptoms-based and calendar-based methods

Condition	Category: Symptoms-based method	Category: Calendar-based method	Clarifications/Evidence/Comments
Personal Characteristics and Reproductive History			
Pregnancy	NA	NA	**Clarification:** FAB methods are not relevant during pregnancy.
Life stage			**Comment:** Menstrual irregularities are common in postmenarche and perimenopause and might complicate the use of FAB methods.
a. Postmenarche	Caution	Caution	
b. Perimenopause	Caution	Caution	
Breastfeeding			**Comment:** Use of FAB methods when breastfeeding might be less effective than when not breastfeeding.
a. <6 weeks postpartum	Delay	Delay	**Comment:** Women who are primarily breastfeeding and are amenorrheic are unlikely to have sufficient ovarian function to produce detectable fertility signs and hormonal changes during the first 6 months postpartum. However, the likelihood of resumption of fertility increases with time postpartum and with substitution of breast milk by other foods.
b. ≥6 weeks	Caution	Delay	
c. After menses begin	Caution	Caution	**Clarification:** When the woman notices fertility signs, particularly cervical secretions, she can use a symptoms-based method. First postpartum menstrual cycles in breastfeeding women vary significantly in length. Return to regularity takes several cycles. When she has had at least three postpartum menses and her cycles are regular again, she can use a calendar-based method. When she has had at least four postpartum menses and her most recent cycle lasted 26–32 days, she can use the standard days method. Before that time, a barrier method should be offered if the woman plans to use a FAB method later.
Postpartum (nonbreastfeeding women)			
a. <4 weeks	Delay	Delay	**Clarification:** Nonbreastfeeding women are not likely to have detectable fertility signs or hormonal changes before 4 weeks postpartum. Although the risk for pregnancy is low, ovulation before first menses is common; therefore, a method appropriate for the postpartum period should be offered.
b. ≥4 weeks	Accept	Delay	**Clarification:** Nonbreastfeeding women are likely to have sufficient ovarian function to produce detectable fertility signs, hormonal changes, or both at this time; likelihood increases rapidly with time postpartum. Women can use calendar-based methods as soon as they have completed three postpartum menses. Methods appropriate for the postpartum period should be offered before that time.
Postabortion	Caution	Delay	**Clarification:** After abortion, women are likely to have sufficient ovarian function to produce detectable fertility signs, hormonal changes, or both; likelihood increases with time postabortion. Women can start using calendar-based methods after they have had at least one postabortion menses (e.g., women who before this pregnancy primarily had cycles of 26–32 days can then use the standard days method). Methods appropriate for the postabortion period should be offered before that time.
Reproductive Tract Infections and Disorders			
Irregular vaginal bleeding	Delay	Delay	**Clarification:** Presence of this condition makes FAB methods unreliable. Therefore, barrier methods should be recommended until the bleeding pattern is compatible with proper method use. The condition should be evaluated and treated as necessary.
Vaginal discharge	Delay	Accept	**Clarification:** Because vaginal discharge makes recognition of cervical secretions difficult, the condition should be evaluated and treated if needed before providing methods based on cervical secretions.
Other			
Use of drugs that affect cycle regularity, hormones, or fertility signs	Caution /Delay	Caution/Delay	**Clarification:** Use of certain mood-altering drugs such as lithium, tricyclic antidepressants, and antianxiety therapies, as well as certain antibiotics and anti-inflammatory drugs, might alter cycle regularity or affect fertility signs. The condition should be carefully evaluated and a barrier method offered until the degree of effect has been determined or the drug is no longer being used.
Diseases that elevate body temperature			
a. Chronic diseases	Caution	Accept	**Clarification:** Elevated temperatures might make basal body temperature difficult to interpret but have no effect on cervical secretions. Thus, use of a method that relies on temperature should be delayed until the acute febrile disease abates. Temperature-based methods are not appropriate for women with chronically elevated temperatures. In addition, some chronic diseases interfere with cycle regularity, making calendar-based methods difficult to interpret.
b. Acute diseases	Delay	Accept	

Abbreviations: FAB = fertility awareness–based; NA = not applicable.

Appendix G

Lactational Amenorrhea Method

The Bellagio Consensus provided the scientific basis for defining the conditions under which breastfeeding can be used safely and effectively for birth-spacing purposes; programmatic guidelines were developed at a meeting of family planning experts for its use as a method of family planning, and the method was then given the name the lactational amenorrhea method (*1*,*2*). These guidelines include the following three criteria, all of which must be met to ensure adequate protection from an unplanned pregnancy: 1) amenorrhea; 2) fully or nearly fully breastfeeding (no interval of >4–6 hours between breastfeeds); and 3) <6 months postpartum.

All major medical organizations recommend exclusive breastfeeding for the first 6 months of life, with continuing breastfeeding through the first year and beyond for as long as mutually desired (*3*). No medical conditions exist for which use of the lactational amenorrhea method for contraception is restricted. However, breastfeeding might not be recommended for women or infants with certain conditions.

Women with conditions that make pregnancy an unacceptable risk should be advised that the lactational amenorrhea method might not be appropriate for them because of its relatively higher typical-use failure rates. The lactational amenorrhea method does not protect against sexually transmitted diseases (STDs), including human immunodeficiency virus (HIV), and women using this method should be counseled that consistent and correct use of the male latex condom reduces the risk for transmission of HIV and other STDs. Use of female condoms can provide protection from transmission of STDs, although data are limited.

HIV Infection

HIV can be transmitted from mother to infant through breastfeeding. Therefore, in the United States, where replacement feeding is affordable, feasible, acceptable, sustainable, and safe, breastfeeding for women with HIV is not recommended (*3*,*4*).

Other Medical Conditions

The American Academy of Pediatrics (AAP) also recommends against breastfeeding for women with active untreated tuberculosis disease, untreated brucellosis, varicella, H1N1 influenza, or positivity for human T-cell lymphotropic virus types I or II or for those who have herpes simplex lesions on a breast. In addition, infants with classic galactosemia should not breastfeed (*3*).

Medication Used During Breastfeeding

AAP recommends that the benefits of breastfeeding outweigh the risk of exposure to most therapeutic agents via human milk. More information about specific drugs and radioactive compounds is provided by AAP (*5*) and LactMed (http://toxnet.nlm.nih.gov).

References

1. Kennedy KI, Rivera R, McNeilly AS. Consensus statement on the use of breastfeeding as a family planning method. Contraception 1989;39:477–96. http://dx.doi.org/10.1016/0010-7824(89)90103-0
2. Labbok M, Cooney K, Coly S. Guidelines: breastfeeding, family planning, and the Lactational Amenorrhea Method-LAM. Washington, DC: Institute for Reproductive Health; 1994.
3. American Academy of Pediatrics Section on Breastfeeding. Breastfeeding and the use of human milk [Policy statement]. Pediatrics 2012;129:e827–41. http://dx.doi.org/10.1542/peds.2011-3552
4. Perinatal HIV Guidelines Working Group. Public Service Task Force recommendations for use of antiretroviral drugs in pregnant HIV-infected women for maternal health and interventions to reduce perinatal HIV transmission in the United States. Rockville, MD: Public Health Service Task Force; 2009.
5. Sachs HC; Committee On Drugs. The transfer of drugs and therapeutics into human breast milk: an update on selected topics. Pediatrics 2013;132:e796–809. http://dx.doi.org/10.1542/peds.2013-1985

Appendix H

Coitus Interruptus (Withdrawal)

Coitus interruptus, also known as withdrawal, is a traditional family planning method in which the man completely removes his penis from the vagina and away from the external genitalia of the female partner before he ejaculates. Coitus interruptus prevents sperm from entering the woman's vagina, thereby preventing contact between spermatozoa and the ovum.
This method might be appropriate for couples

- who are highly motivated and able to use this method effectively;
- with religious or philosophical reasons for not using other methods of contraception;
- who need contraception immediately and have entered into a sexual act without alternative methods available;
- who need a temporary method while awaiting the start of another method; or
- who have intercourse infrequently.

Some benefits of coitus interruptus are that the method, if used correctly, does not affect breastfeeding and is always available for primary use or use as a back-up method. In addition, coitus interruptus involves no economic cost or use of chemicals and has no directly associated health risks. Coitus interruptus does not protect against sexually transmitted diseases (STDs), including human immunodeficiency virus (HIV), and women using this method should be counseled that consistent and correct use of the male latex condom reduces the risk for transmission of HIV and other STDs. Use of female condoms can provide protection from transmission of STDs, although data are limited.

Coitus interruptus is unforgiving of incorrect use, and its effectiveness depends on the willingness and ability of the couple to use withdrawal with every act of intercourse. Women with conditions that make pregnancy an unacceptable risk should be advised that coitus interruptus might not be appropriate for them because of its relatively higher typical-use failure rates.

Appendix I

Female and Male Sterilization

Tubal sterilization for women and vasectomy for men are permanent, safe, and highly effective methods of contraception. In general, no medical conditions absolutely restrict a person's eligibility for sterilization (with the exception of known allergy or hypersensitivity to any materials used to complete the sterilization method). However, certain conditions place a woman at high surgical risk; in these cases, careful consideration should be given to the risks and benefits of other acceptable alternatives, including long-acting, highly effective, reversible methods and vasectomy. Female and male sterilization do not protect against sexually transmitted diseases (STDs), including human immunodeficiency virus (HIV), and women using these methods should be counseled that consistent and correct use of the male latex condom reduces the risk for transmission of HIV and other STDs. Use of female condoms can provide protection from transmission of STDs, although data are limited.

Because these methods are intended to be irreversible, persons who choose sterilization should be certain that they want to prevent pregnancy permanently. Most persons who choose sterilization remain satisfied with their decision. However, a small proportion of women regret this decision (1%–26% from different studies, with higher rates of regret reported by women who were younger at sterilization) (*1*,*2*). Regret among men about vasectomy has been reported to be approximately 5% (*3*), similar to the proportion of women who report regretting their husbands' vasectomy (6%) (*4*). Therefore, all persons should be appropriately counseled about the permanency of sterilization and the availability of highly effective, reversible methods of contraception.

References

1. Hillis SD, Marchbanks PA, Tylor LR, Peterson HB. Poststerilization regret: findings from the United States Collaborative Review of Sterilization. Obstet Gynecol 1999;93:889–95.
2. Peterson HB. Sterilization. Obstet Gynecol 2008;111:189–203. http://dx.doi.org/10.1097/01.AOG.0000298621.98372.62
3. Ehn BE, Liljestrand J. A long-term follow-up of 108 vasectomized men. Good counselling routines are important. Scand J Urol Nephrol 1995;29:477–81. http://dx.doi.org/10.3109/00365599509180030
4. Jamieson DJ, Kaufman SC, Costello C, Hillis SD, Marchbanks PA, Peterson HB; US Collaborative Review of Sterilization Working Group. A comparison of women's regret after vasectomy versus tubal sterilization. Obstet Gynecol 2002;99:1073–9.

Appendix J

Classifications for Emergency Contraception

A copper-containing intrauterine device (Cu-IUD) can be used within 5 days of unprotected intercourse as an emergency contraceptive. However, when the time of ovulation can be estimated, the Cu-IUD can be inserted beyond 5 days after intercourse, if necessary, as long as the insertion does not occur >5 days after ovulation. The eligibility criteria for interval Cu-IUD insertion also apply for the insertion of Cu-IUDs as emergency contraception (Box J1) (Table J1).

Classifications for emergency contraceptive pills (ECPs) are given for ulipristal acetate (UPA), levonorgestrel (LNG), and combined oral contraceptives (COCs). Cu-IUDs, UPA, LNG, and COCs do not protect against sexually transmitted diseases (STDs), including human immunodeficiency virus (HIV), and women using these methods should be counseled that consistent and correct use of the male latex condom reduces the risk for transmission of HIV and other STDs. Use of female condoms can provide protection from transmission of STDs, although data are limited.

BOX J1. Categories for classifying emergency contraception

1 = A condition for which there is no restriction for the use of the contraceptive method.
2 = A condition for which the advantages of using the method generally outweigh the theoretical or proven risks.
3 = A condition for which the theoretical or proven risks usually outweigh the advantages of using the method.
4 = A condition that represents an unacceptable health risk if the contraceptive method is used.

TABLE J1. Classifications for emergency contraception, including the copper-containing intrauterine device, ulipristal acetate, levonorgestrel, and combined oral contraceptives*

Condition	Category				Clarifications/Evidence/Comments
	Cu-IUD	UPA	LNG	COC	
Personal Characteristics and Reproductive History					
Pregnancy	4	NA	NA	NA	**Clarification (IUD):** The IUD is not indicated during pregnancy and should not be used because of the risk for serious pelvic infection and septic spontaneous abortion. **Clarification (ECPs):** Although this method is not indicated for a woman with a known or suspected pregnancy, no harm to the woman, the course of her pregnancy, or the fetus if ECPs are inadvertently used is known to exist. **Evidence:** Evidence suggests that poor pregnancy outcomes are rare among pregnant women who used ECPs during conception cycle or early in pregnancy *(1)*.
Breastfeeding	1	1	1	1	**Clarification (UPA):** Breastfeeding is not recommended for 24 hours after taking UPA because it is excreted in breast milk, with highest concentrations in the first 24 hours, and maximum maternal serum levels are reached 1–3 hours after administration. Mean UPA concentrations in breast milk decrease markedly from 0 to 24–48 hours and then slowly decrease over 5 days *(2)*. Breast milk should be expressed and discarded for 24 hours after taking UPA. **Evidence:** Breastfeeding outcomes do not seem to differ between women exposed to LNG and those who are not exposed. One pharmacokinetic study demonstrated that LNG passes to breast milk but in minimal quantities *(1)*.
Past ectopic pregnancy	1	1	1	1	—
History of bariatric surgery This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).					
a. Restrictive procedures: decrease storage capacity of the stomach (vertical banded gastroplasty, laparoscopic adjustable gastric band, or laparoscopic sleeve gastrectomy)	1	1	1	1	—
b. Malabsorptive procedures: decrease absorption of nutrients and calories by shortening the functional length of the small intestine (Roux-en-Y gastric bypass or biliopancreatic diversion)	1	1	1	1	**Comment:** Bariatric surgical procedures involving a malabsorptive component have the potential to decrease oral contraceptive effectiveness, perhaps further decreased by postoperative complications such as long-term diarrhea, vomiting, or both. Because of these malabsorptive concerns, an emergency IUD might be more appropriate than ECPs.

See table footnotes on page 94.

TABLE J1. (*Continued*) Classifications for emergency contraception, including the copper-containing intrauterine device, ulipristal acetate, levonorgestrel, and combined oral contraceptives*

Condition	Category				Clarifications/Evidence/Comments
	Cu-IUD	UPA	LNG	COC	
Cardiovascular Disease					
History of severe cardiovascular disease (ischemic heart disease, cerebrovascular attack, or other thromboembolic conditions) This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	1	2	2	2	**Comment:** The duration of ECP use is less than that of regular use of COCs or POPs and thus would be expected to have less clinical impact.
Rheumatic Diseases					
Rheumatoid arthritis					
a. Receiving immunosuppressive therapy	2	1	1	1	—
b. Not receiving immunosuppressive therapy	1	1	1	1	—
Neurologic Conditions					
Migraine	1	1	1	2	**Comment:** The duration of ECP use is less than that of regular use of COCs and thus would be expected to have less clinical impact.
Gastrointestinal Conditions					
Inflammatory bowel disease (ulcerative colitis or Crohn's disease)	1	1	1	1	—
Severe liver disease (including jaundice) This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	1	2	2	2	**Comment:** The duration of ECP use is less than that of regular use of COCs or POPs and thus would be expected to have less clinical impact.
Solid Organ Transplantation					
Solid organ transplantation This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).					
a. Complicated: graft failure (acute or chronic), rejection, or cardiac allograft vasculopathy	3	1	1	1	—
b. Uncomplicated	2	1	1	1	—
Other					
Repeated ECP use	1	1	1	1	**Clarification:** Recurrent ECP use is an indication that the woman requires further counseling about other contraceptive options. Frequently repeated ECP use might be harmful for women with conditions classified as 2, 3, or 4 for CHC or POC use. **Evidence:** In one case-control study, risk for ectopic pregnancy compared with intrauterine pregnancy did not increase after repeated use of LNG ECPs compared with nonuse *(1)*.
Sexual assault	2	1	1	1	**Clarification (IUD):** Women who have experienced sexual assault are at increased risk for STDs. According to CDC STD treatment guidelines, routine presumptive treatment of chlamydia, gonorrhea, and trichomonas is recommended after sexual assault *(3)*. Women with current purulent cervicitis or chlamydial infection or gonococcal infection should not undergo IUD insertion (category 4).
Obesity (BMI ≥30 kg/m^2)	1	2	2	2	**Clarification (ECPs):** ECPs might be less effective among women with BMI ≥30 kg/m^2 than among women with BMI <25 kg/m^2. Despite this, no safety concerns exist. **Evidence:** Limited evidence from secondary data analyses suggests that women with BMI ≥30 kg/m^2 experience an increased risk for pregnancy after use of LNG compared with women with BMI <25 kg/m^2. Two analyses suggest obese women might also experience an increased risk for pregnancy after use of UPA compared with nonobese women, although this increase was not significant in one study *(4)*.
CYP3A4 inducers (e.g., bosentan, carbamazepine, felbamate, griseofulvin, oxcarbazepine, phenytoin, rifampin, St. John's wort, topiramate, efavirenz, and lumacaftor)	1	2	2	2	**Clarification (ECPs):** Strong CYP3A4 inducers might reduce the effectiveness of ECPs. **Evidence:** According to labelling information, rifampin markedly decreases UPA levels by ≥90%, which might decrease its efficacy *(2)*. Therefore, theoretical concerns extend to use of other CYP3A4 inducers as well as to COC and LNG ECPs, which have metabolic pathways similar to those of UPA. A small pharmacokinetic study found that concomitant efavirenz use decreased LNG levels in women taking LNG ECPs (0.75 mg) by 56% compared with LNG ECPs alone *(5)*.

Abbreviations: BMI = body mass index; CHC = combined hormonal contraceptive; COC = combined hormonal contraceptive; Cu-IUD = copper-containing intrauterine device; ECP = emergency contraceptive pill; HIV = human immunodeficiency virus; IUD = intrauterine device; LNG = levonorgestrel; NA = not applicable; POC = progestin-only contraceptive; POP = progestin-only pill; STD = sexually transmitted disease; UPA = ulipristal acetate.

References

1. Jatlaoui TC, Riley H, Curtis KM. Safety data for levonorgestrel, ulipristal acetate and Yuzpe regimens for emergency contraception. Contraception 2016;93:93–112. http://dx.doi.org/10.1016/j.contraception.2015.11.001
2. Watson Pharmaceuticals. Ella [Prescribing information]. Morristown, NJ; 2010. https://www.accessdata.fda.gov/drugsatfda_docs/label/2010/022474s000lbl.pdf
3. Workowski KA, Bolan GA. Sexually transmitted diseases treatment guidelines, 2015. MMWR Recomm Rep 2015;64(No. RR-03).
4. Jatlaoui TC, Curtis KM. Safety and effectiveness data for emergency contraceptive pills among women with obesity: a systematic review. Contraception 2016. Epub May 24, 2016. http://dx.doi.org/10.1016/j.contraception.2016.05.002
5. Carten ML, Kiser JJ, Kwara A, Mawhinney S, Cu-Uvin S. Pharmacokinetic interactions between the hormonal emergency contraception, levonorgestrel (Plan B), and efavirenz. Infect Dis Obstet Gynecol 2012;2012:137192.

Appendix K

Summary of Classifications for Hormonal Contraceptive Methods and Intrauterine Devices

Health-care providers can use the summary table as a quick reference guide to the classifications for hormonal contraceptive methods and intrauterine contraception to compare classifications across these methods (Box K1) (Table K1). See the respective appendix for each method for clarifications to the numeric categories, as well as for summaries of the evidence and additional comments. Hormonal contraceptives and intrauterine devices do not protect against sexually transmitted diseases (STDs), including human immunodeficiency virus (HIV), and women using these methods should be counseled that consistent and correct use of the male latex condom reduces the risk for transmission of HIV and other STDs. Use of female condoms can provide protection from transmission of STDs, although data are limited.

BOX K1. Categories for classifying hormonal contraceptives and intrauterine devices

1 = A condition for which there is no restriction for the use of the contraceptive method.
2 = A condition for which the advantages of using the method generally outweigh the theoretical or proven risks.
3 = A condition for which the theoretical or proven risks usually outweigh the advantages of using the method.
4 = A condition that represents an unacceptable health risk if the contraceptive method is used.

TABLE K1. Summary of classifications for hormonal contraceptive methods and intrauterine devices

Condition	Cu-IUD	LNG-IUD	Implants	DMPA	POP	CHCs
Personal Characteristics And Reproductive History						
Pregnancy	4*	4*	NA*	NA*	NA*	NA*
Age	Menarche to <20 years: 2 ≥20 years: 1	Menarche to <20 years: 2 ≥20 years: 1	Menarche to <18 years: 1 18–45 years: 1 >45 years: 1	Menarche to <18 years: 2 18–45 years: 1 >45 years: 2	Menarche to <18 years: 1 18–45 years: 1 >45 years: 1	Menarche to <40 years: 1 ≥40 years: 2
Parity						
a. Nulliparous	2	2	1	1	1	1
b. Parous	1	1	1	1	1	1
Breastfeeding						
a. <21 days postpartum	—	—	2*	2*	2*	4*
b. 21 to <30 days postpartum						
i. With other risk factors for VTE (e.g., age ≥35 years, previous VTE, thrombophilia, immobility, transfusion at delivery, peripartum cardiomyopathy, BMI ≥30 kg/m^2, postpartum hemorrhage, postcesarean delivery, preeclampsia, or smoking)	—	—	2*	2*	2*	3*
ii. Without other risk factors for VTE	—	—	2*	2*	2*	3*
c. 30–42 days postpartum						
i. With other risk factors for VTE (e.g., age ≥35 years, previous VTE, thrombophilia, immobility, transfusion at delivery, peripartum cardiomyopathy, BMI ≥30 kg/m^2, postpartum hemorrhage, postcesarean delivery, preeclampsia, or smoking)	—	—	1*	1*	1*	3*
ii. Without other risk factors for VTE	—	—	1*	1*	1*	2*
d. >42 days postpartum	—	—	1*	1*	1*	2*

See table footnotes on page 103.

TABLE K1. (*Continued*) Summary of classifications for hormonal contraceptive methods and intrauterine devices

Condition	Cu-IUD	LNG-IUD	Implants	DMPA	POP	CHCs
Postpartum (nonbreastfeeding women)						
a. <21 days postpartum	—	—	1	1	1	4
b. 21–42 days postpartum						
i. With other risk factors for VTE (e.g., age ≥35 years, previous VTE, thrombophilia, immobility, transfusion at delivery, peripartum cardiomyopathy, BMI ≥30 kg/m^2, postpartum hemorrhage, postcesarean delivery, preeclampsia, or smoking)	—	—	1	1	1	3*
ii. Without other risk factors for VTE	—	—	1	1	1	2
c. >42 days postpartum	—	—	1	1	1	1
Postpartum (including cesarean delivery)						
a. <10 minutes after delivery of the placenta						
i. Breastfeeding	1*	2*	—	—	—	—
ii. Nonbreastfeeding	1*	1*	—	—	—	—
b. 10 minutes after delivery of the placenta to <4 weeks (breastfeeding or nonbreastfeeding)	2*	2*	—	—	—	—
c. ≥4 weeks (breastfeeding or nonbreastfeeding)	1*	1*	—	—	—	—
d. Postpartum sepsis	4	4	—	—	—	—
Postabortion						
a. First trimester	1*	1*	1*	1*	1*	1*
b. Second trimester	2*	2*	1*	1*	1*	1*
c. Immediate postseptic abortion	4	4	1*	1*	1*	1*
Past ectopic pregnancy	1	1	1	1	2	1
History of pelvic surgery (see Postpartum [Including Cesarean Delivery] section)	1	1	1	1	1	1
Smoking						
a. Age <35 years	1	1	1	1	1	2
b. Age ≥35 years						
i. <15 cigarettes per day	1	1	1	1	1	3
ii. ≥15 cigarettes per day	1	1	1	1	1	4
Obesity						
a. BMI ≥30 kg/m^2	1	1	1	1	1	2
b. Menarche to <18 years and BMI ≥30 kg/m^2	1	1	1	2	1	2
History of bariatric surgery This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).						
a. Restrictive procedures: decrease storage capacity of the stomach (vertical banded gastroplasty, laparoscopic adjustable gastric band, or laparoscopic sleeve gastrectomy)	1	1	1	1	1	1
b. Malabsorptive procedures: decrease absorption of nutrients and calories by shortening the functional length of the small intestine (Roux-en-Y gastric bypass or biliopancreatic diversion)	1	1	1	1	3	COCs: 3 Patch and ring: 1
Cardiovascular Disease						
Multiple risk factors for atherosclerotic cardiovascular disease (e.g., older age, smoking, diabetes, hypertension, low HDL, high LDL, or high triglyceride levels)	1	2	2*	3*	2*	3/4*
Hypertension Systolic blood pressure ≥160 mm Hg or diastolic blood pressure ≥100 mm Hg are associated with increased risk for adverse health events as a result of pregnancy (Box 2).						
a. Adequately controlled hypertension	1*	1*	1*	2*	1*	3*

See table footnotes on page 103.

TABLE K1. (*Continued*) Summary of classifications for hormonal contraceptive methods and intrauterine devices

Condition	Cu-IUD	LNG-IUD	Implants	DMPA	POP	CHCs
b. Elevated blood pressure levels (properly taken measurements)						
i. Systolic 140–159 mm Hg or diastolic 90–99 mm Hg	1*	1*	1*	2*	1*	3*
ii. Systolic ≥160 mm Hg or diastolic ≥100 mm Hg	1*	2*	2*	3*	2*	4*
c. Vascular disease	1*	2*	2*	3*	2*	4*
History of high blood pressure during pregnancy (when current blood pressure is measurable and normal)	1	1	1	1	1	2
Deep venous thrombosis/ Pulmonary embolism						
a. History of DVT/PE, not receiving anticoagulant therapy						
i. Higher risk for recurrent DVT/PE (one or more risk factors) • History of estrogen-associated DVT/PE • Pregnancy-associated DVT/PE • Idiopathic DVT/PE • Known thrombophilia, including antiphospholipid syndrome • Active cancer (metastatic, receiving therapy, or within 6 months after clinical remission), excluding nonmelanoma skin cancer • History of recurrent DVT/PE	1	2	2	2	2	4
ii. Lower risk for recurrent DVT/PE (no risk factors)	1	2	2	2	2	3
b. Acute DVT/PE	2	2	2	2	2	4
c. DVT/PE and established anticoagulant therapy for at least 3 months						
i. Higher risk for recurrent DVT/PE (one or more risk factors) • Known thrombophilia, including antiphospholipid syndrome • Active cancer (metastatic, receiving therapy, or within 6 months after clinical remission), excluding nonmelanoma skin cancer • History of recurrent DVT/PE	2	2	2	2	2	4*
ii. Lower risk for recurrent DVT/PE (no risk factors)	2	2	2	2	2	3*
d. Family history (first-degree relatives)	1	1	1	1	1	2
e. Major surgery						
i. With prolonged immobilization	1	2	2	2	2	4
ii. Without prolonged immobilization	1	1	1	1	1	2
f. Minor surgery without immobilization	1	1	1	1	1	1
Known thrombogenic mutations (e.g., factor V Leiden; prothrombin mutation; and protein S, protein C, and antithrombin deficiencies) This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	1*	2*	2*	2*	2*	4*
Superficial venous disorders						
a. Varicose veins	1	1	1	1	1	1
b. Superficial venous thrombosis (acute or history)	1	1	1	1	1	3*
Current and history of ischemic heart disease This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	1	Initiation 2 / Continuation 3	Initiation 2 / Continuation 3	3	Initiation 2 / Continuation 3	4

See table footnotes on page 103.

TABLE K1. (*Continued*) Summary of classifications for hormonal contraceptive methods and intrauterine devices

Condition	Cu-IUD	LNG-IUD	Implants		DMPA	POP		CHCs
			Initiation	Continuation		Initiation	Continuation	
Stroke (history of cerebrovascular accident) This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	1	2	2	3	3	2	3	4
Valvular heart disease Complicated valvular heart disease is associated with increased risk for adverse health events as a result of pregnancy (Box 2).								
a. Uncomplicated	1	1	1		1	1		2
b. Complicated (pulmonary hypertension, risk for atrial fibrillation, or history of subacute bacterial endocarditis)	1	1	1		1	1		4
Peripartum cardiomyopathy This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).								
a. Normal or mildly impaired cardiac function (New York Heart Association Functional Class I or II: patients with no limitation of activities or patients with slight, mild limitation of activity) *(1)*								
i. <6 months	2	2	1		1	1		4
ii. ≥6 months	2	2	1		1	1		3
b. Moderately or severely impaired cardiac function (New York Heart Association Functional Class III or IV: patients with marked limitation of activity or patients who should be at complete rest) *(1)*.	2	2	2		2	2		4
Rheumatic Diseases								
Systemic lupus erythematosus This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	Initiation	Continuation			Initiation	Continuation		
a. Positive (or unknown) antiphospholipid antibodies	1*	1*	3*		3*	3*	3*	4*
b. Severe thrombocytopenia	3*	2*	2*		2*	3*	2*	2*
c. Immunosuppressive therapy	2*	1*	2*		2*	2*	2*	2*
d. None of the above	1*	1*	2*		2*	2*	2*	2*
Rheumatoid arthritis	Initiation	Continuation	Initiation	Continuation				
a. Receiving immunosuppressive therapy	2	1	2	1	1	2/3*	1	2
b. Not receiving immunosuppressive therapy	1		1		1	2	1	2
Neurologic Conditions								
Headaches								
a. Nonmigraine (mild or severe)	1	1	1		1	1		1*
b. Migraine								
i. Without aura (This category of migraine includes menstrual migraine.)	1	1	1		1	1		2*
ii. With aura	1	1	1		1	1		4*
Epilepsy This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	1	1	1*		1*	1*		1*
Multiple sclerosis								
a. With prolonged immobility	1	1	1		2	1		3
b. Without prolonged immobility	1	1	1		2	1		1
Depressive Disorders								
Depressive disorders	1*	1*	1*		1*	1*		1*
Reproductive Tract Infections and Disorders								
Vaginal bleeding patterns		Initiation	Continuation					
a. Irregular pattern without heavy bleeding	1	1	1	2	2	2		1
b. Heavy or prolonged bleeding (includes regular and irregular patterns)	2*	1*	2*	2*	2*	2*		1*

See table footnotes on page 103.

TABLE K1. (*Continued*) Summary of classifications for hormonal contraceptive methods and intrauterine devices

Condition	Cu-IUD		LNG-IUD		Implants	DMPA	POP	CHCs
Unexplained vaginal bleeding (suspicious for serious condition) before evaluation	Initiation: 4*	Continuation: 2*	Initiation: 4*	Continuation: 2*	3*	3*	2*	2*
Endometriosis	2		1		1	1	1	1
Benign ovarian tumors (including cysts)	1		1		1	1	1	1
Severe dysmenorrhea	2		1		1	1	1	1
Gestational trophoblastic disease This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).								
a. Suspected gestational trophoblastic disease (immediate postevacuation)								
i. Uterine size first trimester	1*		1*		1*	1*	1*	1*
ii. Uterine size second trimester	2*		2*		1*	1*	1*	1*
b. Confirmed gestational trophoblastic disease (after initial evacuation and during monitoring)	Initiation	Continuation	Initiation	Continuation				
i. Undetectable/nonpregnant β-hCG levels	1*	1*	1*	1*	1*	1*	1*	1*
ii. Decreasing β-hCG levels	2*	1*	2*	1*	1*	1*	1*	1*
iii. Persistently elevated β-hCG levels or malignant disease, with no evidence or suspicion of intrauterine disease	2*	1*	2*	1*	1*	1*	1*	1*
iv. Persistently elevated β-hCG levels or malignant disease, with evidence or suspicion of intrauterine disease	4*	2*	4*	2*	1*	1*	1*	1*
Cervical ectropion	1		1		1	1	1	1
Cervical intraepithelial neoplasia	1		2		2	2	1	2
Cervical cancer (awaiting treatment)	Initiation	Continuation	Initiation	Continuation				
	4	2	4	2	2	2	1	2
Breast disease Breast cancer is associated with increased risk of adverse health events as a result of pregnancy (Box 2).								
a. Undiagnosed mass	1		2		2*	2*	2*	2*
b. Benign breast disease	1		1		1	1	1	1
c. Family history of cancer	1		1		1	1	1	1
d. Breast cancer								
i. Current	1		4		4	4	4	4
ii. Past and no evidence of current disease for 5 years	1		3		3	3	3	3
Endometrial hyperplasia	1		1		1	1	1	1
Endometrial cancer This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	Initiation: 4	Continuation: 2	Initiation: 4	Continuation: 2	1	1	1	1
Ovarian cancer This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	1		1		1	1	1	1
Uterine fibroids	2		2		1	1	1	1
Anatomical abnormalities								
a. Distorted uterine cavity (any congenital or acquired uterine abnormality distorting the uterine cavity in a manner that is incompatible with IUD insertion)	4		4		—	—	—	—
b. Other abnormalities (including cervical stenosis or cervical lacerations) not distorting the uterine cavity or interfering with IUD insertion	2		2		—	—	—	—
Pelvic inflammatory disease								
a. Past PID	Initiation	Continuation	Initiation	Continuation				
i. With subsequent pregnancy	1	1	1	1	1	1	1	1
ii. Without subsequent pregnancy	2	2	2	2	1	1	1	1
b. Current PID	4	2*	4	2*	1	1	1	1

See table footnotes on page 103.

TABLE K1. (*Continued*) Summary of classifications for hormonal contraceptive methods and intrauterine devices

Condition	Cu-IUD		LNG-IUD		Implants	DMPA	POP	CHCs
Sexually transmitted diseases	Initiation	Continuation	Initiation	Continuation				
a. Current purulent cervicitis or chlamydial infection or gonococcal infection	4	2*	4	2*	1	1	1	1
b. Vaginitis (including *Trichomonas vaginalis* and bacterial vaginosis)	2	2	2	2	1	1	1	1
c. Other factors related to STDs	2*	2	2*	2	1	1	1	1
HIV								
	Initiation	Continuation	Initiation	Continuation				
High risk for HIV	2	2	2	2	1	1*	1	1
HIV infection For women with HIV infection who are not clinically well or not receiving ARV therapy, this condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	—	—	—	—	1*	1*	1*	1*
a. Clinically well receiving ARV therapy	1	1	1	1	—	—	—	—
b. Not clinically well or not receiving ARV therapy	2	1	2	1	—	—	—	—
Other Infections								
Schistosomiasis Schistosomiasis with fibrosis of the liver is associated with increased risk for adverse health events as a result of pregnancy (Box 2).								
a. Uncomplicated	1		1		1	1	1	1
b. Fibrosis of the liver (if severe, see Cirrhosis)	1		1		1	1	1	1
Tuberculosis This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	Initiation	Continuation	Initiation	Continuation				
a. Nonpelvic	1	1	1	1	1*	1*	1*	1*
b. Pelvic	4	3	4	3	1*	1*	1*	1*
Malaria	1		1		1	1	1	1
Endocrine Conditions								
Diabetes Insulin-dependent diabetes; diabetes with nephropathy, retinopathy, or neuropathy; diabetes with other vascular disease; or diabetes of >20 years' duration are associated with increased risk of adverse health events as a result of pregnancy (Box 2).								
a. History of gestational disease	1		1		1	1	1	1
b. Nonvascular disease								
i. Non-insulin dependent	1		2		2	2	2	2
ii. Insulin dependent	1		2		2	2	2	2
c. Nephropathy, retinopathy, or neuropathy	1		2		2	3	2	3/4*
d. Other vascular disease or diabetes of >20 years' duration	1		2		2	3	2	3/4*
Thyroid disorders								
a. Simple goiter	1		1		1	1	1	1
b. Hyperthyroid	1		1		1	1	1	1
c. Hypothyroid	1		1		1	1	1	1
Gastrointestinal Conditions								
Inflammatory bowel disease (ulcerative colitis or Crohn's disease)	1		1		1	2	2	2/3*
Gallbladder disease								
a. Symptomatic								
i. Treated by cholecystectomy	1		2		2	2	2	2
ii. Medically treated	1		2		2	2	2	3
iii. Current	1		2		2	2	2	3
b. Asymptomatic	1		2		2	2	2	2
History of cholestasis								
a. Pregnancy related	1		1		1	1	1	2
b. Past COC related	1		2		2	2	2	3

See table footnotes on page 103.

TABLE K1. (*Continued*) Summary of classifications for hormonal contraceptive methods and intrauterine devices

Condition	Cu-IUD	LNG-IUD	Implants	DMPA	POP	CHCs	
Viral hepatitis						Initiation	Continuation
a. Acute or flare	1	1	1	1	1	3/4*	2
b. Carrier	1	1	1	1	1	1	1
c. Chronic	1	1	1	1	1	1	1
Cirrhosis Severe cirrhosis is associated with increased risk for adverse health events as a result of pregnancy (Box 2).							
a. Mild (compensated)	1	1	1	1	1	1	
b. Severe (decompensated)	1	3	3	3	3	4	
Liver tumors Hepatocellular adenoma and malignant liver tumors are associated with increased risk for adverse health events as a result of pregnancy (Box 2).							
a. Benign							
i. Focal nodular hyperplasia	1	2	2	2	2	2	
ii. Hepatocellular adenoma	1	3	3	3	3	4	
b. Malignant (hepatoma)	1	3	3	3	3	4	
Respiratory Conditions							
Cystic fibrosis This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	1*	1*	1*	2*	1*	1*	
Anemias							
Thalassemia	2	1	1	1	1	1	
Sickle cell disease This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	2	1	1	1	1	2	
Iron-deficiency anemia	2	1	1	1	1	1	
Solid Organ Transplantation							
Solid organ transplantation This condition is associated with increased risk for adverse health events as a result of pregnancy (Box 2).	Initiation	Continuation	Initiation	Continuation			
a. Complicated: graft failure (acute or chronic), rejection, or cardiac allograft vasculopathy	3	2	3	2	2	2	2	4
b. Uncomplicated	2	2	2	2	2	2	2	2*
Drug Interactions							
Antiretroviral therapy	Initiation	Continuation	Initiation	Continuation			
a. Nucleoside reverse transcriptase inhibitors (NRTIs)							
i. Abacavir (ABC)	1/2*	1*	1/2*	1*	1	1	1	1
ii. Tenofovir (TDF)	1/2*	1*	1/2*	1*	1	1	1	1
iii. Zidovudine (AZT)	1/2*	1*	1/2*	1*	1	1	1	1
iv. Lamivudine (3TC)	1/2*	1*	1/2*	1*	1	1	1	1
v. Didanosine (DDI)	1/2*	1*	1/2*	1*	1	1	1	1
vi. Emtricitabine (FTC)	1/2*	1*	1/2*	1*	1	1	1	1
vii. Stavudine (D4T)	1/2*	1*	1/2*	1*	1	1	1	1
b. Nonnucleoside reverse transcriptase inhibitors (NNRTIs)							
i. Efavirenz (EFV)	1/2*	1*	1/2*	1*	2*	1*	2*	2*
ii. Etravirine (ETR)	1/2*	1*	1/2*	1*	1	1	1	1
iii. Nevirapine (NVP)	1/2*	1*	1/2*	1*	1	1	1	1
iv. Rilpivirine (RPV)	1/2*	1*	1/2*	1*	1	1	1	1
c. Ritonavir-boosted protease inhibitors							
i. Ritonavir-boosted atazanavir (ATV/r)	1/2*	1*	1/2*	1*	2*	1*	2*	2*
ii. Ritonavir-boosted darunavir (DRV/r)	1/2*	1*	1/2*	1*	2*	1*	2*	2*
iii. Ritonavir-boosted fosamprenavir (FPV/r)	1/2*	1*	1/2*	1*	2*	1*	2*	2*
iv. Ritonavir-boosted lopinavir (LPV/r)	1/2*	1*	1/2*	1*	1	1	1	1
v. Ritonavir-boosted saquinavir (SQV/r)	1/2*	1*	1/2*	1*	2*	1*	2*	2*
vi. Ritonavir-boosted tipranavir (TPV/r)	1/2*	1*	1/2*	1*	2*	1*	2*	2*

See table footnotes on page 103.

TABLE K1. (*Continued*) Summary of classifications for hormonal contraceptive methods and intrauterine devices

Condition	Cu-IUD		LNG-IUD		Implants	DMPA	POP	CHCs
d. Protease inhibitors without ritonavir								
i. Atazanavir (ATV)	1/2*	1*	1/2*	1*	1	1	1	2*
ii. Fosamprenavir (FPV)	1/2*	1*	1/2*	1*	2*	2*	2*	3*
iii. Indinavir (IDV)	1/2*	1*	1/2*	1*	1	1	1	1
iv. Nelfinavir (NFV)	1/2*	1*	1/2*	1*	2*	1*	2*	2*
e. CCR5 co-receptor antagonists								
i. Maraviroc (MVC)	1/2*	1*	1/2*	1*	1	1	1	1
f. HIV integrase strand transfer inhibitors								
i. Raltegravir (RAL)	1/2*	1*	1/2*	1*	1	1	1	1
ii. Dolutegravir (DTG)	1/2*	1*	1/2*	1*	1	1	1	1
iii. Elvitegravir (EVG)	1/2*	1*	1/2*	1*	1	1	1	1
g. Fusion inhibitors								
i. Enfuvirtide	1/2*	1*	1/2*	1*	1	1	1	1
Anticonvulsant therapy								
a. Certain anticonvulsants (phenytoin, carbamazepine, barbiturates, primidone, topiramate, and oxcarbazepine)	1		1		2*	1*	3*	3*
b. Lamotrigine	1		1		1	1	1	3*
Antimicrobial therapy								
a. Broad-spectrum antibiotics	1		1		1	1	1	1
b. Antifungals	1		1		1	1	1	1
c. Antiparasitics	1		1		1	1	1	1
d. Rifampin or rifabutin therapy	1		1		2*	1*	3*	3*
Psychotropic medications								
a. SSRIs	1		1		1	1	1	1
St. John's wort	1		1		2	1	2	2

Abbreviations: BMI = body mass index; COC = combined oral contraceptive; Cu-IUD = copper-containing IUD; DMPA = depot medroxyprogesterone acetate; DVT = deep venous thrombosis; hCG = human chorionic gonadotropin; HDL = high-density lipoprotein; HIV = human immunodeficiency virus.; IUD = intrauterine device; LDL = low-density lipoprotein; LNG-IUD = levonorgestrel-releasing IUD; NA = not applicable; PE = pulmonary embolism; PID = pelvic inflammatory disease; POP = progestin-only pill; SSRI = selective serotonin reuptake inhibitor; STD = sexually transmitted disease.
* Consult the appendix for this contraceptive method for a clarification to this classification.

References

1. The Criteria Committee of the New York Heart Association. Nomenclature and criteria for diagnosis of diseases of the heart and great vessels. 9th ed. Boston, MA: Little, Brown & Co; 1994.

The *Morbidity and Mortality Weekly Report (MMWR)* Series is prepared by the Centers for Disease Control and Prevention (CDC) and is available free of charge in electronic format. To receive an electronic copy each week, visit *MMWR*'s free subscription page at *http://www.cdc.gov/mmwr/mmwrsubscribe.html*. Paper copy subscriptions are available through the Superintendent of Documents, U.S. Government Printing Office, Washington, DC 20402; telephone 202-512-1800.

Readers who have difficulty accessing this PDF file may access the HTML file at http://www.cdc.gov/mmwr/volumes/65/rr/rr6503a1.htm?s_cid=rr6503a1_w. Address all inquiries about the *MMWR* Series, including material to be considered for publication, to Executive Editor, *MMWR* Series, Mailstop E-90, CDC, 1600 Clifton Rd., N.E., Atlanta, GA 30329-4027 or to *mmwrq@cdc.gov.*

ISSN: 1057-5987 (Print)

Centers for Disease Control and Prevention
MMWR
Morbidity and Mortality Weekly Report
Recommendations and Reports / Vol. 63 / No. 4
April 25, 2014

Providing Quality Family Planning Services

Recommendations of CDC and the U.S. Office of Population Affairs

Continuing Education Examination available at http://www.cdc.gov/mmwr/cme/conted.html.

U.S. Department of Health and Human Services
Centers for Disease Control and Prevention

CONTENTS

Disclosure of Relationship

CDC, our planners, content experts, and their spouses/partners wish to disclose that they have no financial interests or other relationships with the manufacturers of commercial products, suppliers of commercial services, or commercial supporters. Planners have reviewed content to ensure there is no bias.

The *MMWR* series of publications is published by the Center for Surveillance, Epidemiology, and Laboratory Services, Centers for Disease Control and Prevention (CDC), U.S. Department of Health and Human Services, Atlanta, GA 30329-4027.

Suggested Citation: [Author names; first three, then et al., if more than six.] [Title]. MMWR 2014;63(No. RR-#):[inclusive page numbers].

Providing Quality Family Planning Services

Recommendations of CDC and the U.S. Office of Population Affairs

Prepared by
Loretta Gavin, PhD,[1] Susan Moskosky, MS,[2] Marion Carter, PhD,[1] Kathryn Curtis, PhD,[1] Evelyn Glass, MSPH,[2] Emily Godfrey, MD,[1] Arik Marcell, MD,[3] Nancy Mautone-Smith, MSW,[2] Karen Pazol, PhD,[1] Naomi Tepper, MD,[1] Lauren Zapata, PhD[1]
[1]*Division of Reproductive Health, National Center for Chronic Disease Prevention and Health Promotion, CDC*
[2]*Office of Population Affairs, US Department of Health and Human Services, Rockville, Maryland*
[3]*The Johns Hopkins University and the Male Training Center for Family Planning and Reproductive Health, Baltimore, Maryland*

Summary

This report provides recommendations developed collaboratively by CDC and the Office of Population Affairs (OPA) of the U.S. Department of Health and Human Services (HHS). The recommendations outline how to provide quality family planning services, which include contraceptive services, pregnancy testing and counseling, helping clients achieve pregnancy, basic infertility services, preconception health services, and sexually transmitted disease services. The primary audience for this report is all current or potential providers of family planning services, including those working in service sites that are dedicated to family planning service delivery as well as private and public providers of more comprehensive primary care.

The United States continues to face substantial challenges to improving the reproductive health of the U.S. population. Nearly one half of all pregnancies are unintended, with more than 700,000 adolescents aged 15–19 years becoming pregnant each year and more than 300,000 giving birth. One of eight pregnancies in the United States results in preterm birth, and infant mortality rates remain high compared with those of other developed countries.

This report can assist primary care providers in offering family planning services that will help women, men, and couples achieve their desired number and spacing of children and increase the likelihood that those children are born healthy. The report provides recommendations for how to help prevent and achieve pregnancy, emphasizes offering a full range of contraceptive methods for persons seeking to prevent pregnancy, highlights the special needs of adolescent clients, and encourages the use of the family planning visit to provide selected preventive health services for women, in accordance with the recommendations for women issued by the Institute of Medicine and adopted by HHS.

Introduction

The United States continues to face challenges to improving the reproductive health of the U.S. population. Nearly half (49%) of all pregnancies are unintended (*1*). Although adolescent birth rates declined by more than 61% during 1991–2012, the United States has one of the highest adolescent pregnancy rates in the developed world, with >700,000 adolescents aged 15–19 years becoming pregnant each year and >300,000 giving birth (*2,3*). Approximately one of eight pregnancies in the United States results in a preterm birth, and infant mortality rates remain high compared with other developed countries (*3,4*). Moreover, all of these outcomes affect racial and ethnic minority populations disproportionately (*1–4*).

Corresponding preparers: Loretta Gavin, PhD, Division of Reproductive Health, National Center for Chronic Disease Prevention and Health Promotion, CDC. Telephone: 770-488-6284; E-mail: lcg6@cdc.gov; Susan Moskosky, MS, Office of Population Affairs, US Department of Health and Human Services. Telephone: 240-453-2818; E-mail: susan.moskosky@hhs.gov.

Family planning services can help address these and other public health challenges by providing education, counseling, and medical services (*5*). Family planning services include the following:

- providing contraception to help women and men plan and space births, prevent unintended pregnancies, and reduce the number of abortions;
- offering pregnancy testing and counseling;
- helping clients who want to conceive;
- providing basic infertility services;
- providing preconception health services to improve infant and maternal outcomes and improve women's and men's health; and
- providing sexually transmitted disease (STD) screening and treatment services to prevent tubal infertility and improve the health of women, men, and infants.

This report provides recommendations developed collaboratively by CDC and the Office of Population Affairs (OPA) of the U.S. Department of Health and Human Services (HHS). The recommendations outline how to provide family planning services by:

- defining a core set of family planning services for women and men,
- describing how to provide contraceptive and other clinical services, serve adolescents, and perform quality improvements, and
- encouraging the use of the family planning visit to provide selected preventive health services for women, in accordance with the recommendations for women issued by the Institute of Medicine (IOM) and adopted by HHS (*6*).

The collaboration between CDC and OPA drew on the strengths of both agencies. CDC has a long-standing history of developing evidence-based recommendations for clinical care, and OPA's Title X Family Planning Program (*7*) has served as the national leader in direct family planning service delivery since the Title X program was established in 1970.

This report provides recommendations for providing care to clients of reproductive age who are in need of family planning services. These recommendations are intended for all current or potential providers of family planning services, including those funded by the Title X program.

Current Context of Family Planning Services

Women of reproductive age often report that their family planning provider is also their usual source of health care (*8*). As the U.S. health-care system evolves in response to increased efforts to expand health insurance coverage, contain costs, and emphasize preventive care (*9*), providers of family planning services will face new challenges and opportunities in care delivery. For example, they will have increased opportunities to serve new clients and to serve as gateways for their clients to other essential health-care services. In addition, primary care and other providers who provide a range of health-care services will be expected to integrate family planning services for all persons of reproductive age, including those whose primary reason for their health-care visit might not be family planning. Strengthened, multidirectional care coordination also will be needed to improve health outcomes. For example, this type of care coordination will be needed with clients referred to specialist care after initial screening at a family planning visit, as well as with specialists referring clients with family planning needs to family planning providers.

Defining Quality in Family Planning Service Delivery

The central premise underpinning these recommendations is that improving the quality of family planning services will lead to improved reproductive health outcomes (*10*–*12*). IOM defines health-care quality as the extent to which health-care services improve health outcomes in a manner that is consistent with current professional knowledge (*10*,*13*). According to IOM, quality health care has the following attributes:

- **Safety.** These recommendations integrate other CDC recommendations about which contraceptive methods can be provided safely to women with various medical conditions, and integrate CDC and U.S. Preventive Services Task Force (USPSTF) recommendations on STD, preconception, and related preventive health services.
- **Effectiveness.** These recommendations support offering a full range of Food and Drug Administration (FDA)–approved contraceptive methods as well as counseling that highlights the effectiveness of contraceptive methods overall and, in specific patient situations, draws attention to the effectiveness of specific clinical preventive health services and identifies clinical preventive health services for which the potential harms outweigh the benefits (i.e., USPSTF "D" recommendations).
- **Client-centered approach.** These recommendations encourage taking a client-centered approach by 1) highlighting that the client's primary purpose for visiting the service site must be respected, 2) noting the importance of confidential services and suggesting ways to provide them, 3) encouraging the availability of a broad range of contraceptive methods so that clients can make a selection based on their individual needs and preferences, and 4) reinforcing the need to deliver services in a culturally competent manner so as to meet the needs of all clients, including adolescents, those with limited English proficiency, those with disabilities, and those who are lesbian, gay, bisexual, transgender, or questioning their sexual identity (LGBTQ). Organizational policies, governance structures, and individual attitudes and practices all contribute to the cultural competence of a health-care entity and its staff. Cultural competency within a health-care setting refers to attitudes, practices, and policies that enable professionals to work effectively in cross-cultural situations (*14*–*16*).
- **Timeliness.** These recommendations highlight the importance of ensuring that services are provided to clients in a timely manner.
- **Efficiency.** These recommendations identify a core set of services that providers can focus on delivering, as well as ways to maximize the use of resources.
- **Accessibility.** These recommendations address how to remove barriers to contraceptive use, use the family planning visit to provide access to a broader range of primary care and behavioral health services, use the primary care visit to

provide access to contraceptive and other family planning services, and strengthen links to other sources of care.
- **Equity.** These recommendations highlight the need for providers of family planning services to deliver high-quality care to all clients, including adolescents, LGBTQ persons, racial and ethnic minorities, clients with limited English proficiency, and persons living with disabilities.
- **Value.** These recommendations highlight services (i.e., contraception and other clinical preventive services) that have been shown to be very cost-effective (*17*–*19*).

Methods

Recommendations Development Process

The recommendations were developed jointly under the auspices of CDC's Division of Reproductive Health and OPA, in consultation with a wide range of experts and key stakeholders. More information about the processes used to conduct systematic reviews, the role of technical experts in reviewing the evidence, and the process of using the evidence to develop recommendations is provided (Appendix A). A multistage process was used to develop the recommendations that drew on established procedures for developing clinical guidelines (*20*,*21*). First, an Expert Work Group* was formed comprising family planning clinical providers, program administrators, and representatives from relevant federal agencies and professional medical associations to help define the scope of the recommendations. Next, literature about three priority topics (i.e., counseling and education, serving adolescents, and quality improvement) was reviewed by using the USPSTF methodology for conducting systematic reviews (*22*). The results were presented to three technical panels† comprising subject matter experts (one panel for each priority topic) who considered the quality of the evidence and made suggestions for what recommendations might be supported on the basis of the evidence. In a separate process, existing clinical recommendations on women's and men's preventive services were compiled from more than 35 federal and professional medical associations, and these results were presented to two technical panels of subject matter experts, one that addressed women's clinical services and one that addressed men's clinical services. The panels provided individual feedback about which clinical preventive services should be offered in a family planning setting and which clinical recommendations should receive the highest consideration.

CDC and OPA used the input from the subject matter experts to develop a set of core recommendations and asked the Expert Work Group to review them. The members of the Expert Work Group were more familiar with the family planning service delivery context than the members of the Technical Panel and thus could better comment on the feasibility and appropriateness of the recommendations, as well as the supporting evidence. The Expert Work Group considered the core recommendations by using the following criteria: 1) the quality of the evidence; 2) the positive and negative consequences of implementing the recommendations on health outcomes, costs or cost-savings, and implementation challenges; and 3) the relative importance of these consequences, (e.g., the likelihood that implementation of the recommendation will have a substantial effect on health outcomes might be considered more than the logistical challenges of implementing it) (*20*). In certain cases, when the evidence from the literature reviews was inconclusive or incomplete, recommendations were made on the basis of expert opinion. Finally, CDC and OPA staff considered the individual feedback from Expert Work Group members when finalizing the core recommendations and writing the recommendations document. A description of how the recommendations link to the evidence is provided together with the rationale for the inclusion of each recommendation in this report (Appendix B).

The evidence used to prepare these recommendations will appear in background papers that will be published separately. Resources that will help providers implement the recommendations will be provided through a web-based tool kit that will be available at http://www.hhs.gov/opa.

Audience for the Recommendations

The primary audience for this report is all providers or potential providers of family planning services to clients of reproductive age, including providers working in clinics that are dedicated to family planning service delivery, as well as private and public providers of more comprehensive primary care. Providers of dedicated family planning services might be less familiar with the specific recommendations for the delivery of preconception services. Providers of more comprehensive primary care might be less familiar with the delivery of contraceptive services, pregnancy testing and counseling, and services to help clients achieve pregnancy.

This report can be used by medical directors to write clinical protocols that describe how care should be provided. Job aids and other resources for use in service sites are being developed and will be made available when ready through OPA's website (http://www.hhs.gov/opa).

* A list of the members of the Expert Work Group appears on page 52.
† A list of the members of the technical panels appears on pages 52 and 53.

In this report, the term "provider" refers to any staff member who is involved in providing family planning services to a client. This includes physicians, physician assistants, nurse practitioners, nurse-midwives, nursing staff, and health educators. The term "service site" represents the numerous settings in which family planning services are delivered, which include freestanding service sites, community health centers, private medical facilities, and hospitals. A list of special terms used in this report is provided (Box 1).

The recommendations are designed to guide general clinical practice; however, health-care providers always should consider the individual clinical circumstances of each person seeking family planning services. Similarly, these recommendations might need to be adapted to meet the needs of particular populations, such as clients who are HIV-positive or who are substance users.

Organization of the Recommendations

This report is divided into nine sections. An initial section provides an overview of steps to assess the needs of a client and decide what family planning services to offer. Subsequent sections describe how to provide each of the following services: contraceptive services, pregnancy testing and counseling, helping clients achieve pregnancy, basic infertility services, preconception health services, STD services and related preventive health services. A final section on quality improvement describes actions that all providers of family planning services should consider to ensure that services are of high quality. More detailed information about selected topics addressed in the recommendations is provided (Appendices A–F).

These recommendations focus on the direct delivery of care to individual clients. However, parallel steps might need to be taken to maintain the systems required to support the provision of quality services for all clients (e.g., record-keeping procedures that preserve client confidentiality, procedures that improve efficiency and reduce clients' wait time, staff training to ensure that all clients are treated with respect, and the establishment and maintenance of a strong system of care coordination and referrals).

Client Care

Family planning services are embedded within a broader framework of preventive health services (Figure 1). In this report, health services are divided into three main categories:

- **Family planning services.** These include contraceptive services for clients who want to prevent pregnancy and space births, pregnancy testing and counseling, assistance to achieve pregnancy, basic infertility services, STD services (including HIV/AIDS), and other preconception health services (e.g., screening for obesity, smoking, and mental health). STD/HIV and other preconception health services are considered family planning services because they improve women's and men's health and can influence a person's ability to conceive or to have a healthy birth outcome.
- **Related preventive health services.** These include services that are considered to be beneficial to reproductive health,

BOX 1. Definitions of quality terms used in this report

Accessible. The timely use of personal health services to achieve the best possible health outcomes.*

Client-centered. Care is respectful of, and responsive to, individual client preferences, needs, and values; client values guide all clinical decisions.†

Effective. Services are based on scientific knowledge and provided to all who could benefit and are not provided to those not likely to benefit.†

Efficient. Waste is avoided, including waste of equipment, supplies, ideas, and energy.†

Equitable. Care does not vary in quality because of the personal characteristics of clients (e.g., sex, race/ethnicity, geographic location, insurance status, or socioeconomic status).†

Evidence-based. The process of integrating science-based interventions with community preferences to improve the health of populations.§

Health-care quality. The degree to which health-care services for individuals and populations increase the likelihood of desired health outcomes and are consistent with current professional knowledge.†

Process. Whether services are provided correctly and completely and how clients perceive the care they receive.¶

Safe. Avoids injuries to clients from the care that is intended to help them.†

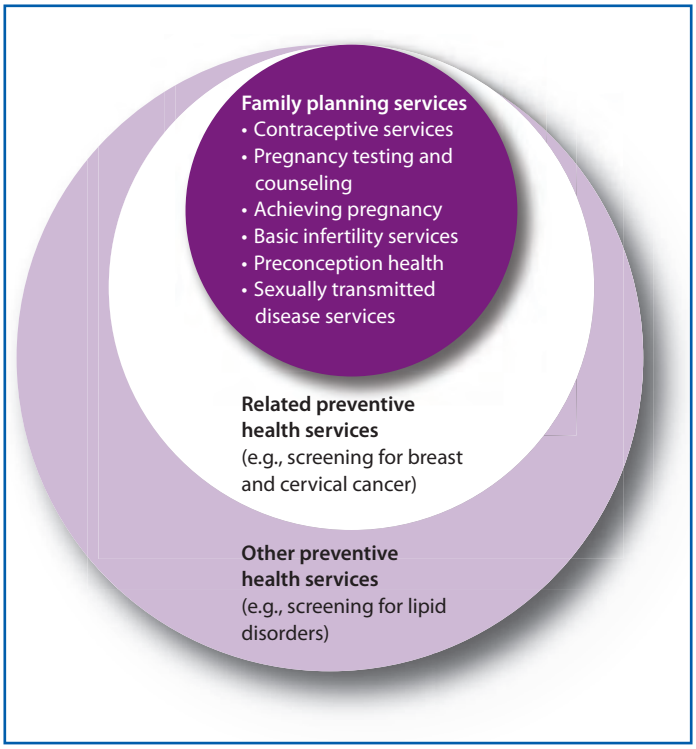
Structure. The characteristics of the settings in which providers deliver health care, including material resources, human resources, and organizational structure.¶

Timely. Waits and sometimes harmful delays for both those who receive and those who provide care are reduced.†

Value. The care provides good return relative to the costs involved, such as a return on investment or a reduction in the per capita cost of health care.*

* Source: Institute of Medicine. Future directions for the national healthcare quality and disparities reports. Ulmer C, Bruno M, Burke S, eds. Washington, DC: The National Academies Press; 2010.
† Source: Institute of Medicine. Crossing the quality chasm: a new health system for the 21st century. Committee on Quality of Health Care in America, ed. Washington, DC: National Academies of Science; 2001.
§ Source: Kohatsu ND, Robinson JG, Torner JC. Evidence-based public health: an evolving concept. Am J Prev Med 2004;27:417–21.
¶ Source: Donabedian A. The quality of care. JAMA 1988;260:1743–8.

FIGURE 1. Family planning and related and other preventive health services



are closely linked to family planning services, and are appropriate to deliver in the context of a family planning visit but that do not contribute directly to achieving or preventing pregnancy (e.g., breast and cervical cancer screening).

- **Other preventive health services.** These include preventive health services for women that were not included above (*6*), as well as preventive services for men. Screening for lipid disorders, skin cancer, colorectal cancer, or osteoporosis are examples of this type of service. Although important in the context of primary care, these have no direct link to family planning services.

Providers of family planning services should be trained and equipped to offer all family planning and related preventive health services so that they can provide optimal care to clients, with referral for specialist care, as needed. Other preventive health services should be available either on-site or by referral, but these recommendations do not address this category of services. Information about preventive services that are beyond the scope of this report is available at http://www.uspreventiveservicestaskforce.org.

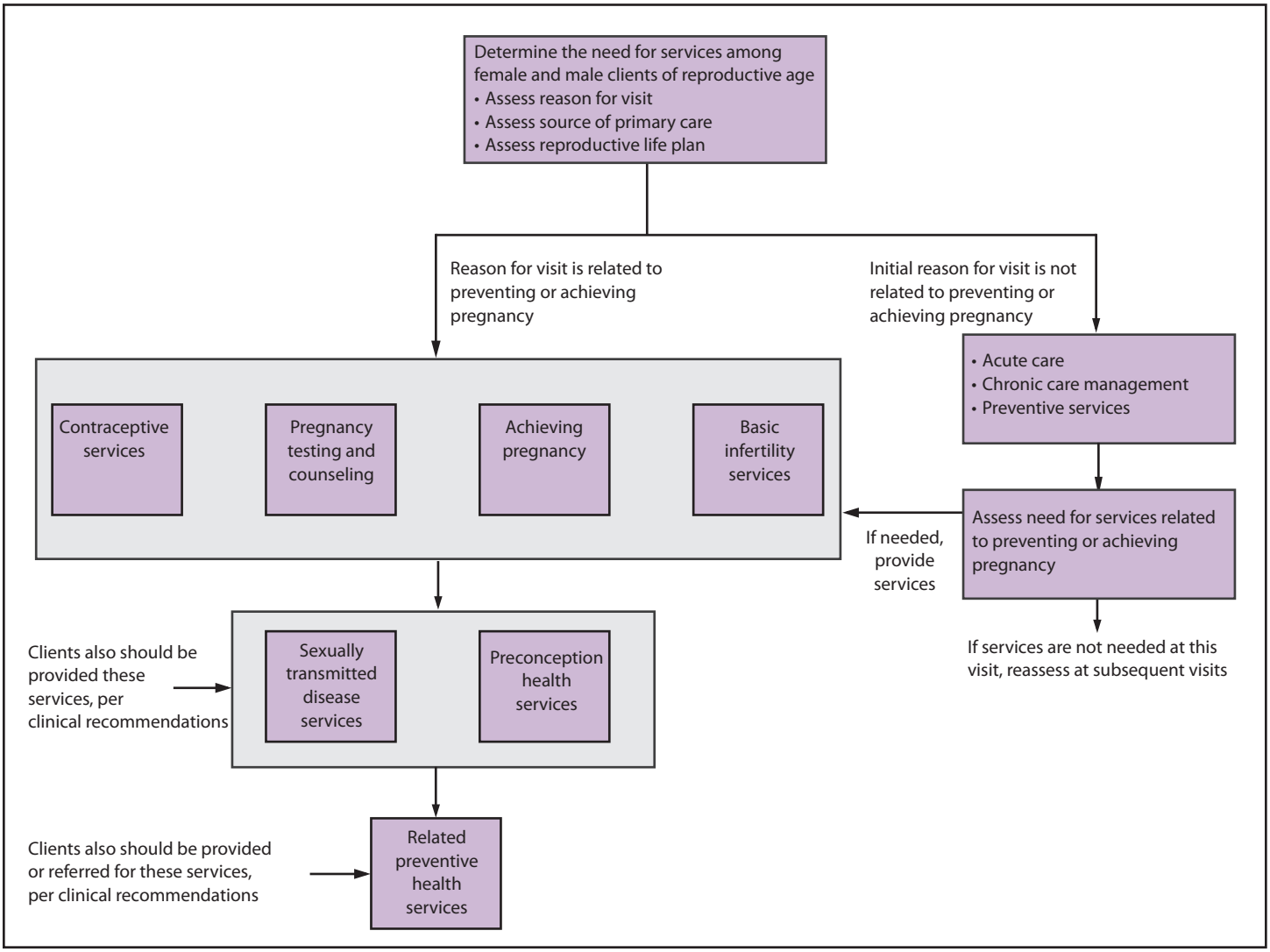
Determining the Client's Need for Services

These recommendations apply to two types of encounters with women and men of reproductive age. In the first type of encounter, the primary reason for a client's visit to a health-care provider is related to preventing or achieving pregnancy, (i.e., contraceptive services, pregnancy testing and counseling, or becoming pregnant). Other aspects of managing pregnancy (e.g., prenatal and delivery care) are not addressed in these recommendations. For clients seeking to prevent or achieve pregnancy, providers should assess whether the client needs other related services and offer them to the client. In the second type of encounter, the primary reason for a client's visit to a health-care provider is not related to preventing or achieving pregnancy. For example, the client might come in for acute care (e.g., a male client coming in for STD symptoms or as a contact of a person with an STD), for chronic care, or for another preventive service. In this situation, providers not only should address the client's primary reason for the visit but also assess the client's need for services related to preventing or achieving pregnancy.

A clinical pathway of family planning services for women and men of reproductive age is provided (Figure 2). The following questions can help providers determine what family planning services are most appropriate for a given visit.

- **What is the client's reason for the visit?** It is essential to understand the client's goals for the visit and address those needs to the extent possible.
- **Does the client have another source of primary health care?** Understanding whether a provider is the main source of primary care for a client will help identify what preventive services a provider should offer. If a provider is the client's main source of primary care, it will be important to assess the client's needs for the other services listed in this report. If the client receives ongoing primary care from another provider, the provider should confirm that the client's preventive health needs are met while avoiding the delivery of duplicative services.
- **What is the client's reproductive life plan?** An assessment should be made of the client's reproductive life plan, which outlines personal goals about becoming pregnant (*23–25*) (Box 2).The provider should avoid making assumptions about the client's needs based on his or her characteristics, such as sexual orientation or disabilities. For clients whose initial reason for coming to the service site was not related to preventing or achieving pregnancy, asking questions about his or her reproductive life plan might help identify unmet reproductive health-care needs. Identifying a need for contraceptive services might be particularly important given the high rate of unintended pregnancy in the United States.
  - If the client does not want a child at this time and is sexually active, then offer contraceptive services.
  - If the client desires pregnancy testing, then provide pregnancy testing and counseling.
  - If the client wants to have a child now, then provide services to help the client achieve pregnancy.

FIGURE 2. Clinical pathway of family planning services for women and men of reproductive age



– If the client wants to have a child and is experiencing difficulty conceiving, then provide basic infertility services.

- **Does the client need preconception health services?** Preconception health services (such as screening for obesity, smoking, and mental health) are a subset of all preventive services for women and men. Preconception health care is intended to promote the health of women and men of reproductive age before conception, with the goal of improving pregnancy-related outcomes (*24*). Preconception health services are also important because they improve the health of women and men, even if they choose not to become pregnant. The federal and professional medical recommendations cited in this report should be followed when determining which preconception health services a client might need.
- **Does the client need STD services?** The need for STD services, including HIV/AIDS testing, should be considered at every visit. Many clients requesting contraceptive services also might meet the criteria for being at risk of one or more STDs. Screening for chlamydia and gonorrhea is especially important in a family planning context because these STDs contribute to tubal infertility if left untreated. STD services are also necessary to maximize preconception health. The federal recommendations cited in this report should be followed when determining which STD services a client might need. Aspects of managing symptomatic STDs are not addressed in these recommendations.
- **What other related preventive health services does the client need?** Whether the client needs related preventive health services, such as breast and cervical cancer screening for female clients, should be assessed. The federal and professional medical recommendations cited in this report should be followed when determining which related preventive health services a client might need.

BOX 2. Recommended questions to ask when assessing a client's reproductive life plan

Providers should discuss a reproductive life plan with clients receiving contraceptive, pregnancy testing and counseling, basic infertility, sexually transmitted disease, and preconception health services in accordance with CDC's recommendation that all persons capable of having a child should have a reproductive life plan.*

Providers should assess the client's reproductive life plan by asking the client questions such as:

- Do you have any children now?
- Do you want to have (more) children?
- How many (more) children would you like to have and when?

* Source: CDC. Recommendations to improve preconception health and health care—United States: a report of the CDC/ATSDR Preconception Care Work Group and the Select Panel on Preconception Care. MMWR 2006;55(No. RR-6).

The individual client's needs should be considered when determining what services to offer at a given visit. It might not be feasible to deliver all the needed services in a single visit, and they might need to be delivered over the course of several visits. Providers should tailor services to meet the specific needs of the population they serve. For example, clients who are trying to achieve pregnancy and those at high risk of unintended pregnancy should be given higher priority for preconception health services. In some cases, the provider will deliver the initial screening service but then refer to another provider for further diagnosis or follow-up care.

The delivery of preconception, STD, and related preventive health services should not become a barrier to a client's ability to receive services related to preventing or achieving pregnancy. For these clients, receiving services related to preventing or achieving pregnancy is the priority; if other family planning services cannot be delivered at the initial visit, then follow-up visits should be scheduled.

In addition, professional recommendations for how to address the needs of diverse clients, such as LGBTQ persons (*26*–*32*) or persons with disabilities (*33*), should be consulted and integrated into procedures, as appropriate. For example, as noted before, providers should avoid making assumptions about a client's gender identity, sexual orientation, race, or ethnicity; all requests for services should be treated without regard to these characteristics. Similarly, services for adolescents should be provided in a "youth-friendly" manner, which means that they are accessible, equitable, acceptable, appropriate, comprehensive, effective, and efficient for youth, as recommended by the World Health Organization (*34*).

Contraceptive Services

Providers should offer contraceptive services to clients who wish to delay or prevent pregnancy. Contraceptive services should include consideration of a full range of FDA-approved contraceptive methods, a brief assessment to identify the contraceptive methods that are safe for the client, contraceptive counseling to help a client choose a method of contraception and use it correctly and consistently, and provision of one or more selected contraceptive method(s), preferably on site, but by referral if necessary. Contraceptive counseling is defined as a process that enables clients to make and follow through on decisions about their contraceptive use. Education is an integral component of the contraceptive counseling process that helps clients to make informed decisions and obtain the information they need to use contraceptive methods correctly.

Key steps in providing contraceptive services, including contraceptive counseling and education, have been outlined (Box 3). These key steps are in accordance with the five principles of quality counseling (Appendix C). To help a client who is initiating or switching to a new method of contraception, providers should follow these steps. These steps most likely will be implemented iteratively when working with a client and should help clients adopt, change, or maintain contraceptive use.

Step 1. Establish and maintain rapport with the client. Providers should strive to establish and maintain rapport. Strategies to achieve these goals include the following:

- using open-ended questions;
- demonstrating expertise, trustworthiness, and accessibility;
- ensuring privacy and confidentiality;
- explaining how personal information will be used;
- encouraging the client to ask questions and share information;
- listening to and observing the client; and
- being encouraging and demonstrating empathy and acceptance.

Step 2. Obtain clinical and social information from the client. Providers should ask clients about their medical history to identify methods that are safe. In addition, to learn more about factors that might influence a client's choice of a contraceptive method, providers should confirm the client's pregnancy intentions or reproductive life plan, ask about the client's contraceptive experiences and preferences, and conduct a sexual health assessment. When available, standardized tools should be used.

- **Medical history.** A medical history should be taken to ensure that methods of contraception being considered by a client are safe for that particular client. For a female client, the medical history should include menstrual history (including last menstrual period, menstrual frequency, length and amount of bleeding, and other

BOX 3. Steps in providing contraceptive services, including contraceptive counseling* and education

- Establish and maintain rapport with the client.
- Obtain clinical and social information from the client.
- Work with the client interactively to select the most effective and appropriate contraceptive method.
- Conduct a physical assessment related to contraceptive use, only when warranted.
- Provide the contraceptive method along with instructions about correct and consistent use, help the client develop a plan for using the selected method and for follow up, and confirm client understanding.

* Key principles of providing quality counseling including education have been outlined (Appendix C).

patterns of uterine/vaginal bleeding), gynecologic and obstetrical history, contraceptive use, allergies, recent intercourse, recent delivery, miscarriage, or termination, and any relevant infectious or chronic health condition and other characteristics and exposures (e.g., age, postpartum, and breastfeeding) that might affect the client's medical eligibility criteria for contraceptive methods (*35*). Clients considering combined hormonal contraception should be asked about smoking tobacco, in accordance with CDC guidelines on contraceptive use (*35*). Additional details about the methods of contraception that are safe to use for female clients with specific medical conditions and characteristics (e.g., hypertension) are addressed in previously published guidelines (*35*). For a male client, a medical history should include use of condoms, known allergies to condoms, partner use of contraception, recent intercourse, whether his partner is currently pregnant or has had a child, miscarriage, or termination, and the presence of any infectious or chronic health condition. However, the taking of a medical history should not be a barrier to making condoms available in the clinical setting (i.e., a formal visit should not be a prerequisite for a client to obtain condoms).

- **Pregnancy intention or reproductive life plan.** Each client should be encouraged to clarify decisions about her or his reproductive life plan (i.e., whether the client wants to have any or more children and, if so, the desired timing and spacing of those children) (*24*).
- **Contraceptive experiences and preferences.** Method-specific experiences and preferences should be assessed by asking questions such as, "What method(s) are you currently using, if any?"; "What methods have you used in the past?"; "Have you previously used emergency contraception?"; "Did you use contraception at last sex?"; "What difficulties did you experience with prior methods if any (e.g., side effects or noncompliance)?"; "Do you have a specific method in mind?"; and "Have you discussed method options with your partner, and does your partner have any preferences for which method you use?" Male clients should be asked if they are interested in vasectomy.
- **Sexual health assessment.** A sexual history and risk assessment that considers the client's sexual practices, partners, past STD history, and steps taken to prevent STDs (*36*) is recommended to help the client select the most appropriate method(s) of contraception. Correct and consistent condom use is recommended for those at risk for STDs. CDC recommendations for how to conduct a sexual health assessment have been summarized (Box 4).

Step 3. Work with the client interactively to select the most effective and appropriate contraceptive method. Providers should work with the client interactively to select an effective and appropriate contraceptive method. Specifically, providers should educate the client about contraceptive methods that the client can safely use, and help the client consider potential barriers to using the method(s) under consideration. Use of decision aids (e.g., computerized programs that help a client to identify a range of methods that might be appropriate for the client based on her physical characteristics such as health conditions or preferences about side effects) before or while waiting for the appointment can facilitate and maximize the utility of the time spent on this step.

Providers should inform clients about all contraceptive methods that can be used safely. Before the health-care visit, clients might have only limited information about all or specific methods of contraception (*37*). A broad range of methods, including long-acting reversible contraception (i.e., intrauterine devices [IUDs] and implants), should be discussed with all women and adolescents, if medically appropriate.

Providers are encouraged to present information on potential reversible methods of contraception by using a tiered approach (i.e., presenting information on the most effective methods first, before presenting information on less effective methods) (*38*,*39*). This information should include an explanation that long-acting reversible contraceptive methods are safe and effective for most women, including those who have never given birth and adolescents (*35*). Information should be tailored and presented to ensure a client-centered approach. It is not appropriate to omit presenting information on a method solely because the method is not available at the service site. If not all methods are available at the service site, it is important to have strong referral links in place to other providers to maximize opportunities for clients to obtain their preferred method that is medically appropriate.

BOX 4. Steps in conducting a sexual health assessment*

- **Practices:** Explore the types of sexual activity in which the patient engages (e.g., vaginal, anal, or oral sex).
- **Pregnancy prevention:** Discuss current and future contraceptive options. Ask about current and previous use of methods, use of contraception at last sex, difficulties with contraception, and whether the client has a particular method in mind.
- **Partners:** Ask questions to determine the number, gender (men, women, or both), and concurrency of the patient's sex partners (if partner had sex with another partner while still in a sexual relationship with the patient). It might be necessary to define the term "partner" to the patient or use other, relevant terminology.
- **Protection from sexually transmitted diseases (STDs):** Ask about condom use, with whom they do or do not use condoms, and situations that make it harder or easier to use condoms. Topics such as monogamy and abstinence also can be discussed.
- **Past STD history:** Ask about any history of STDs, including whether their partners have ever had an STD. Explain that the likelihood of an STD is higher with a past history of an STD.

* Source: CDC. Sexually transmitted diseases treatment guidelines, 2010. MMWR 2010;59(No. RR-12).

For clients who have completed childbearing or do not plan to have children, permanent sterilization (female or male) is an option that may be discussed. Both female and male sterilization are safe, are highly effective, and can be performed in an office or outpatient surgery setting (*40*,*41*). Women and men should be counseled that these procedures are not intended to be reversible and that other highly effective, reversible methods of contraception (e.g., implants or IUDs) might be an alternative if they are unsure about future childbearing. Clients interested in sterilization should be referred to an appropriate source of care if the provider does not perform the procedure.

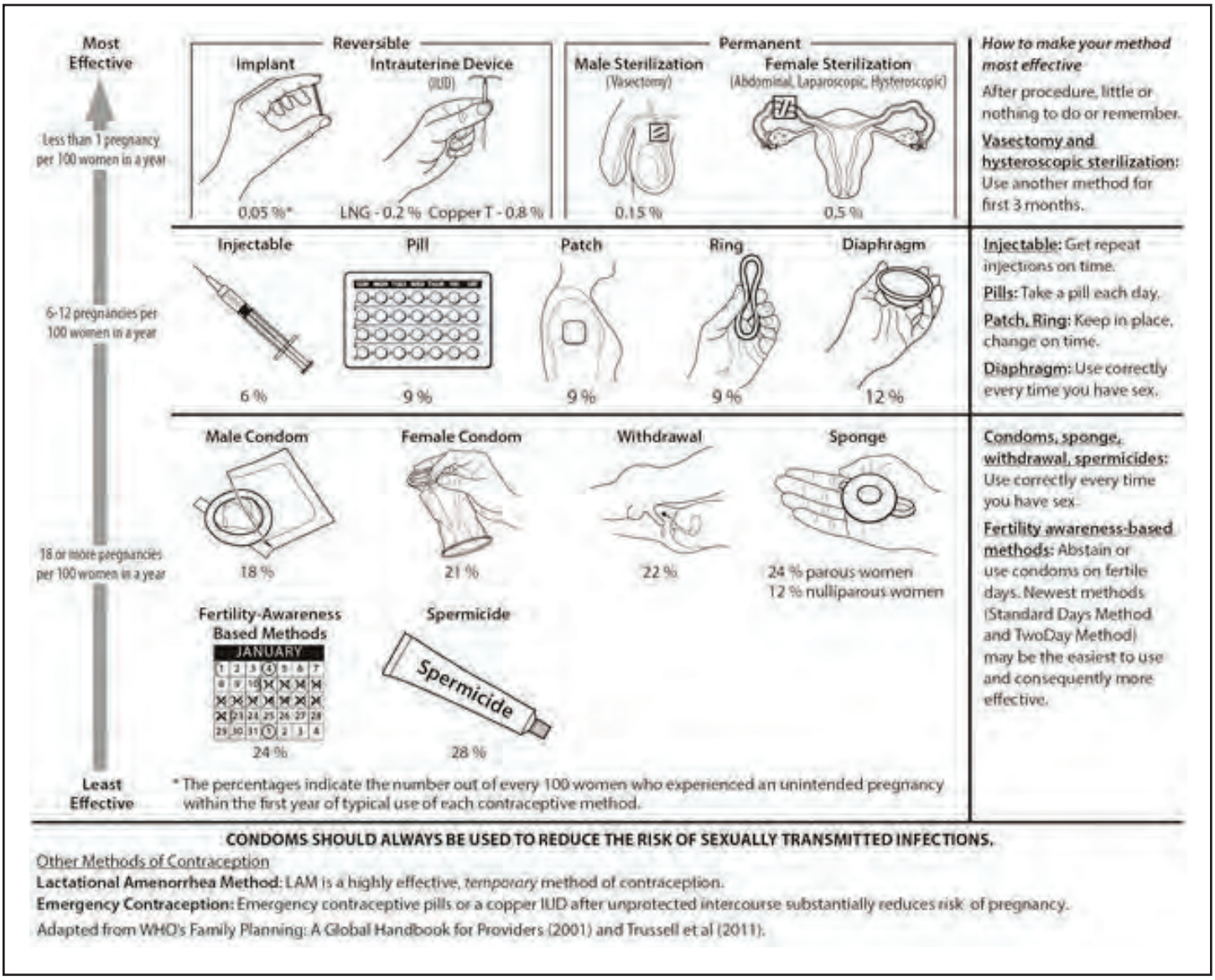
When educating clients about contraceptive methods that the clients can use safely, providers should ensure that clients understand the following:

- **Method effectiveness.** A contraceptive method's rate of typical effectiveness, or the percentage of women experiencing an unintended pregnancy during the first year of typical use, is an important consideration (Figure 3; Appendix D) (*38*,*42*).
- **Correct use of the method.** The mode of administration and understanding how to use the method correctly might be important considerations for the client when choosing a method. For example, receiving a contraceptive injection every 3 months might not be acceptable to a woman who fears injections. Similarly, oral contraceptives might not be acceptable to a woman who is concerned that she might not be able to remember to take a pill every day.
- **Noncontraceptive benefits.** Many contraceptives have noncontraceptive benefits, in addition to preventing pregnancy, such as reducing heavy menstrual bleeding. Although the noncontraceptive benefits are not generally the major determinant for selecting a method, awareness of these benefits can help clients decide between two or more suitable methods and might enhance the client's motivation to use the method correctly and consistently.
- **Side effects.** Providers should inform the client about risks and side effects of the method(s) under consideration, help the client understand that certain side effects of contraceptive methods might disappear over time, and encourage the client to weigh the experience of coping with side effects against the experience and consequences of an unintended pregnancy. The provider should be prepared to discuss and correct misperceptions about side effects. Clients also should be informed about warning signs for rare, but serious, adverse events with specific contraceptive methods, such as stroke and venous thromboembolism with use of combined hormonal methods.
- **Protection from STDs, including HIV.** Clients should be informed that contraceptive methods other than condoms offer no protection against STDs, including HIV. Condoms, when used correctly and consistently, help reduce the risk of STDs, including HIV, and provide protection against pregnancy. Dual protection (i.e., protection from both pregnancy and STDs) is important for clients at risk of contracting an STD, such as those with multiple or potentially infected partner(s). Dual protection can be achieved through correct and consistent use of condoms with every act of sexual intercourse, or correct and consistent use of a condom to prevent infection plus another form of contraception to prevent pregnancy. (For more information about preventing and treating STDs, see STD Services.)

When educating clients about the range of contraceptive methods, providers should ensure that clients have information that is medically accurate, balanced, and provided in a nonjudgmental manner. To assist clients in making informed decisions, providers should educate clients in a manner that can be readily understood and retained. The content, format, method, and medium for delivering education should be evidence-based (see Appendix E).

When working with male clients, when appropriate, providers should discuss information about female-controlled methods

FIGURE 3. The typical effectiveness of Food and Drug Administration–approved contraceptive methods



(including emergency contraception) encourage discussion of contraception with partners, and provide information about how partners can access contraceptive services. Male clients should also be reminded that condoms should be used correctly and consistently to reduce risk of STDs, including HIV.

When working with any client, encourage partner communication about contraception, as well as understanding partner barriers (e.g., misperceptions about side effects) and facilitators (e.g., general support) of contraceptive use (*43–46*).

The provider should help the client consider potential barriers to using the method(s) under consideration. This includes consideration of the following factors:

- **Social-behavioral factors.** Social-behavioral factors might influence the likelihood of correct and consistent use of contraception (*47*). Providers should help the client consider the advantages and disadvantages of the method(s) being considered, the client's feelings about using the method(s), how her or his partner is likely to respond, the client's peers' perceptions of the method(s), and the client's confidence in being able to use the method correctly and consistently (e.g., using a condom during every act of intercourse or remembering to take a pill every day) (*37*).
- **Intimate partner violence and sexual violence.** Current and past intimate partner sexual or domestic violence might impede the correct and consistent use of contraception, and might be a consideration when choosing a method (*47–49*). For example, an IUD might

be preferred because it does not require the partner's participation. The medical history might provide information on signs of current or past violence and, if not, providers should ask clients about relationship issues that might be potential barriers to contraceptive use. In addition, clients experiencing intimate partner violence or sexual violence should be referred for appropriate care.

- **Mental health and substance use behaviors.** Mental health (e.g., depression, anxiety disorders, and other mental disorders) and substance use behaviors (e.g., alcohol use, prescription abuse, and illicit drug use) might affect a client's ability to correctly and consistently use contraception (*47*,*50*). The medical history might provide information about the signs of such conditions or behaviors, and if not, providers should ask clients about substance use behaviors or mental health disorders, such as depression or anxiety, that might interfere with the motivation or ability to follow through with contraceptive use. If needed, clients with mental health disorders or risky substance use behaviors should be referred for appropriate care.

Step 4. Conduct a physical assessment related to contraceptive use, when warranted. Most women will need no or few examinations or laboratory tests before starting a method of contraception. Guidance on necessary examinations and tests related to initiation of contraception is available (*42*). A list of assessments that need to be conducted when providing reversible contraceptive services to a female client seeking to initiate or switch to a new method of reversible contraception is provided (Table 1) (*42*). Clinical evaluation of a client electing permanent sterilization should be guided by the clinician who performs the procedure. Recommendations for contraceptive use are available (*42*). Key points include the following:

- Blood pressure should be taken before initiating the use of combined hormonal contraception.
- Providers should assess the current pregnancy status of clients receiving contraception (*42*), which provides guidance on how to be reasonably certain that a woman is not pregnant at the time of contraception initiation. In most cases, a detailed history provides the most accurate assessment of pregnancy risk in a woman about to start using a contraceptive method. Routine pregnancy testing for every woman is not necessary.
- Weight measurement is not needed to determine medical eligibility for any method of contraception because all methods generally can be used among obese women. However, measuring weight and calculating BMI at baseline might be helpful for monitoring any changes and counseling women who might be concerned about weight change perceived to be associated with their contraceptive method.
- Unnecessary medical procedures and tests might create logistical, emotional, or economic barriers to contraceptive access for some women, particularly adolescents and low-income women, who have high rates of unintended pregnancies (*1*,*51*,*52*). For both adolescent and adult female clients, the following examinations and tests are not needed routinely to provide contraception safely to a healthy client (although they might be needed to address other non-contraceptive health needs) (*42*):
  - pelvic examinations, unless inserting an intrauterine device (IUD) or fitting a diaphragm;
  - cervical cytology or other cancer screening, including clinical breast exam;
  - human immunodeficiency virus (HIV) screening; and
  - laboratory tests for lipid, glucose, liver enzyme, and hemoglobin levels or thrombogenic mutations.

For male clients, no physical examination needs to be performed before distributing condoms.

Step 5. Provide the contraceptive method along with instructions about correct and consistent use, help the client develop a plan for using the selected method and for follow-up, and confirm client understanding.

- A broad range of FDA-approved contraceptive methods should be available onsite. Referrals for methods not available onsite should be provided for clients who indicate they prefer those methods. When providing contraception, providers should instruct the client about correct and consistent use and employ the following strategies to facilitate a client's use of contraception:
  - Provide onsite dispensing;
  - Begin contraception at the time of the visit rather than waiting for next menses (also known as "quick start") if the provider can reasonably be certain that the client is not pregnant (*42*). A provider can be reasonably certain that a woman is not pregnant if she has no symptoms or signs of pregnancy and meets any one of the following criteria (*42*,*53*):
    - is ≤7 days after the start of normal menses,
    - has not had sexual intercourse since the start of last normal menses,
    - has been using a reliable method of contraception correctly and consistently,
    - is ≤7 days after spontaneous or induced abortion,
    - is within 4 weeks postpartum,
    - is fully or nearly fully breastfeeding (exclusively breastfeeding or the vast majority [≥85%] of feeds are breastfeeds), amenorrheic, and <6 months postpartum;
  - Provide or prescribe multiple cycles (ideally a full year's supply) of oral contraceptive pills, the patch, or the ring

TABLE 1. Assessments to conduct when a female client is initiating a new method of reversible contraception

	Cu-IUD and LNG-IUD	Implant	Injectable	Combined hormonal contraception	Progestin-only pills	Condom	Diaphragm or cervical cap	Spermicide
Examination								
Blood pressure	C	C	C	A*	C	C	C	C
Weight (BMI) (weight [kg]/height [m]^2)	—†	—†	—†	—†	—†	C	C	C
Clinical breast examination	C	C	C	C	C	C	C	C
Bimanual examination and cervical inspection	A	C	C	C	C	C	A§	C
Laboratory test								
Glucose	C	C	C	C	C	C	C	C
Lipids	C	C	C	C	C	C	C	C
Liver enzymes	C	C	C	C	C	C	C	C
Hemoglobin	C	C	C	C	C	C	C	C
Thrombogenic mutations	C	C	C	C	C	C	C	C
Cervical cytology (Papanicolaou smear)	C	C	C	C	C	C	C	C
STD screening with laboratory tests	—¶	C	C	C	C	C	C	C
HIV screening with laboratory tests	C	C	C	C	C	C	C	C

Source: CDC. U.S. selected practice recommendations for contraceptive use 2013. MMWR 2013;62(No. RR-5).
Abbreviations: A = Class A: essential and mandatory in all circumstances for safe and effective use of the contraceptive method; B = Class B: contributes substantially to safe and effective use, but implementation might be considered within the public health and/or service context (the risk of not performing an examination or test should be balanced against the benefits of making the contraceptive method available); C = Class C: does not contribute substantially to safe and effective use of the contraceptive method; Cu-IUD = copper-containing intrauterine device; LNG-IUD = levonorgestrel releasing intrauterine device.
* In cases in which access to health care might be limited, the blood pressure measurement can be obtained by the woman in a nonclinical setting (e.g., pharmacy or fire station) and self-reported to the provider.
† Weight (BMI) measurement is not needed to determine medical eligibility for any methods of contraception because all methods can be used (U.S. Medical Eligibility Criteria 1) or generally can be used (U.S. Medical Eligibility Criteria 2) among obese women (Source: CDC. U.S. medical eligibility criteria for contraceptive use 2010. MMWR 2010;59[No. RR-4]). However, measuring weight and calculating BMI at baseline might be helpful for monitoring any changes and counseling women who might be concerned about weight change perceived to be associated with their contraceptive method.
§ A bimanual examination (not cervical inspection) is needed for diaphragm fitting.
¶ Most women do not require additional STD screening at the time of IUD insertion, if they have already been screened according to CDC's STD treatment guidelines (Sources: CDC. STD treatment guidelines. Atlanta, GA: US Department of Health and Human Services, CDC; 2013. Available at http://www.cdc.gov/std/treatment. CDC. Sexually transmitted diseases treatment guidelines, 2010. MMWR. 2010;59[No. RR-12]). If a woman has not been screened according to guidelines, screening can be performed at the time of IUD insertion and insertion should not be delayed. Women with purulent cervicitis or current chlamydial infection or gonorrhea should not undergo IUD insertion (U.S. Medical Eligibility Criteria 4). Women who have a very high individual likelihood of STD exposure (e.g., those with a currently infected partner) generally should not undergo IUD insertion (U.S. Medical Eligibility Criteria 3) (Source: CDC. U.S. medical eligibility criteria for contraceptive use 2010. MMWR 2010;59[No. RR-4]). For these women, IUD insertion should be delayed until appropriate testing and treatment occurs.

to minimize the number of times a client has to return to the service site;
  - Make condoms easily and inexpensively available; and
  - If a client chooses a method that is not available on-site or the same day, provide the client another method to use until she or he can start the chosen method.

- Help the client develop a plan for using the selected method. Using a method incorrectly or inconsistently and having gaps in contraceptive protection because of method switching both increase the likelihood of an unintended pregnancy (*37*). After the method has been provided, or a plan put into place to obtain the chosen method, providers should help the client develop an action plan for using the selected method.

  Providers should encourage clients to anticipate reasons why they might not use their chosen method(s) correctly or consistently, and help them develop strategies to deal with these possibilities. For example, for a client selecting oral contraceptive pills who might forget to take a pill, the provider can work with the client to identify ways to routinize daily pill taking (e.g., use of reminder systems such as daily text messages or cell phone alarms). Providers also may inform clients about the availability of emergency contraceptive pills and may provide clients an advance supply of emergency contraceptive pills on-site or by prescription, if requested.

  Side effects (e.g., irregular vaginal bleeding) are a primary reason for method discontinuation (*54*), so providers should discuss ways the client might deal with potential side effects to increase satisfaction with the method and improve continuation (*42*).

- Develop a plan for follow-up. Providers should discuss an appropriate follow-up plan with the client to meet their individual needs, considering the client's risk for discontinuation. Follow-up provides an opportunity to inquire about any initial difficulties the client might be experiencing, and might reinforce the perceived accessibility of the provider and increase rapport. Alternative modes of follow-up other than visits to the service site, such as telephone, e-mail, or text messaging, should be considered (assuming confidentiality can be assured), as needed.

  As noted previously, if a client chooses a method that is not available on-site or during the visit, the provider

should schedule a follow-up visit with the client or provide a referral for her or him to receive the method. The client should be provided another method to use until she or he can start the chosen method.
- Confirm the client's understanding. Providers should assess whether the client understands the information that was presented. The client's understanding of the most important information about her or his chosen contraceptive method should be documented in the medical record (e.g., by a checkbox or written statement).

The teach-back method may be used to confirm the client's understanding by asking the client to repeat back messages about risks and benefits and appropriate method use and follow-up. If providers assess the client's understanding, then the check box or written statement can be used in place of a written method-specific informed consent form. Topics that providers may consider having the client repeat back include the following: typical method effectiveness; how to use the method correctly; protection from STDs; warning signs for rare, but serious, adverse events and what to do if they experience a warning sign; and when to return for follow-up.

Provide Counseling for Returning Clients

When serving contraceptive clients who return for ongoing care related to contraception, providers should ask if the client has any concerns with the method and assess its use. The provider should assess any changes in the client's medical history, including changes in risk factors and medications that might affect safe use of the contraceptive method. If the client is using the method correctly and consistently and there are no concerns about continued use, an appropriate follow-up plan should be discussed and more contraceptive supplies given (*42*). If the client or provider has concerns about the client's correct or consistent use of the method, the provider should ask if the client would be interested in considering a different method of contraception. If the client is interested, the steps described above should be followed.

Counseling Adolescent Clients

Providers should give comprehensive information to adolescent clients about how to prevent pregnancy (*55–57*). This information should clarify that avoiding sex (i.e., abstinence) is an effective way to prevent pregnancy and STDs. If the adolescent indicates that she or he will be sexually active, providers should give information about contraception and help her or him to choose a method that best meets her or his individual needs, including the use of condoms to reduce the risk of STDs. Long-acting reversible contraception is a safe and effective option for many adolescents, including those who have not been pregnant or given birth (*35*).

Providers of family planning services should offer confidential services to adolescents and observe all relevant state laws and any legal obligations, such as notification or reporting of child abuse, child molestation, sexual abuse, rape, or incest, as well as human trafficking (*58,59*). Confidentiality is critical for adolescents and can greatly influence their willingness to access and use services (*60–67*). As a result, multiple professional medical associations have emphasized the importance of providing confidential services to adolescents (*68–70*).

Providers should encourage and promote communication between the adolescent and his or her parent(s) or guardian(s) about sexual and reproductive health (*71–86*). Adolescents who come to the service site alone should be encouraged to talk to their parents or guardians. Educational materials and programs can be provided to parents or guardians that help them talk about sex and share their values with their child (*72,87*). When both parent or guardian and child have agreed, joint discussions can address family values and expectations about dating, relationships, and sexual behavior.

In a given year, approximately 20% of adolescent births represent repeat births (*88*), so in addition to providing postpartum contraception, providers should refer pregnant and parenting adolescents to home visiting and other programs that have been demonstrated to provide needed support and reduce rates of repeat teen pregnancy (*89–94*).

Services for adolescents should be provided in a "youth-friendly" manner, which means that they are accessible, equitable, acceptable, appropriate, comprehensive, effective, and efficient for youth as recommended by the World Health Organization (*34*).

Pregnancy Testing and Counseling

Providers of family planning services should offer pregnancy testing and counseling services as part of core family planning services, in accordance with recommendations of major professional medical organizations, such as the American College of Obstetricians and Gynecologists (ACOG) and the American Academy of Pediatrics (AAP) (*95–97*).

Pregnancy testing is a common reason for a client to visit a provider of family planning services. Approximately 65% of pregnancies result in live births, 18% in induced abortion, and 17% spontaneous fetal loss (*98*). Among live births, only 1% of infants are placed for adoption within their first month of life (*99*).

The visit should include a discussion about her reproductive life plan and a medical history that includes asking about any coexisting conditions (e.g., chronic medical illnesses, physical disability, psychiatric illness) (*95,96*). In most cases,

a qualitative urine pregnancy test will be sufficient; however, in certain cases, the provider may consider performing a quantitative serum pregnancy test, if exact hCG levels would be helpful for diagnosis and management. The test results should be presented to the client, followed by a discussion of options and appropriate referrals.

Options counseling should be provided in accordance with recommendations from professional medical associations, such as ACOG and AAP (*95*–*97*). A female client might wish to include her partner in the discussion; however, if a client chooses not to involve her partner, confidentiality must be assured.

Positive Pregnancy Test

If the pregnancy test is positive, the clinical visit should include an estimation of gestational age so that appropriate counseling can be provided. If a woman is uncertain about the date of her last normal menstrual period, a pelvic examination might be needed to help assess gestational age. In addition, clients should receive information about the normal signs and symptoms of early pregnancy, and should be instructed to report any concerns to a provider for further evaluation. If ectopic pregnancy or other pregnancy abnormalities or problems are suspected, the provider should either manage the condition or refer the client for immediate diagnosis and management.

Referral to appropriate providers of follow-up care should be made at the request of the client, as needed. Every effort should be made to expedite and follow through on all referrals. For example, providers might provide a resource listing or directory of providers to help the client identify options for care. Depending upon a client's needs, the provider may make an appointment for the client, or call the referral site to let them know the client was referred. Providers also should assess the client's social support and refer her to appropriate counseling or other supportive services, as needed.

For clients who are considering or choose to continue the pregnancy, initial prenatal counseling should be provided in accordance with the recommendations of professional medical associations, such as ACOG (*97*). The client should be informed that some medications might be contraindicated in pregnancy, and any current medications taken during pregnancy need to be reviewed by a prenatal care provider (e.g., an obstetrician or midwife). In addition, the client should be encouraged to take a daily prenatal vitamin that includes folic acid; to avoid smoking, alcohol, and other drugs; and not to eat fish that might have high levels of mercury (*97*). If there might be delays in obtaining prenatal care, the client should be provided or referred for any needed STD screening (including HIV) and vaccinations (*36*).

Negative Pregnancy Test

Women who are not pregnant and who do not want to become pregnant at this time should be offered contraceptive services, as described previously. The contraceptive counseling session should explore why the client thought that she was pregnant and sought pregnancy testing services, and whether she has difficulties using her current method of contraception. A negative pregnancy test also provides an opportunity to discuss the value of making a reproductive life plan. Ideally, these services will be offered in the same visit as the pregnancy test because clients might not return at a later time for contraceptive services.

Women who are not pregnant and who are trying to become pregnant should be offered services to help achieve pregnancy or basic infertility services, as appropriate (see "Clients Who Want to Become Pregnant" and "Basic Infertility Services"). They also should be offered preconception health and STD services (see "Preconception Health Services" and "STD services").

Clients Who Want to Become Pregnant

Providers should advise clients who wish to become pregnant in accordance with the recommendations of professional medical organizations, such as the American Society for Reproductive Medicine (ASRM) (*100*).

Providers should ask the client (or couple) how long she or they have been trying to get pregnant and when she or they hope to become pregnant. If the client's situation does not meet one of the standard definitions of infertility (see "Basic Infertility Services"), then she or he may be counseled about how to maximize fertility. Key points are as follows:

- The client should be educated about peak days and signs of fertility, including the 6-day interval ending on the day of ovulation that is characterized by slippery, stretchy cervical mucus and other possible signs of ovulation.
- Women with regular menstrual cycles should be advised that vaginal intercourse every 1–2 days beginning soon after the menstrual period ends can increase the likelihood of becoming pregnant.
- Methods or devices designed to determine or predict the time of ovulation (e.g., over-the-counter ovulation kits, digital telephone applications, or cycle beads) should be discussed.
- It should be noted that fertility rates are lower among women who are very thin or obese, and those who consume high levels of caffeine (e.g., more than five cups per day).
- Smoking, consuming alcohol, using recreational drugs, and using most commercially available vaginal lubricants should be discouraged as these might reduce fertility.

Basic Infertility Services

Providers should offer basic infertility care as part of core family planning services in accordance with the recommendations of professional medical organizations, such as ACOG, ASRM, and the American Urological Association (AUA) (*96*,*101*,*102*).

Infertility commonly is defined as the failure of a couple to achieve pregnancy after 12 months or longer of regular unprotected intercourse (*101*). Earlier assessment (such as 6 months of regular unprotected intercourse) is justified for women aged >35 years, those with a history of oligo-amenorrhea (infrequent menstruation), those with known or suspected uterine or tubal disease or endometriosis, or those with a partner known to be subfertile (the condition of being less than normally fertile though still capable of effecting fertilization) (*101*). An early evaluation also might be warranted if risk factors of male infertility are known to be present or if there are questions regarding the male partner's fertility potential (*102*). Infertility visits to a family planning provider are focused on determining potential causes of the inability to achieve pregnancy and making any needed referrals to specialist care (*101*,*102*). ASRM recommends that evaluation of both partners should begin at the same time (*101*).

Basic Infertility Care for Women

The clinical visit should focus on understanding the client's reproductive life plan (*24*) and her difficulty in achieving pregnancy through a medical history, sexual health assessment and physical exam, in accordance with recommendations developed by professional medical associations such as ASRM (*101*) and ACOG (*96*). The medical history should include past surgery, including indications and outcome(s), previous hospitalizations, serious illnesses or injuries, medical conditions associated with reproductive failure (e.g., thyroid disorders, hirsutism, or other endocrine disorders), and childhood disorders; results of cervical cancer screening and any follow-up treatment; current medication use and allergies; and family history of reproductive failure. In addition, a reproductive history should include how long the client has been trying to achieve pregnancy; coital frequency and timing, level of fertility awareness, and results of any previous evaluation and treatment; gravidity, parity, pregnancy outcome(s), and associated complications; age at menarche, cycle length and characteristics, and onset/severity of dysmenorrhea; and sexual history, including pelvic inflammatory disease, history of STDs, or exposure to STDs. A review of systems should emphasize symptoms of thyroid disease, pelvic or abdominal pain, dyspareunia, galactorrhea, and hirsutism (*101*).

The physical examination should include: height, weight, and body mass index (BMI) calculation; thyroid examination to identify any enlargement, nodule, or tenderness; clinical breast examination; and assessment for any signs of androgen excess. A pelvic examination should assess for: pelvic or abdominal tenderness, organ enlargement or mass; vaginal or cervical abnormality, secretions, or discharge; uterine size, shape, position, and mobility; adnexal mass or tenderness; and cul-de-sac mass, tenderness, or nodularity. If needed, clients should be referred for further diagnosis and treatment (e.g., serum progesterone levels, follicle-stimulating hormone/luteinizing hormone levels, thyroid function tests, prolactin levels, endometrial biopsy, transvaginal ultrasound, hysterosalpingography, laparoscopy, and clomiphene citrate).

Basic Infertility Care for Men

Infertility services should be provided for the male partner of an infertile couple in accordance with recommendations developed by professional medical associations such as AUA (*102*). Providers should discuss the client's reproductive life plan, take a medical history, and conduct a sexual health assessment. AUA recommends that the medical history include a reproductive history (*102*). The medical history should include systemic medical illnesses (e.g., diabetes mellitus), prior surgeries and past infections; medications (prescription and nonprescription) and allergies; and lifestyle exposures. The reproductive history should include methods of contraception, coital frequency and timing; duration of infertility and prior fertility; sexual history; and gonadal toxin exposure, including heat. Patients also should be asked about their female partners' history of pelvic inflammatory disease, their partners' histories of STDs, and problems with sexual dysfunction.

In addition, a physical examination should be conducted with particular focus given to 1) examination of the penis, including the location of the urethral meatus; 2) palpation of the testes and measurement of their size; 3) presence and consistency of both the vas deferens and epididymis; 4) presence of a varicocele; 5) secondary sex characteristics; and 6) a digital rectal exam (*102*). Male clients concerned about their fertility should have a semen analysis. If this test is abnormal, they should be referred for further diagnosis (i.e., second semen analysis, endocrine evaluation, post-ejaculate urinalysis, or others deemed necessary) and treatment. The semen analysis is the first and most simple screen for male fertility.

Infertility Counseling

Counseling provided during the clinical visit should be guided by information elicited from the client during the medical and reproductive history and the findings of the

physical exam. If there is no apparent cause of infertility and the client does not meet the definition above, providers should educate the client about how to maximize fertility (see "Clients Who Want to Become Pregnant"). ACOG notes the importance of addressing the emotional and educational needs of clients with infertility and recommends that providers consider referring clients for psychological support, infertility support groups, or family counseling (*96*).

Preconception Health Services

Providers of family planning services should offer preconception health services to female and male clients in accordance with CDC's recommendations to improve preconception health and health care (*24*).

Preconception health services are beneficial because of their effect on pregnancy and birth outcomes and their role in improving the health of women and men. The term preconception describes any time that a woman of reproductive potential is not pregnant but at risk of becoming pregnant, or when a man is at risk for impregnating his female partner.

Preconception health-care services for women aim to identify and modify biomedical, behavioral, and social risks to a woman's health or pregnancy outcomes through prevention and management. It promotes the health of women of reproductive age before conception, and thereby helps to reduce pregnancy-related adverse outcomes, such as low birthweight, premature birth, and infant mortality (*24*). Moreover, the preconception health services recommended here are equally important because they contribute to the improvement of women's health and well-being, regardless of her childbearing intentions. CDC recommends that preconception health services be integrated into primary care visits made by women of reproductive age, such as family planning visits (*24*).

In the family planning setting, providers may prioritize screening and counseling about preconception health for couples that are trying to achieve pregnancy and couples seeking basic infertility services. Women who are using contraception to prevent or delay pregnancy might also benefit from preconception health services, especially those at high risk of unintended pregnancy. A woman is at high risk of unintended pregnancy if she is using no method or a less effective method of contraception (e.g., barrier methods, rhythm, or withdrawal), or has a history of contraceptive discontinuation or incorrect use (*38*,*39*). A woman is at lower risk of unintended pregnancy if she is using a highly effective method, such as an IUD or implant, or has an established history of using methods of contraception, such as injections, pills, patch, or ring correctly and consistently (*38*,*39*). Clients who do not want to become pregnant should also be provided preconception health services, since they are recommended by USPSTF for the purpose of improving the health of adults.

Recommendations for improving the preconception health of men also have been identified, although the evidence base for many of the recommendations for men is less than that for women (*103*). This report includes preconception health services that address men as partners in family planning (i.e., both preventing and achieving pregnancy), their direct contributions to infant health (e.g., genetics), and their role in improving the health of women (e.g., through reduced STD/HIV transmission). Moreover, these services are important for improving the health of men regardless of their pregnancy intention.

In a family planning setting, all women planning or capable of pregnancy should be counseled about the need to take a daily supplement containing 0.4 to 0.8 mg of folic acid, in accordance with the USPSTF recommendation (Grade A) (*104*).

Other preconception health services for women and men should include discussion of a reproductive life plan and sexual health assessment (Boxes 2 and 4), as well as the screening services described below (*24*,*103*,*105*). Services should be provided in accordance with the cited clinical recommendations, and any needed follow up (further diagnosis, treatment) should be provided either on-site or through referral.

Medical History

For female clients, the medical history should include the reproductive history, history of poor birth outcomes (i.e., preterm, cesarean delivery, miscarriage, and stillbirth), environmental exposures, hazards and toxins (e.g., smoking, alcohol, other drugs), medications that are known teratogens, genetic conditions, and family history (*24*,*105*).

For male clients, the medical history should include asking about the client's past medical and surgical history that might impair his reproductive health (e.g., genetic conditions, history of reproductive failures, or conditions that can reduce sperm quality, such as obesity, diabetes mellitus, and varicocele) and environmental exposures, hazards and toxins (e.g., smoking) (*103*).

Intimate Partner Violence

Providers should screen women of childbearing age for intimate partner violence and provide or refer women who screen positive to intervention services, in accordance with USPSTF (Grade B) recommendations (*106*).

Alcohol and Other Drug Use

For female and male adult clients, providers should screen for alcohol use in accordance with the USPSTF recommendation (Grade B) for how to do so, and provide behavioral counseling

interventions, as indicated (*107*). Screening adults for other drug use and screening adolescents for alcohol and other drug use has the potential to reduce misuse of alcohol and other drugs, and can be recommended (*105*,*108*,*109*). However, the USPSTF recommendation for screening for other drugs in adults, and for alcohol and other drugs in adolescents, is an "I," and patients should be informed that there is insufficient evidence to assess the balance of benefits and harms of this screening (*107*,*110*).

Tobacco Use

For female and male clients, providers should screen for tobacco use in accordance with the USPSTF recommendation (*111*,*112*) for how to do so. Adults (Grade A) who use tobacco products should be provided or referred for tobacco cessation interventions, including brief behavioral counseling sessions (<10 minutes) and pharmacotherapy delivered in primary care settings (*111*). Adolescents (Grade B) should be provided intervention to prevent initiation of tobacco use (*112*).

Immunizations

For female and male clients, providers should screen for immunization status in accordance with recommendations of CDC's Advisory Committee on Immunization Practices (*113*) and offer vaccination, as indicated, or provide referrals to community providers for immunization. Female and male clients should be screened for age-appropriate vaccinations, such as influenza and tetanus–diphtheria–pertussis (Tdap), measles, mumps, and rubella (MMR), varicella, pneumococcal, and meningococcal. In addition, ACOG recommends that rubella titer be performed in women who are uncertain about MMR immunization (*108*). (For vaccines for reproductive health-related conditions, i.e., human papillomavirus and hepatitis B, see "Sexually Transmitted Disease Services.")

Depression

For all clients, providers should screen for depression when staff-assisted depression care supports are in place to ensure accurate diagnosis, effective treatment, and follow-up (*114*,*115*). Staff-assisted care supports are defined as clinical staff members who assist the primary care clinician by providing some direct depression care, such as care support or coordination, case management, or mental health treatment. The lowest effective staff supports consist of a screening nurse who advises primary care clinicians of a positive screen and provides a protocol facilitating referral to behavioral therapy.

Providers also may follow American Psychiatric Association (*116*) and American Academy of Child and Adolescent Psychiatry (*117*) recommendations to assess risk for suicide among persons experiencing depression and other risk factors.

Height, Weight, and Body Mass Index

For all clients, providers should screen adult (Grade B) and adolescent (Grade B) clients for obesity in accordance with the USPSTF recommendation, and obese adults should be referred for intensive counseling and behavioral interventions to promote sustained weight loss (*118*,*119*). Clients likely will need to be referred for this service. These interventions typically comprise 12 to 26 sessions in a year and include multiple behavioral management activities, such as group sessions, individual sessions, setting weight-loss goals, improving diet or nutrition, physical activity sessions, addressing barriers to change, active use of self-monitoring, and strategizing how to maintain lifestyle changes.

Blood Pressure

For female and male clients, providers should screen for hypertension in accordance with the USPSTF's recommendation (Grade A) that blood pressure be measured routinely among adults (*120*) and the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure's recommendation that persons with blood pressure less than 120/80 be screened every 2 years, and every year if prehypertensive (i.e., blood pressure 120–139/80–89) (*121*). Providers also may follow AAP's recommendation that adolescents receive annual blood pressure screening (*109*).

Diabetes

For female and male clients, providers should follow the USPSTF recommendation (Grade B) to screen for type 2 diabetes in asymptomatic adults with sustained blood pressure (either treated or untreated) >135/80 mmHg (*122*).

Sexually Transmitted Disease Services

Providers should offer STD services in accordance with CDC's STD treatment and HIV testing guidelines (*36*,*123*,*124*). It is important to test for chlamydia annually among young sexually active females and for gonorrhea routinely among all sexually active females at risk for infection because they can cause tubal infertility in women if left untreated. Testing for syphilis, HIV/AIDS, and hepatitis C should be conducted as recommended (*36*,*123*,*124*). Vaccination for human papillomavirus (HPV) and hepatitis B are also important parts of STD services and preconception care (*113*).

STD services should be provided for persons with no signs or symptoms suggestive of an STD. STD diagnostic management recommendations are not included in these guidelines, so providers should refer to CDC's STD treatment guidelines

(36) when caring for clients with STD symptoms. STD services include the following steps, which should be provided at the initial visit and at least annually thereafter:

Step 1. Assess: The provider should discuss the client's reproductive life plan, conduct a standard medical history and sexual health assessment (see text box above), and check immunization status. A pelvic exam is not indicated in patients with no symptoms suggestive of an STD.

Step 2. Screen: A client who is at risk of an STD (i.e., sexually active and not involved in a mutually monogamous relationship with an uninfected partner) should be screened for HIV and the other STDs listed below, in accordance with CDC's STD treatment guidelines (*36*) and recommendations for HIV testing of adults, adolescents, and pregnant women in health-care settings (*123*). Clients also should follow CDC's recommendations for testing for hepatitis C (*124*), and the Advisory Committee on Immunization Practice's recommendations on reproductive health-related immunizations (*113*). It is important to follow these guidelines both to ensure that clients receive needed services and to avoid unnecessary screening.

Chlamydia

For female clients, providers should screen all sexually active women aged ≤25 years for chlamydia annually, in addition to sexually active women aged >25 years with risk factors for chlamydia infection (*36*). Women aged >25 years at higher risk include sexually active women who have a new or more than one sex partner or who have a partner who has other concurrent partners. Females with chlamydia infection should be rescreened for re-infection at 3 months after treatment. Pregnant women should be screened for chlamydia at the time of their pregnancy test if there might be delays in obtaining prenatal care (*36*).

For male clients, chlamydia screening can be considered for males seen at sites with a high prevalence of chlamydia, such as adolescent clinics, correctional facilities, and STD clinics (*36*,*125*,*126*). Providers should screen men who have sex with men (MSM) for chlamydia at anatomic sites of exposure, in accordance with CDC's STD treatment guidelines (*36*). Males with symptoms suggestive of chlamydia (urethral discharge or dysuria or whose partner has chlamydia) should be tested and empirically treated at the initial visit. Males with chlamydia infection should be re-screened for reinfection at 3 months (*36*).

Gonorrhea

For female clients, providers should screen clients for gonorrhea, in accordance with CDC's STD treatment guidelines (*36*). Routine screening for *N. gonorrhoeae* in all sexually active women at risk for infection is recommended annually (*36*). Women aged <25 years are at highest risk for gonorrhea infection. Other risk factors that place women at increased risk include a previous gonorrhea infection, the presence of other STDs, new or multiple sex partners, inconsistent condom use, commercial sex work, and drug use. Females with gonnorrhea infection should be re-screened for re-infection at 3 months after treatment. Pregnant women should be screened for gonorrhea at the time of their pregnancy test if there might be delays in obtaining prenatal care (*36*).

For male clients, providers should screen MSM for gonorrhea at anatomic sites of exposure, in accordance with CDC's STD treatment guidelines (*36*). Males with symptoms suggestive of gonorrhea (urethral discharge or dysuria or whose partner has gonorrhea) should be tested and empirically treated at the initial visit. Males with gonorrhea infection should be re-screened for reinfection at 3 months after treatment (*36*,*126*–*128*).

Syphilis

For female and male clients, providers should screen clients for syphilis, in accordance with CDC's STD treatment guidelines (*36*). CDC recommends that persons at risk for syphilis infection should be screened. Populations at risk include MSM, commercial sex workers, persons who exchange sex for drugs, those in adult correctional facilities and those living in communities with high prevalence of syphilis (*36*). Pregnant women should be screened for syphilis at the time of their pregnancy test if there might be delays in obtaining prenatal care (*36*).

HIV/AIDS

For female and male clients, providers should screen clients for HIV/AIDS, in accordance with CDC HIV testing guidelines (*123*). Providers should follow CDC recommendations that all clients aged 13–64 years be screened routinely for HIV infection and that all persons likely to be at high risk for HIV be rescreened at least annually (*123*). Persons likely to be at high risk include injection-drug users and their sex partners, persons who exchange sex for money or drugs, sex partners of HIV-infected persons, and MSM or heterosexual persons who themselves or whose sex partners have had more than one sex partner since their most recent HIV test. CDC further recommends that screening be provided after the patient is notified that testing will be performed as part of general medical consent unless the patient declines (opt-out screening) or otherwise prohibited by state law. The USPSTF also recommends screening for HIV (Grade A) (*129*).

Hepatitis C

For female and male clients, CDC recommends one-time testing for hepatitis C (HCV) without prior ascertainment of HCV risk for persons born during 1945–1965, a population with a disproportionately high prevalence of HCV infection

and related disease. Persons identified as having HCV infection should receive a brief screening for alcohol use and intervention as clinically indicated, followed by referral to appropriate care for HCV infection and related conditions. These recommendations do not replace previous guidelines for HCV testing that are based on known risk factors and clinical indications. Rather, they define an additional target population for testing: persons born during 1945–1965 (*124*). USPSTF also recommends screening persons at high risk for infection for hepatitis C and one-time screening for HCV infection for persons in the 1945–1965 birth cohort (Grade B) (*130*).

Immunizations Related to Reproductive Health

Female clients aged 11–26 years should be offered either human papillomavirus (HPV) 2 or HPV4 vaccine for the prevention of HPV and cervical cancer if not previously vaccinated, although the series can be started in persons as young as age 9 years (*113*); recommendations include starting at age 11–12 years and catch up vaccine among females aged 13–26 who have not been vaccinated previously or have not completed the 3-dose series through age 26. Routine hepatitis B vaccination should be offered to all unvaccinated children and adolescents aged <19 years and all adults who are unvaccinated and do not have any documented history of hepatitis B infection (*113*).

Male clients aged 11–21 years (minimum age: 9 years) should be offered HPV4 vaccine, if not vaccinated previously; recommendations include starting at age 11–12 years and catch up vaccine among males aged 13–21 years who have not been vaccinated previously or have not completed the 3-dose series through age 21 years; vaccination is recommended among at-risk males, including MSM and immune-compromised males through age 26 years if not vaccinated previously or males who have not completed the 3-dose series through age 26 years. Heterosexual males aged 22–26 years may be vaccinated (*131*). Routine hepatitis B vaccination should be offered to all unvaccinated children and adolescents aged <19 years, and all unvaccinated adults who do not have a documented history of hepatitis B infection (*113*).

Step 3. Treat: A client with an STD and her or his partner(s) should be treated in a timely fashion to prevent complications, re-infection and further spread of the infection in the community in accordance with CDC's STD treatment guidelines; clients with HIV infection should be linked to HIV care and treatment (*36*,*123*). Clients should be counseled about the need for partner evaluation and treatment to avoid reinfection at the time the client receives the positive test results. For partners of clients with chlamydia or gonorrhea, one option is to schedule them to come in with the client; another option for partners who cannot come in with the client is expedited partner therapy (EPT), as permissible by state laws, in which medication or a prescription is provided to the patient to give to the partner to ensure treatment. EPT is a partner treatment strategy for partners who are unable to access care and treatment in a timely fashion. Because of concerns related to resistant gonorrhea, efforts to bring in for treatment partners of patients with gonorrhea infection are recommended; EPT for gonorrhea should be reserved for situations in which efforts to treat partners in a clinical setting are unsuccessful and EPT is a gonorrhea treatment of last resort.

All clients treated for chlamydia or gonorrhea should be rescreened 3 months after treatment; HIV-infected females with *Trichomonas vaginalis* should be linked to HIV care and rescreened for *T. vaginalis* at 3 months. If needed, the client also should be vaccinated for hepatitis B and HPV (*113*). Ideally, STD treatment should be directly observed in the facility rather than a prescription given or called in to a pharmacy. If a referral is made to a service site that has the necessary medication available on-site, such as the recommended injectable antimicrobials for gonorrhea and syphilis, then the referring provider must document that treatment was given.

Step 4. Provide risk counseling: If the client is at risk for or has an STD, high-intensity behavioral counseling for sexual behavioral risk reduction should be provided in accordance with the USPSTF recommendation (Grade B) (*132*). One high-intensity behavioral counseling model that is similar to the contraceptive counseling model is Project Respect (*133*), which could be implemented in family planning settings. All sexually active adolescents are at risk, and adults are at increased risk if they have current STDs, had an STD in the past year, have multiple sexual partners, are in nonmonogamous relationships, or are sexually active and live in a community with a high rate of STDs.

Other key messages to give infected clients before they leave the service site include the following: a) refrain from unprotected sexual intercourse during the period of STD treatment, 2) encourage partner(s) to be screened or to get treatment as quickly as possible in accordance with CDC's STD treatment guidelines (partners in the past 60 days for chlamydia and gonorrhea, 3 to 6 months plus the duration of lesions or signs for primary and secondary syphilis, respectively) if the partner did not accompany the client to the service site for treatment, and 3) return for retesting in 3 months. If the partner is unlikely to access treatment quickly, then EPT for chlamydia or gonorrhea should be considered, if permissible by state law.

A client using or considering contraceptive methods other than condoms should be advised that these methods do not protect against STDs. Providers should encourage a client who is not in a mutually monogamous relationship with an

uninfected partner to use condoms. Patients who do not know their partners' infection status should be encouraged to get tested and use condoms or avoid sexual intercourse until their infection status is known.

Related Preventive Health Services

For many women and men of reproductive age, a family planning service site is their only source of health care; therefore, visits should include provision of or referral to other preventive health services. Providers of family planning services that do not have the capacity to offer comprehensive primary care services should have strong links to other community providers to ensure that clients have access to primary care. If a client does not have another source of primary care, priority should be given to providing related reproductive health services or providing referrals, as needed.

For clients without a primary care provider, the following screening services should be provided, with appropriate follow-up, if needed, while linking the client to a primary care provider. These services should be provided in accordance with federal and professional medical recommendations cited below regarding the frequency of screening, the characteristics of the clients that should be screened, and the screening procedures to be used.

Medical History

USPSTF recommends that women be asked about family history that would be suggestive of an increased risk for deleterious mutations in BRCA1 or BRCA2 genes (e.g., receiving a breast cancer diagnosis at an early age, bilateral breast cancer, history of both breast and ovarian cancer, presence of breast cancer in one or more female family members, multiple cases of breast cancer in the family, both breast and ovarian cancer in the family, one or more family members with two primary cases of cancer, and Ashkenazi background). Women with identified risk(s) should be referred for genetic counseling and evaluation for BRCA testing (Grade B) (*134*). The USPSTF also recommends that women at increased risk for breast cancer should be counseled about risk-reducing medications (Grade B) (*135*).

Cervical Cytology

Providers should provide cervical cancer screening to clients receiving related preventive health services. Providers should follow USPSTF recommendations to screen women aged 21–65 years with cervical cytology (Pap smear) every 3 years, or for women aged 30–65 years, screening with a combination of cytology and HPV testing every 5 years (Grade A) (*136*).

Cervical cytology no longer is recommended on an annual basis. Further, it is not recommended (Grade D) for women aged <21 years (*136*). Women with abnormal test results should be treated in accordance with professional standards of care, which may include colposcopy (*96*,*137*). The need for cervical cytology should not delay initiation or hinder continuation of a contraceptive method (*42*).

Providers should also follow ACOG and AAP recommendations that a genital exam should accompany a cervical cancer screening to inspect for any suspicious lesions or other signs that might indicate an undiagnosed STD (*96*,*97*,*138*).

Clinical Breast Examamination

Despite a lack of definitive data for or against, clinical breast examination has the potential to detect palpable breast cancer and can be recommended. ACOG recommends annual examination for all women aged >19 years (*108*). ACS recommends screening every 3 years for women aged 20–39 years, and annually for women aged ≥40 years (*139*). However, the USPSTF recommendation for clinical breast exam is an I, and patients should be informed that there is insufficient evidence to assess the balance of benefits and harms of the service (*140*).

Mammography

Providers should follow USPSTF recommendations (Grade B) to screen women aged 50–74 years on a biennial basis; they should screen women aged <50 years if other conditions support providing the service to an individual patient (*140*).

Genital Examination

For adolescent males, examination of the genitals should be conducted. This includes documentation of normal growth and development and other common genital findings, including hydrocele, varicocele, and signs of STDs (*141*). Components of this examination include inspecting skin and hair, palpating inguinal nodes, scrotal contents and penis, and inspecting the perinanal region (as indicated).

Summary of Recommendations for Providing Family Planning and Related Preventive Health Services

The screening components for each family planning and related preventive health service are provided in summary checklists for women (Table 2) and men (Table 3). When considering how to provide the services listed in these recommendations (e.g., the screening components for each

service, risk groups that should be screened, the periodicity of screening, what follow-up steps should be taken if screening reveals the presence of a health condition), providers should follow CDC and USPSTF recommendations cited above, or, in the absence of CDC and USPSTF recommendations, the recommendations of professional medical associations. Following these recommendations is important both to ensure clients receive needed care and to avoid unnecessary screening of clients who do not need the services.

The summary tables describe multiple screening steps, which refer to the following: 1) the process of asking questions about a client's history, including a determination of whether risk factors for a disease or health condition exist; 2) performing a physical exam; and 3) performing laboratory tests in at-risk asymptomatic persons to help detect the presence of a specific disease, infection, or condition. Many screening recommendations apply only to certain subpopulations (e.g., specific age groups, persons who engage in specific risk behaviors or who have specific health conditions), or some screening recommendations apply to a particular frequency (e.g., a cervical cancer screening is generally recommended every 3 years rather than annually). Providers should be aware that the USPSTF also has recommended that certain screening services not be provided because the harm outweighs the benefit (see Appendix F).

When screening results indicate the potential or actual presence of a health condition, the provider should either provide or refer the client for the appropriate further diagnostic testing or treatment in a manner that is consistent with the relevant federal or professional medical associations' clinical recommendations.

Conducting Quality Improvement

Service sites that offer family planning services should have a system for conducting quality improvement, which is designed to review and strengthen the quality of services on an ongoing basis. Quality improvement is the use of a deliberate and continuous effort to achieve measurable improvements in the identified indicators of quality of care, which improve the health of the community (*142*). By improving the quality of care, family planning outcomes, such as reduced rates of unintended pregnancy, improved patient experiences, and reduced costs, are more likely to be achieved (*10*,*12*,*143*,*144*).

Several frameworks for conducting quality improvement have been developed (*144–146*). This section presents a general overview of three key steps that providers should take when conducting quality improvement of family planning services: 1) determine which measures are needed to monitor quality; 2) collect the information needed; and 3) use the findings to make changes to improve quality (*147*). Ideally, these steps will be conducted on a frequent (optimally, quarterly) and ongoing basis. However, since quality cuts across all aspects of a program, not all domains of quality can necessarily be considered at all times. Within a sustainable system of quality improvement, programs can opt to focus on a subset of quality dimensions and their respective measures.

Determining Which Measures Are Needed

Performance measures provide information about how well the service site is meeting pre-established goals (*148*). The following questions should be considered when selecting performance measures (*143*):

- Is the topic important to measure and report? For example, does it address a priority aspect of health care, and is there opportunity for improvement?
- What is the level of evidence for the measure (e.g., that a change in the measure is likely to represent a true change in health outcomes)? Does the measure produce consistent (reliable) and credible (valid) results about the quality of care?
- Are the results meaningful and understandable and useful for informing quality improvement?
- Is the measure feasible? Can it be implemented without undue burden (e.g., captured with electronic data or electronic health records)?

Performance measures should consider the quality of the structure of services (e.g., the characteristics of the settings in which providers deliver health care, including material resources, human resources, and organizational structure), the process by which care is provided (whether services are provided correctly and completely, and how clients perceive the care they receive), and the outcomes of that care (e.g., client behaviors or health conditions that result) (*149*). They also may assess each dimension of quality services (*10*,*13*). Examples of measures that can be used for monitoring the quality of family planning services (*150*) and suggested measures that might help providers monitor quality of care have been listed (Table 6). However, other measures have been developed that also might be useful (*151–153*). Service sites that offer family planning services should select, measure, and assess at least one intermediate or outcome measure on an ongoing basis, for which the service site can be accountable. Structure- and process-based measures that assess the eight dimensions of quality services may be used to better determine how to improve quality (*154*).

Collecting Information

Once providers have determined what information is needed, the next steps are to collect and use that information to improve the quality of care. Commonly used methods of data collection include the following:

TABLE 2. Checklist of family planning and related preventive health services for women

Screening components	Family planning services (provide services in accordance with the appropriate clinical recommendation)					Related preventive health services
	Contraceptive services*	Pregnancy testing and counseling	Basic infertility services	Preconception health services	STD services†	
History						
Reproductive life plan§	Screen	Screen	Screen	Screen	Screen	
Medical history§,**	Screen	Screen	Screen	Screen	Screen	Screen
Current pregnancy status§	Screen					
Sexual health assessment§,**	Screen		Screen	Screen	Screen	
Intimate partner violence §,¶,**				Screen		
Alcohol and other drug use§,¶,**				Screen		
Tobacco use§,¶	Screen (combined hormonal methods for clients aged ≥35 years)			Screen		
Immunizations§				Screen	Screen for HPV & HBV§§	
Depression§,¶				Screen		
Folic acid§,¶				Screen		
Physical examamination						
Height, weight and BMI§,¶	Screen (hormonal methods)††		Screen	Screen		
Blood pressure§,¶	Screen (combined hormonal methods)			Screen§§		
Clinical breast exam**			Screen			Screen§§
Pelvic exam§,**	Screen (initiating diaphragm or IUD)	Screen (if clinically indicated)	Screen			
Signs of androgen excess**			Screen			
Thyroid exam**			Screen			
Laboratory testing						
Pregnancy test **	Screen (if clinically indicated)	Screen				
Chlamydia§, ¶	Screen¶¶				Screen§§	
Gonorrhea§, ¶	Screen¶¶				Screen§§	
Syphilis§,¶					Screen§§	
HIV/AIDS§,¶					Screen§§	
Hepatitis C§,¶					Screen§§	
Diabetes§,¶				Screen§§		
Cervical cytology¶						Screen§§
Mammography¶						Screen§§

Abbreviations: BMI = body mass index; HBV = hepatitis B virus; HIV/AIDS = human immunodeficiency virus/acquired immunodeficiency syndrome; HPV = human papillomavirus; IUD = intrauterine device; STD = sexually transmitted disease.

* This table presents highlights from CDC's recommendations on contraceptive use. However, providers should consult appropriate guidelines when treating individual patients to obtain more detailed information about specific medical conditions and characteristics (Source: CDC. U.S. medical eligibility criteria for contraceptive use 2010. MMWR 2010;59(No. RR-4).

† STD services also promote preconception health but are listed separately here to highlight their importance in the context of all types of family planning visits. The services listed in this column are for women without symptoms suggestive of an STD.

§ CDC recommendation.

¶ U.S. Preventive Services Task Force recommendation.

** Professional medical association recommendation.

†† Weight (BMI) measurement is not needed to determine medical eligibility for any methods of contraception because all methods can be used (U.S. Medical Eligibility Criteria 1) or generally can be used (U.S. Medical Eligibility Criteria 2) among obese women (Source: CDC. U.S. medical eligibility criteria for contraceptive use 2010. MMWR 2010;59[No. RR-4]). However, measuring weight and calculating BMI at baseline might be helpful for monitoring any changes and counseling women who might be concerned about weight change perceived to be associated with their contraceptive method.

§§ Indicates that screening is suggested only for those persons at highest risk or for a specific subpopulation with high prevalence of an infection or condition.

¶¶ Most women do not require additional STD screening at the time of IUD insertion if they have already been screened according to CDC's STD treatment guidelines (Sources: CDC. STD treatment guidelines. Atlanta, GA: US Department of Health and Human Services, CDC; 2013. Available at http://www.cdc.gov/std/treatment. CDC. Sexually transmitted diseases treatment guidelines, 2010. MMWR 2010;59[No. RR-12]). If a woman has not been screened according to guidelines, screening can be performed at the time of IUD insertion and insertion should not be delayed. Women with purulent cervicitis or current chlamydial infection or gonorrhea should not undergo IUD insertion (U.S. Medical Eligibility Criteria 4) women who have a very high individual likelihood of STD exposure (e.g. those with a currently infected partner) generally should not undergo IUD insertion (U.S. Medical Eligibility Criteria 3) (Source: CDC. US medical eligibility criteria for contraceptive use 2010. MMWR 2010;59[No. RR-4]). For these women, IUD insertion should be delayed until appropriate testing and treatment occurs.

- **Review of medical records.** All records that detail service delivery activities can be reviewed, including encounters and claims data, client medical records, facility logbooks, and others. It is important that records be carefully designed, sufficiently detailed, provide accurate information, and have access restricted to protect confidentiality. The use of electronic health records can facilitate some types of medical record review.
- **Exit interview with the client.** A patient is asked (through either a written or in-person survey) to describe what happened during the encounter or their assessment of their satisfaction with the visit. Both quantitative (close-ended questions) and qualitative (open-ended questions) methods can be used. Limitations include a bias toward clients reporting higher degrees of satisfaction, and the

TABLE 3. Checklist of family planning and related preventive health services for men

Screening components and source of recommendation	Family planning services (provide services in accordance with the appropriate clinical recommendation)				Related preventive health services
	Contraceptive services*	Basic infertility services	Preconception health services†	STD services§	
History					
Reproductive life plan¶	Screen	Screen	Screen	Screen	
Medical history¶,††	Screen	Screen	Screen	Screen	
Sexual health assessment¶,††	Screen	Screen	Screen	Screen	
Alcohol & other drug use ¶,**,††			Screen		
Tobacco use¶,**			Screen		
Immunizations¶			Screen	Screen for HPV & HBV§§	
Depression¶,**			Screen		
Physical examination					
Height, weight, and BMI¶,**			Screen		
Blood pressure**,††			Screen§§		
Genital exam††		Screen (if clinically indicated)		Screen (if clinically indicated)	Screen§§
Laboratory testing					
Chlamydia¶				Screen§§	
Gonorrhea¶				Screen§§	
Syphilis¶,**				Screen§§	
HIV/AIDS¶,**				Screen§§	
Hepatitis C¶,**				Screen§§	
Diabetes¶,**			Screen§§		

Abbreviations: HBV = hepatitis B virus; HIV/AIDS = human immunodeficiency virus/acquired immunodeficiency syndrome; HPV = human papillomavirus virus; STD = sexually transmitted disease.

* No special evaluation needs to be done prior to making condoms available to males. However, when a male client requests advice on pregnancy prevention, he should be provided contraceptive services as described in the section "Provide Contraceptive Services."

† The services listed here represent a sub-set of recommended preconception health services for men that were recommended and for which there was a direct link to fertility or infant health outcomes (Source: Frey K, Navarro S, Kotelchuck M, Lu M. The clinical content of preconception care: preconception care for men. Am J Obstet Gynecol 2008;199[6 Suppl 2]:S389–95).

§ STD services also promote preconception health, but are listed separately here to highlight their importance in the context of all types of family planning visit. The services listed in this column are for men without symptoms suggestive of an STD.

¶ CDC recommendation.

** U.S. Preventive Services Task Force recommendation.

†† Professional medical association recommendation.

§§ Indicates that screening is suggested only for those persons at highest risk or for a specific subpopulation with high prevalence of infection or other condition.

provider's behavior might be influenced if she or he knows clients are being interviewed.

- **Facility audit.** Questions about a service site's structure (e.g., on-site availability of a broad range of FDA-approved methods) and processes (e.g., skills and technical competence of staff, referral mechanisms) can be used to determine the readiness of the facility to serve clients.
- **Direct observation.** A provider's behavior is observed during an actual encounter with a client. Evaluation of a full range of competencies, including communication skills, can be carried out. A main limitation is that the observer's presence might influence the provider's performance.
- **Interview with the health-care provider.** Providers are interviewed about how specific conditions are managed. Both closed- and open-ended questions can be used, although it is important to frame the question so that the 'correct' answer is not suggested. A limitation is that providers tend to over-report their performance.

Consideration and Use of the Findings

After data are collected, they should be tabulated, analyzed, and used to improve care. Staff whose performance was assessed should be involved in the development of the data collection tools and analysis of results. Analysis should address the following questions (*155*):

- What is the performance level of the facility?
- Is there a consistent pattern of performance among providers?
- What is the trend in performance?
- What are the causes of poor performance?
- How can performance gaps be minimized?

Given the findings, service site staff should use a systematic approach to identifying ways to improve the quality of care. One example of a systematic approach to improving the quality of care is the "Plan, Do, Study, and Act" (PDSA) model (*147*,*156*), in which staff first develop a plan for improving quality, then execute the plan on a small scale, evaluate feedback to confirm or adjust the plan, and finally, make the plan

TABLE 4. Suggested measures of the quality of family planning services

Type of measure and dimension of quality	Measure	Source
Health outcome	• Unintended pregnancy • Teen pregnancy • Birth spacing • Proportion of female users at risk for unintended pregnancy who adopt or continue use of an FDA-approved contraceptive method (measured for any method; highly effective methods; or long-acting reversible methods) [Intermediate outcome]	PIMS*
Safe (Structure)	• Proportion of providers that follow the most current CDC recommendations on contraceptive safety	
Effective (Structure, or the characteristics of the settings in which providers deliver health care, including material resources, human resources, and organizational structure)	• Site dispenses or provides on-site a full range of FDA-approved contraceptive methods to meet the diverse reproductive needs and goals of clients; short-term hormonal, long-acting reversible contraception (LARC), emergency contraception (EC). • Proportion of female users aged ≥24 years who are screened annually for chlamydial infection. • Proportion of female users aged ≥24 years who are screened annually for gonorrhea. • Proportion of users who were tested for HIV during the past 12 months. • Proportion of female users aged ≥21 years who have received a Pap smear within the past 3 years.	PIMS*
Client-centered (Process, or whether services are provided correctly and completely, and how clients perceive the care they receive)	• Proportion of clients who report the provider communicates well, shows respect, spends enough time with the client, and is informed about the client's medical history. • Proportion of clients who report that – Staff are helpful and treat clients with courtesy and respect. – His or her privacy is respected. – She or he receives contraceptive method that is acceptable to her or him.	CAHPS† RQIP§
Efficient (Structure)	• Site uses electronic health information technology or electronic health records to improve client reproductive health.	PIMS*
Timely (Structure and process)	• Average number of days to the next appointment. • Site offers routine contraceptive resupply on a walk-in basis. • Site offers on-site HIV testing (using rapid technology). • Site offers on-site HPV and hepatitis B vaccination.	PIMS*
Accessible (Structure and process)	• Site offers family planning services during expanded hours of operation. • Proportion of total family planning encounters that are encounters with ongoing or continuing users. • Proportion of clients who report that his or her care provider follows up to give test results, has up-to-date information about care from specialists, and discusses other prescriptions. • Site has written agreements (e.g., MOUs) with the key partner agencies for health care (especially prenatal care, primary care, HIV/AIDS) and social service (domestic violence, food stamps) referrals.	PIMS* CAHPS–PCMH item set on care coordination†
Equitable (Structure)	• Site offers language assistance at all points of contact for the most frequently encountered language(s).	PIMS*
Value	• Average cost per client.	CDC¶

Abbreviations: CAPHS = Agency for Healthcare Research and Quality's Consumer Assessment of Health Care Providers and Systems; FDA = Food and Drug Administration; HPV = human papillomavirus; MOU = memorandum of understanding; PIMS = Performance Information and Monitoring System; RQIP = Regional Quality Indicators Program.

* **Source:** Fowler C. Title X Family Planning Program Performance Information and Monitoring System (PIMS): Description of Proposed Performance Measures [DRAFT]. Washington, DC: Research Triangle Institute; 2012.

† **Source:** Agency for Healthcare Research and Quality. Consumer Assessment of Healthcare Providers and Systems (CAHPS). Available at https://www.cahps.ahrq.gov/default.asp.

§ **Source:** John Snow International. The Regional Quality Indicators Project (RQIP). Boston, MA: John Snow International; 2014. Available at http://www.jsi.com/JSIInternet/USHealth/project/display.cfm?ctid=na&cid=na&tid=40&id=2621.

¶ **Sources:** Haddix A, Corso P, Gorsky R. Costs. In: Haddix A, Teutsch S, Corso P, eds. Prevention effectiveness: a guide to decision analysis and economic evaluation. 2nd ed. Oxford, UK: Oxford University Press; 2003; Stiefel M, Nolan K. A guide to measuring the triple aim: population health, experience of care, and per capita cost. Cambridge, MA: Institute for Healthcare Improvements; 2012.

permanent. Examples of steps that may be taken to improve the quality of care include developing job aids, providing task-specific training for providers, conducting more patient education, or strengthening relationships with referral sites through formal memoranda of understanding (*146*).

Conclusion

The United States continues to face substantial challenges to improving the reproductive health of the U.S. population. The recommendations in this report can contribute to improved reproductive health by defining a core set of family planning

services for women and men, describing how to provide contraceptive and other family planning services to both adult and adolescent clients, and encouraging the use of the family planning visit to provide selected preventive health services for women and men. This guidance is intended to assist primary care providers to offer the family planning services that will help persons and couples achieve their desired number and spacing of children and increase the likelihood that those children are born healthy.

Recommendations are updated periodically. The most recent versions are available at http://www.hhs.gov/opa.

References

1. Finer LB, Zolna MR. Unintended pregnancy in the United States: incidence and disparities, 2006. Contraception 2011;84:478–85.
2. CDC. Adolescent pregnancy and childbirth—United States, 1991–2008. In: CDC. CDC health disparities and inequalities report—United States, 2011. MMWR 2011;60(Suppl; January 14, 2011):105–8.
3. Martin J, Hamilton B, Osterman J, et al. Births: final data for 2012. Natl Vital Stat Rep 2013;62(9).
4. Mathews T, Macdorman M. Infant mortality statistics from the 2010 period linked birth/infant data set. Natl Vital Stat Rep 2013:62(8).
5. US Department of Health and Human Services, Office of Population Affairs. Mission statement for the Office of Family Planning. Washington, DC: US Department of Health and Human Services; 2013. Available at http://www.hhs.gov/opa/about-opa-and-initiatives/mission.
6. Institute of medicine. Clinical preventive services for women: closing the gaps. Washington, DC: The National Academies Press; 2011. Available at http://www.nap.edu/catalog.php?record_id=13181.
7. US Department of Health and Human Services, Office of Population Affairs. Title X family planning. Washington, DC: US Department of Health and Human Services; 2013. Available at http://www.hhs.gov/opa/title-x-family-planning/index.html.
8. Frost J. U.S. women's use of sexual and reproductive health services: trends, sources of care and factors associated with use, 1995–2010. New York, NY: Guttmacher Institute; 2013.
9. US Department of Health and Human Services Coverage of certain preventive services under the Affordable Care Act: final rules. 45 CFR Parts 147 and 156. July 19, 2010. Washington, DC: US Department of Health and Human Services; 2010.
10. Institute of Medicine. Committee on Quality of Health Care in America. Crossing the quality chasm: a new health system for the 21st century. Washington, DC: National Academies of Science; 2001.
11. Bruce J. Fundamental elements of the quality of care: a simple framework. Stud Fam Plann 1990;21:61–91.
12. Becker D, Koenig MA, Kim YM, Cardona K, Sonenstein FL. The quality of family planning services in the United States: findings from a literature review. Perspect Sex Reprod Health 2007;39:206–15.
13. Institute of Medicine. Future directions for the national health care quality and disparities reports. Ullmer CBM, Burke S, eds. Washington, DC: The National Academies Press; 2010.
14. Tucker CM. US Department of Health and Human Services, Advisory Committee on Minority Health. Reducing health disparities by promoting patient-centered culturally and linguistically sensitive/competent health care. Rockville, MD: US Department of Health and Human Services, Public Health Service; 2009.
15. US Department of Health and Human Services, Office of Minority Health. The national standards for culturally and linguistically appropriate services in health and health care. Washington, DC: US Department of Health and Human Services; 2012. Available at https://www.thinkculturalhealth.hhs.gov/Content/clas.asp#clas_standards.
16. Olavarria M, Beaulac J, Belanger A, Young M, Aubry T. Organizational cultural competence in community health and social service organizations: how to conduct a self-assessment. J Cult Divers 2009;16:140–50.
17. Frost J, Finer L, Tapales A. The impact of publicly funded family planning clinic services on unintended pregnancies and government cost savings. J Health Care Poor Underserved 2008;19:778–96.
18. Trussell J, Henry N, Hassan F, Prezioso A, Law A, Filonenko A. Burden of unintended pregnancy in the United States: potential savings with increased use of long-acting reversible contraception. Contraception 2013;87:154–61.
19. Foster DG, Rostovtseva DP, Brindis CD, Biggs MA, Hulett D, Darney PD. Cost savings from the provision of specific methods of contraception in a publicly funded program. Am J Public Health 2009;99:446–51.
20. GRADE Working Group. GRADE: going from evidence to recommendations. BMJ 2008;336:1049–51.
21. Institute of Medicine. Clinical practice guidelines we can trust. Washington, DC: The National Academies Press; 2011. Available at http://www.nap.edu/catalog.php?record_id=13058.
22. US Preventive Services Task Force. USPSTF: methods and processes. Rockville, MD: US Department of Health and Human Services, Agency for Healthcare Research and Quality; 2013. Available at http://www.uspreventiveservicestaskforce.org/methods.htm.
23. Lu M. Recommendations for preconception care. Am Fam Physician 2007;76:397–400.
24. CDC. Recommendations to improve preconception health and health care—United States. MMWR 2006;55(No. RR-6).
25. Moos M. Preconceptional health promotion: progress in changing a prevention paradigm. J Perinat Neonatal Nurs 2004;18:2–13.
26. American College of Obstetricians and Gynecologists. Committee Opinion #512: health care for transgender individuals. Obstet Gynecol 2011;118:1454–8.
27. American College of Obstetricians and Gynecologists, Committee on Health Care for Underserved Women. ACOG Committee Opinion No. 525: health care for lesbians and bisexual women. Obstet Gynecol 2012;119:1077–80.
28. McNair R, Hegarty K. Guidelines for the primary care of lesbian, gay and bisexual people: a systematic review. Ann Fam Med 2010;8:533–41.
29. World Professional Association for Transgender Health. Standards of care for the health of transsexual, transgender, and gender-nonconforming people. Minneapolis, MN: World Professional Association for Transgender Health; 2011.
30. The Center of Excellence for Transgender Health. Primary care protocol for transgender patient care. San Francisco, CA: University of California–San Francisco; 2011.
31. American Academy of Pediatrics. Office-based care for lesbian, gay, bisexual, transgender, and questioning youth: Policy statement and Technical report. Pediatrics 2013;132:198–203.
32. Adelson SL, American Academy of Child and Adolescent Psychiatry Committee on Quality Issues. Practice parameter on gay, lesbian or bisexual orientation, gender nonconformity, and gender discordance in children and adoelscents. J Am Acad Child Adolesc Psychiatry 2012;51:957–74.

33. Office on Women's Health. Illnesses and disabilities: sexual and reproductive health. Available at http://www.womenshealth.gov/illnesses-disabilities/your-health/sexuality-reproductive-health.cfm.
34. McIntyre P. Adolescent friendly health services: an agenda for change. Geneva, Switzerland: World Health Organization; 2002.
35. CDC. U.S. medical eligibility criteria for contraceptive use 2010. MMWR 2010;59(No. RR-4).
36. CDC. Sexually transmitted diseases treatment guidelines, 2010. MMWR 2010;59(No. RR-12).
37. Jaccard J. Careful, current, and consistent: tips to improve contraceptive use. Washington, DC: National Campaign to Prevent Teen Pregnancy; 2010.
38. Trussell J. Contraceptive failure in the United States. Contraception 2011;83:397–404.
39. Winner B, Peipert J, Zhao Q, et al. Effectiveness of long-acting reversible contraception. N Engl J Med 2012;366:1998–2007.
40. Beerthuizen R. State-of-the-art of non-hormonal methods of contraception: V. Female sterilisation. Eur J Contracept Reprod Health Care 2010;15:124–35.
41. Michielsen D, Beerthuizen R. State-of-the art of non-hormonal methods of contraception: VI. Male sterilisation. Eur J Contracept Reprod Health Care 2010;15:136–49.
42. CDC. U.S. Selected practice recommendations for contraceptive use 2013. MMWR 2013;62(No. RR-5).
43. Kerns J, Westhoff C, Morroni C, Murphy P. Partner influence on early discontinuation of the pill in a predominantly Hispanic population. Perspect Sex Reprod Health 2003;35:256–60.
44. Forste R, Morgan J. How relationships of US men affect contraceptive use and efforts to prevent STD. Fam Plann Perspect 1998;30:56–62.
45. Severy L, Silver S. Two reasonable people: joint decision-making in contraceptive choice and use. In: LJ S, ed. Advances in population: psychosocial perspectives. London, UK: Jessica Kingsley Publishers; 1994:207–27.
46. Grady W, Klepinger D. BIlly J, Cubbins L. The role of relationship power in couple decisions about contraception in the US. J Biosoc Sci 2010;42:307–25.
47. Jaccard J. Unlocking the contraceptive conundrum. Washington, DC: The National Campaign to Prevent Teen and Unplanned Pregnancy; 2009.
48. Pallitto CC, Campbell JC, O'Campo P. Is intimate partner violence associated with unintended pregnancy? A review of the literature. Trauma Violence Abuse 2005;6:217–35.
49. Paterno MT, Jordan ET. A review of factors associated with unprotected sex among adult women in the United States. J Obstet Gynecol Neonatal Nurs 2012;41:258–74.
50. Rehm J, Shield KD, Joharchi N, Shuper PA. Alcohol consumption and the intention to engage in unprotected sex: systematic review and meta-analysis of experimental studies. Addiction 2012;107:51–9.
51. Harper C, Balistreri E, Boggess J, Leon K, Darney P. Provision of hormonal contraceptives without a mandatory pelvic examination: the first stop demonstration project. Fam Plann Perspect 2001;33:13–8.
52. Stormo A, Hawkins N, Cooper C, Saraiya M. The pelvic examination as a screening tool: practices of US physicians. Arch Intern Med 2011;171:2053–4.
53. Labbok M, Perez A, Valdez V, et al. The Lactational Amenorrhea Method (LAM): a postpartum introductory family planning method with policy and program implications. Adv Contracept Deliv Syst 1994;10:93–109.
54. Huber L, Hogue C, Stein A, et al. Contraceptive use and discontinuation: findings from the contraceptive history, initiation and choice study. Am J Obstet Gynecol 2006;194:1290–5.
55. CDC. Guide to community preventive services: prevention of HIV/AIDS, other STIs and pregnancy, group-based comprehensive risk reduction interventions for adolescents. Atlanta, GA: US Department of Health and Human Services, CDC; 2011. Available at www.thecommunityguide.org/hiv/riskreduction.html.
56. Kirby DB, Laris BA, Rolleri LA. Sex and HIV education programs: their impact on sexual behaviors of young people throughout the world. J Adolesc Health 2007;40:206–17.
57. Oringanje C, Meremikwu MM, Eko H, Esu E, Meremikwu A, Ehiri JE. Interventions for preventing unintended pregnancies among adolescents. Cochrane Database Syst Rev 2009:CD005215.
58. English A. State minor consent laws: a summary. 3rd ed. Washington, DC: Center for Adolescent Health and the Law; 2010.
59. US Department of Health and Human Services. State requirements for reporting child abuse. Washington, DC: US Department of Health and Human Services; 2013. Available at http://www.childwelfare.gov/systemwide/laws_policies/state.
60. Guldi M. Fertility effects of abortion and birth control pill access for minors. Demography 2008;45:817–27.
61. Klein JD, McNulty M, Flatau CN. Adolescents' access to care: teenagers' self-reported use of services and perceived access to confidential care. Arch Pediatr Adolesc Med 1998;152:676–82.
62. Lehrer JA, Pantell R, Tebb K, Shafer MA. Forgone health care among U.S. adolescents: associations between risk characteristics and confidentiality concern. J Adolesc Health 2007;40:218–26.
63. Lyren A, Kodish E, Lazebnik R, O'Riordan MA. Understanding confidentiality: perspectives of African American adolescents and their parents. J Adolesc Health 2006;39:261–5.
64. Reddy DM, Fleming R, Swain C. Effect of mandatory parental notification on adolescent girls' use of sexual health care services. JAMA 2002;288:710–4.
65. Vo DX, Pate OL, Zhao H, Siu P, Ginsburg KR. Voices of Asian American youth: important characteristics of clinicians and clinical sites. Pediatrics 2007;120:e1481–93.
66. Zabin LS, Stark HA, Emerson MR. Reasons for delay in contraceptive clinic utilization. Adolescent clinic and nonclinic populations compared. J Adolesc Health 1991;12:225–32.
67. Zavodny M. Fertility and parental consent for minors to receive contraceptives. Am J Public Health 2004;94:1347–51.
68. Klein J. Achieving quality health services for adolescents: policy statement of the Committee on Adolescence, American Academy of Pediatrics. Pediatrics 2008;121:1263.
69. Ford C, English A, Sigman G. Confidential health care for adolescents: position paper for the Society for Adolescent Medicine. J Adolesc Health 2004;35:160–7.
70. American College of Obstetricians and Gynecologists. Confidentiality in adolescent health care. guidelines for adolescent health care. 2nd ed. Washington, DC: American College of Obstetricians and Gynecologists; 2011.
71. Anderson NLR, Koniak-Griffin D, Keenan CK, Uman G, Duggal BR, Casey C. Evaluating the outcomes of parent-child family life education. Sch Inq Nurs Pract 1999;13:211–38.
72. Guilamo-Ramos V, Bouris A, Jaccard J, Gonzalez B, McCoy W, Aranda D. A parent-based intervention to reduce sexual risk behavior in early adolescence: building alliances between physicians, social workers, and parents. J Adolesc Health 2011;48:159–63.

73. Blake SM, Simkin L, Ledsky R, Perkins C, Calabrese JM. Effects of a parent-child communications intervention on young adolescents' risk for early onset of sexual intercourse. Fam Plann Perspect 2001;33:52–61.
74. Brody GH, Murry VM, Gerrard M, et al. The Strong African American Families Program: translating research into prevention programming. Child Dev 2004;75:900–17.
75. Evans WD, Davis KC, Ashley OS, Blitstein J, Koo H, Zhang Y. Efficacy of abstinence promotion media messages: findings from an online randomized trial. J Adolesc Health 2009;45:409–16.
76. Forehand R, Armistead L, Long N, et al. Efficacy of a parent-based sexual-risk prevention program for African American preadolescents: a randomized controlled trial. Arch Pediatr Adolesc Med 2007;161:1123–9.
77. Haggerty KP, Skinner ML, MacKenzie EP, Catalano RF. A randomized trial of Parents Who Care: effects on key outcomes at 24-month follow-up. Prev Sci 2007;8:249–60.
78. Huston RL, Martin LJ, Foulds DM. Effect of a program to facilitate parent-child communication about sex. Clin Pediatr (Phila) 1990;29:626–33.
79. Lederman RP, Chan W, Roberts-Gray C. Sexual risk attitudes and intentions of youth aged 12–14 years: survey comparisons of parent-teen prevention and control groups. Behavioral Medicine (Washington, DC) 2004;29:155–63.
80. Lederman RP, Chan W, Roberts-Gray C. Parent-adolescent relationship education (PARE): program delivery to reduce risks for adolescent pregnancy and STDs. Behav Med 2008;33:137–43.
81. Lefkowitz ES, Sigman M, Au TK. Helping mothers discuss sexuality and AIDS with adolescents. Child Dev 2000;71:1383–94.
82. Miller BC, Norton MC, Jenson GO. Pregnancy prevention programs: Impact Evaluation of Facts and Feelings: a home-based video sex education curriculum. Fam Relat 1993;42:392–400.
83. Murry VM, Berkel C, Brody GH, Gerrard M, Gibbons FX. The Strong African American Families program: longitudinal pathways to sexual risk reduction. J Adolesc Health 2007;41:333–42.
84. Schuster MA, Corona R, Elliott MN, et al. Evaluation of Talking Parents, Healthy Teens, a new worksite based parenting programme to promote parent-adolescent communication about sexual health: randomised controlled trial. BMJ 2008;337:a308.
85. Stanton B, Cole M, Galbraith J, et al. Randomized trial of a parent intervention: parents can make a difference in long-term adolescent risk behaviors, perceptions, and knowledge. Arch Pediatr Adolesc Med 2004;158:947–55.
86. Stanton BF, Li X, Galbraith J, et al. Parental underestimates of adolescent risk behavior: a randomized, controlled trial of a parental monitoring intervention. J Adolesc Health 2000;26:18–26.
87. American Academy of Pediatrics. Parents matter: your children want to learn about the facts of life from you. Elk Grove Village, IL: American Academy of Pediatrics; ND. Available at http://www2.aap.org/visit/ParentsMatterBrochure.pdf.
88. Hamilton B. Births: final Data for 2010. National Vital Statistics Reports 2012;61(1).
89. Olds DL, Henderson CR Jr, Tatelbaum R, Chamberlin R. Improving the life-course development of socially disadvantaged mothers: a randomized trial of nurse home visitation. Am J Public Health 1988;78:1436–45.
90. Olds DL, Kitzman H, Cole R, et al. Effects of nurse home-visiting on maternal life course and child development: age 6 follow-up results of a randomized trial. Pediatrics 2004;114:1550–9.
91. Olds DL, Kitzman H, Hanks C, et al. Effects of nurse home visiting on maternal and child functioning: age-9 follow-up of a randomized trial. Pediatrics 2007;120:e832–45.
92. Rabin JM, Seltzer V, Pollack S. The long term benefits of a comprehensive teenage pregnancy program. Clin Pediatr (Phila) 1991;30:305–9.
93. Avellar S, Paulsell D, Sama-Miller E, Del Grosso P. Home visiting evidence of effectiveness review. Washington, DC: US Department of Health and Human Services, Office of Planning, Research and Evaluation, Administration for Children and Families; 2012.
94. Black M, Bentley M, Papas M, et al. Delaying second births among adolescent mothers: a randomized, controlled trial of a home-based mentoring program. Pediatrics 2006;118:1087–99.
95. American Academy of Pediatrics. Counseling the adolescent about pregnancy options. Pediatrics 2006;101:938–40.
96. American College of Obstetricians and Gynecologists. Guidelines for women's health care: a resource manual. 3rd ed. Washington, DC: American College of Obstetricians and Gynecologists; 2007.
97. American College of Obstetricians and Gynecologists. Guidelines for perinatal care. 6th ed. Washington, DC: American College of Obstetricians and Gynecologists; 2007.
98. Ventura S, Curtin S, Abma J, Henshaw S. Estimated pregnancy rates and rates of pregnancy outcomes for the US: 1990–2008. Natl Vital Stat Rep 2012;60(7).
99. Jones J. Adoption experiences of women and men and demand for children to adopt by women 18–44 years of age in the United States, 2002. Vital Health Stat 2008;23(7).
100. American Society for Reproductive Medicine in collaboration with the Society for Reproductive Endocrinology and Infertility. Optimizing natural fertility. Fertil Steril 2008;90(Suppl):S1–6.
101. American Society for Reproductive Medicine. Optimal evaluation of the infertile female. Fertil Steril 2006;86.
102. American Urological Association. The optimal evaluation of the infertile male: AUA best practice statement. Linthicum, MD: American Urological Association; 2010.
103. Frey K, Navarro S, Kotelchuck M, Lu M. The clinical content of preconception care: preconception care for men. Am J Obstet Gynecol 2008;6:S389–95.
104. US Preventive Services Task Force. Folic acid to prevent neural tube defects. Rockville, MD: US Department of Health and Human Services, Agency for Healthcare Research and Quality; 2009. Available at http://www.uspreventiveservicestaskforce.org/uspstf/uspsnrfol.htm.
105. Jack B, Atrash H, Coonrod D, Moos M, O'Donnell J, Johnson K. The clinical content of preconception care: an overview and preparation of this supplement. Am J Obstet Gynecol 2008;199(Suppl B):S266–79.
106. US Preventive Services Task Force. Screening for intimate partner violence and abuse of elderly and vulnerable adults. Rockville, MD: US Department of Health and Human Services, Agency for Healthcare Research and Quality; 2013. Available at http://www.uspreventiveservicestaskforce.org/uspstf/uspsipv.htm.
107. US Preventive Services Task Force. Screening and Behavioral Counseling Interventions in Primary Care to Reduce Alcohol Misuse. AHRQ, US Department of Health and Human Services; 2013.
108. American College of Obstetricians and Gynecologists. Committee Opinion No. 483: primary and preventive care: periodic assessments. Obstet Gynecol 2011;117:1008–15.
109. Committee on Practice and Ambulatory Medicine, Bright Futures Periodicity Schedule Workgroup. 2014 recommendations for pediatric preventive health care. Pediatrics 2014;133;568.
110. US Preventive Services Task Force. Screening for illicit drug use. Rockville, MD: US Department of Health and Human Services, Agency for Healthcare Research and Quality; 2008.

111. US Preventive Services Task Force. Counseling and interventions to prevent tobacco use and tobacco-caused disease in adults and pregnant women. Rockville, MD: US Department of Health and Human Services, Agency for Healthcare Research and Quality; 2009. Available at http://www.uspreventiveservicestaskforce.org/uspstf/uspstbac2.htm.
112. US Preventive Services Task Force. Primary care interventions to prevent tobacco use in children and adolescents. Rockville, MD: US Department of Health and Human Services, Agency for Healthcare Research and Quality; 2013.
113. CDC. Advisory Committee on Immunization Practices (ACIP) recommended immunization schedules for persons aged 0 through 18 years and adults aged 19 years and older—United States, 2013. MMWR 2013;62(Suppl 1):1–19.
114. US Preventive Services Task Force. Screening for depression in adults. Rockville, MD: US Department of Health and Human Services, Agency for Healthcare Research and Quality; 2009. Available at http://www.uspreventiveservicestaskforce.org/uspstf/uspsaddepr.htm.
115. US Preventive Services Task Force. Major depressive disorder in children and adolescents. Rockville, MD: US Department of Health and Human Services, Agency for Healthcare Research and Quality; 2009. Available at http://www.uspreventiveservicestaskforce.org/uspstf/uspschdepr.htm.
116. American Psychiatric Association. Practice guideline for the assessment and treatment of patients with suicidal behaviors. Am J Psychiatry 2003;160(Suppl):1–60.
117. American Academy of Child and Adolescent Psychiatry. Practice parameter for the assessment and treatment of children and adolescents with suicidal behavior. J Am Acad Child Adolesc Psychiatry 2001;40(Suppl):24S–51S.
118. US Preventive Services Task Force. Screening for obesity in children and adolescents: US Preventive Services Task Force recommendation statement. Rockville, MD: US Department of Health and Human Services, Agency for Healthcare Research and Quality; 2010. Available at http://www.uspreventiveservicestaskforce.org/uspstf/uspschobes.htm.
119. US Preventive Services Task Force. Screening for and management of obesity in adults. Rockville, MD: US Department of Health and Human Services, Agency for Healthcare Research and Quality; 2012. Available at http://www.uspreventiveservicestaskforce.org/uspstf11/obeseadult/obesers.htm.
120. US Preventive Services Task Force. Screening for high blood pressure in adults. Rockville, MD: US Department of Health and Human Services, Agency for Healthcare Research and Quality; 2007. Available at http://www.uspreventiveservicestaskforce.org/uspstf/uspshype.htm.
121. US Department of Health and Human Services. The seventh report of the Joint National Committee on the Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Washington, DC: US Department of Health and Human Services; 2004.
122. US Preventive Services Task Force. Screening for type 2 diabetes mellitus in adults. Rockville, MD: US Department of Health and Human Services, Agency for Healthcare Research and Quality; 2008. Available at http://www.uspreventiveservicestaskforce.org/uspstf/uspsdiab.htm.
123. CDC. Revised recommendations for HIV testing of adults, adolescents and pregnant women in health care settings. MMWR 2006;55(No. RR-14).
124. CDC. Recommendations for the identification of chronic hepatitis C virus infection among persons born during 1945–1965. MMWR 2012;61(No. RR-4).
125. CDC. Male chlamydia screening consultation. Atlanta, GA: US Department of Health and Human Services, DCD; 2006. Available at http://www.cdc.gov/std/chlamydia.
126. Burstein GR, Eliscu A, Ford K, et al. Expedited partner therapy for adolescents diagnosed with chlamydia or gonorrhea: a position paper of the Society for Adolescent Medicine. J Adolesc Health 2009;45:303–9.
127. American Academy of Pediatrics. Promoting healthy sexual development and sexuality. Bright Futures: guidelines for health supervision of infants, children, and adolescents. Elk Grove, IL: American Academy of Pediatrics; 2007.
128. US Preventive Services Task Force. Screening for gonorrhea. Rockville, MD: US Department of Health and Human Services, Agency for Healthcare Research and Quality; 2005. Available at http://www.uspreventiveservicestaskforce.org/uspstf/uspsgono.htm.
129. US Preventive Services Task Force. Screening for HIV. Rockville, MD: US Department of Health and Human Services, Agency for Healthcare Research and Quality; 2013. Available at http://www.uspreventiveservicestaskforce.org/uspstf/uspshivi.htm.
130. US Preventive Services Task Force. Screening for hepatitis C virus infection in adults. Rockville, MD: US Department of Health and Human Services, Agency for Healthcare Research and Quality; 2013. Available at http://www.uspreventiveservicestaskforce.org/uspstf12/hepc/hepcfinalrs.htm#recommendations.
131. CDC. Recommendations on the use of quadrivalent human papillomavirus vaccine in males—Advisory Committee on Immunization Practices (ACIP). MMWR 2011;60:1705–8.
132. US Preventive Services Task Force. Behavioral counseling to prevent sexually transmitted infections. Rockville, MD: US Department of Health and Human Services, Agency for Healthcare Research and Quality; 2008. Available at http://www.uspreventiveservicestaskforce.org/uspstf/uspsstds.htm.
133. Kamb ML, Fishbein M, Douglas JM Jr, et al. Efficacy of risk-reduction counseling to prevent human immunodeficiency virus and sexually transmitted diseases: a randomized controlled trial. Project RESPECT Study Group. JAMA 1998;280:1161–7.
134. US Preventive Services Task Force. Risk assessment, genetic counseling, and genetic testing for BRCA-related cancer in women. Rockville, MD: US Department of Health and Human Services, Agency for Healthcare Research and Quality; 2013. Available at http://www.uspreventiveservicestaskforce.org/uspstf/uspsbrgen.htm.
135. US Preventive Services Task Force. Medications for risk reduction of primary breast cancer in women. Rockville, MD: US Department of Health and Human Services, Agency for Healthcare Research and Quality; 2013. Available at http://www.uspreventiveservicestaskforce.org/uspstf/uspsbrpv.htm.
136. US Preventive Services Task Force. Screening for cervical cancer. Rockville, MD: US Department of Health and Human Services, Agency for Healthcare Research and Quality; 2012. Available at www.uspreventiveservicestaskforce.org/uspstf11/cervcancer/cervcancerrs.htm.
137. Massad L, Einstein M, Huh W, et al. 2012 updated consensus guidelines for the management of abnormal cervical cancer screening tests and cancer precursors. Obstet Gynecol 2013;121:829–46.
138. Braverman P, Breech L. Gynecologic examination for adolescents in the pediatric office setting. Pediatrics 2010;126:583–90.
139. Smith RA, Brooks D, Cokkinides V, Saslow D, Brawley OW. Cancer screening in the United States, 2013: a review of current American Cancer Society guidelines, current issues in cancer screening, and new guidance on cervical cancer screening and lung cancer screening. CA Cancer J Clin 2013;63:88–105.

140. US Preventive Services Task Force. Screening for breast cancer. Rockville, MD: US Department of Health and Human Services, Agency for Healthcare Research and Quality; 2009. Available at http://www.uspreventiveservicestaskforce.org/uspstf/uspsbrca.htm.
141. Marcell A, Bell D, Joffe A. The male genital examination: a position paper of the Society for Adolescent Health and Medicine: a position paper of the Society for Adolescent Health and Medicine. J Adolesc Health 2012;50:424–5.
142. Riley W, Moran J, Corso L, Beitsch L, Bialek R, Cofsky A. Defining quality improvement in public health. J Public Health Manag Pract 2010;16:5–7.
143. National Quality Forum. ABCs of measurement. Washington, DC: National Quality Forum; 2011. Available at http://www.qualityforum.org/Measuring_Performance/ABCs_of_Measurement.aspx.
144. Berwick D, Nolan T, Whittington J. The Trip Aim: care, health and cost. Health Aff 2008;27:759–69.
145. CDC. National Public Health Performance Standards Program. Atlanta, GA: US Department of Health and Human Services, CDC; 2012. Available at www.cdc.gov/od/ocphp/nphpsp.
146. Berwick D. A user's manual for the IOM's "Quality Chasm" Report. Health Affairs 2002;21:80–90.
147. Langley G, Nolan K, Nolan T, Norman C, Provost L. The Improvement Guide: A practical approach to enhancing organizational performance. 2nd ed. San Francisco, CA: Jossey-Bass Publishers; 2009.
148. Government Accountability Office. Performance measurement and evaluation: definitions and relationships. Washington, DC: Government Accountability Office; 2011.
149. Donabedian A. The quality of care. JAMA 1988;260:1743–8.
150. Fowler C. Title X Family Planning Program Performance Information and Monitoring System (PIMS): description of proposed performance measures. Washington, DC: Research Triangle Institute; 2012.
151. California Family Health Council. Performance measures: guiding quality improvement for family planning services. Los Angeles, CA: California Family Health Council; 2011. Available at http://www.cfhc.org/TitleX/PerformanceMeasures/default.htm.
152. John Snow International. The Regional Quality Indicators Project (RQIP). Boston, MA: John Snow International; 2012. Available at http://www.jsi.com/JSIInternet/USHealth/project/display.cfm?ctid=na&cid=na&tid=40&id=2621.
153. Family Planning Councils of America. Performance Measurement System. Los Angeles, CA: Family Planning Councils of America; ND. Available at http://www.fpcai.org/pms.html.
154. Palmer R. Process-based measures of quality: the need for detailed clinical data in large health care databases. Ann Intern Med 1997;127:733–8.
155. Bouchet B. Health manager's guide: monitoring the quality of primary care. Bethesda, MD: Center for Human Services; ND. Available at http://pdf.usaid.gov/pdf_docs/PNACK584.pdf.
156. Institute for Health Care Improvement. How to improve. Cambridge, MA: Institute for Health Care Improvement; 2014. Available at http://www.ihi.org/knowledge/Pages/HowtoImprove/default.aspx.

Appendix A

How the Recommendations Were Developed

The recommendations were developed jointly under the auspices of CDC's Division of Reproductive Health (DRH) and the Office of Population Affairs (OPA), in consultation with a wide range of experts and key stakeholders. A multistage process that drew on established procedures for developing clinical guidelines (*1,2*) was used to develop the recommendations. In April 2010, an Expert Work Group (EWG) comprising family planning clinical providers, program administrators, representatives from relevant federal agencies, and representatives from professional medical organizations was created to advise OPA and CDC on the structure and content of the revised recommendations and to help make the recommendations more feasible and relevant to the needs of the field. This group made two key initial recommendations: 1) to examine the scientific evidence for three priority areas of focus identified as key components of family planning service delivery, (i.e., counseling and education, serving adolescents, and quality improvement); and 2) to guide providers of family planning services in the use of various recommendations for how to provide clinical care to women and men.

Developing Recommendations on Counseling, Adolescent Services, and Quality Improvement

Systematic reviews of the published literature from January 1985 through December 2010 were conducted for each priority topic to identify evidence-based and evidence-informed approaches to family planning service delivery. Standard methods for conducting the reviews were used, including the development of key questions and analytic frameworks, the identification of the evidence base through a search of the published as well as "gray literature" (i.e., studies published somewhere other than in a peer-reviewed journal), and a synthesis of the evidence in which findings were summarized and the quality of individual studies was considered, using the methodology of the U.S. Preventive Services Task Force (USPSTF) (*3*). Eight databases were searched (i.e., MEDLINE, PsychInfo, PubMed, CINAHL, Cochrane, EMBASE, POPLINE, and the U.K. National Clearinghouse Service Economic Evaluation Database) and were restricted to literature from the United States and other developed countries. Summaries of the evidence used to prepare these recommendations will appear in background papers that will be published separately.

In May 2011, three technical panels (one for each priority topic) comprising subject matter experts were convened to consider the quality of the evidence and suggest what recommendations might be justified on the basis of the evidence. CDC and OPA used this feedback to develop core recommendations for counseling, serving adolescents, and quality improvement. EWG members subsequently reviewed these core recommendations; EWG members differed from the subject matter experts in that they were more familiar with the family planning service delivery context and could comment on the feasibility and appropriateness of the recommendations as well as on their scientific justification. EWG members met to consider the core recommendations using 1) the quality of the evidence; 2) the positive and negative consequences of implementing the recommendations on health outcomes, costs or cost-savings, and implementation challenges; and 3) the relative importance of these consequences (e.g., the ability of the recommendations to have a substantial effect on health outcomes may be weighed more than the logistical challenges of implementing them) (*1*). In certain cases, when the evidence was inconclusive or incomplete, recommendations were made on the basis of expert opinion (see Appendix B). Finally, CDC and OPA staff considered the feedback from EWG members when finalizing the core recommendations and writing this report.

Developing Recommendations on Clinical Services

DRH and OPA staff members synthesized recommendations for clinical care for women and for men that were developed by >35 federal and professional medical organizations. They were assisted in this effort by staff from OPA's Office of Family Planning Male Training Center and from CDC's Division of STD Prevention, Division of Violence Prevention, Division of Immunization Services, and Division of Cancer Prevention and Control. The synthesis was needed because clinical recommendations are sometimes inconsistent with each other and can vary by the extent to which they are evidence-based. The clinical recommendations addressed contraceptive services, achieving pregnancy, basic infertility services, preconception health services, sexually transmitted disease services, and related health-care services.

An attempt was made to apply the Institute of Medicine's criteria for clinical practice guidelines when deciding which professional medical organizations to include in the review (*2*). However, many organizations did not articulate the process used to develop the recommendations fully, and many did not

conduct comprehensive and systematic reviews of the literature. In the end, to be included in the synthesis, the recommending organization had to be a federal agency or major professional medical organization that represents established medical disciplines. In addition, a recommendation had to be made on the basis of an independent review of the evidence or expert opinion and be considered a primary source that was developed for the United States.

In July 2011, two technical panels comprising subject matter experts on clinical services for women and men were convened to review the synthesis of federal and professional medical recommendations, reconcile inconsistent recommendations, and provide individual feedback to CDC and OPA about the implications for family planning service delivery. CDC and OPA used this individual feedback to develop core recommendations for clinical services. The core recommendations were subsequently reviewed by EWG members, and feedback was used to finalize the core recommendations and write this report.

Members of the technical panels recommended that contraceptive services, pregnancy testing and counseling, services to achieve pregnancy, basic infertility care, STD services, and other preconception health services should be considered family planning services. This feedback considered federal statute and regulation, CDC and USPSTF recommendations for clinical care, and EWG members' opinion.

Because CDC's preconception health recommendations include many services, the panel narrowed the range of preconception services that were included by using the following criteria: 1) the Select Panel on Preconception Care (*4*) had assigned an A or B recommendation to that service for women, which means that there was either good or fair evidence to support the recommendation that the condition be considered in a preconception care evaluation (Table 1), or 2) the service was included among recommendations made by experts in preconception health for males (*5*). Services for men that addressed health conditions that affect reproductive capacity or pregnancy outcomes directly were included as preconception health; services that addressed men's health but that were not related directly to pregnancy outcomes were considered to be related preventive health services.

The Expert Work Group noted that more preventive services are recommended than can be offered feasibly in some settings. However, a primary purpose of this report is to set a broad framework within which individual clinics will tailor services to meet the specific needs of the populations that they serve. In addition, EWG members identified specific subgroups that should have the greatest priority for preconception health services (i.e., those trying to achieve pregnancy and those at high risk of unintended pregnancy). Future operational research should provide more information about how to deliver these services most efficiently during multiple visits to clients with diverse needs.

Determining How Clinical Services Should Be Provided

Various federal agencies and professional medical associations have made recommendations for how to provide family planning services. When considering these recommendations, the Expert Work Group used the following hierarchy:

- Highest priority was given to CDC guidelines because they are developed after a rigorous review of scientific evidence. CDC guidelines tailor recommendations for higher risk individuals, (whereas USPSTF focuses on average risk individuals), who are more representative of the clients seeking family planning services.
- When no CDC guideline existed to guide the recommendations, the relevant USPSTF A or B recommendations (which indicate a high or moderate certainty that the benefit is moderate to substantial) were used. USPSTF recommendations are made on the basis of a thorough review of the available evidence.
- If neither a CDC nor a USPSTF A or B recommendation existed, the recommendations of selected major professional medical associations were considered as resources. The American Academy of Pediatrics' (AAP) Bright Futures guidelines (*6*) were used as the primary source of recommendations for adolescents when no CDC or USPSTF recommendations existed.
- For a limited number of recommendations, there were no federal or major professional medical recommendations, but the service was recommended by EWG members on the basis of expert opinion for family planning clients.

In some cases, a service was graded as an I recommendation by USPSTF for the general population (an I recommendation means that the current evidence is insufficient to assess the balance of benefits and harms of the service, so if the service is offered, patients should be informed of this fact), but either CDC, EWG members, or another organization recommended the service for women or men seeking family planning services. The situations in which this occurred and the reasons why the service was recommended despite its receiving an I recommendation by USPSTF have been summarized (Table 2). The approach used to consider the evidence and make recommendations that are used by USPSTF have been summarized (Tables 3 and 4) (*7*).

References

1. GRADE Working Group. GRADE: going from evidence to recommendations. BMJ 2008;336:1049–51.
2. Institute of Medicine. Clinical practice guidelines we can trust. Washington, DC: The National Academies Press; 2011. Available at http://www.nap.edu/catalog.php?record_id=13058.
3. US Preventive Services Task Force. USPSTF: methods and processes. Rockville, MD: US Department of Health and Human Services, Agency for Healthcare Research and Quality; 2013. Available at http://www.uspreventiveservicestaskforce.org/methods.htm.
4. Jack BW, Atrash H, Coonrod D, Moos M, O'Donnell J, Johnson K. The clinical content of preconception care: an overview and preparation of this supplement. Am J Obstet Gynecol 2008;199(Suppl 2):S266–79.
5. Frey KA, Navarro S, Kotelchuck M, Lu M. The clinical content of preconception care: preconception care for men. Am J Obstet Gynecol 2008;199(Suppl 2):S389–95.
6. Committee on Practice and Ambulatory Medicine, Bright Futures Periodicity Schedule Workgroup. 2014 recommendations for pediatric preventive health care. Pediatrics 2014;133;568.
7. US Preventive Services Task Force. Grade definitions. Rockville, MD: US Department of Health and Human Services, Agency for Healthcare Research and Quality; 2013. Available at http://www.uspreventiveservicestaskforce.org/uspstf/grades.htm.

TABLE 1. Select Panel on Preconception Care grading system

Quality of the evidence*	
I-a	Evidence was obtained from at least one properly conducted, randomized, controlled trial that was performed with subjects who were not pregnant.
I-b	Evidence was obtained from at least one properly conducted, randomized, controlled trial that was done not necessarily before pregnancy.
II-1	Evidence was obtained from well-designed, controlled trials without randomization.
II-2	Evidence was obtained from well-designed cohort or case-control analytic studies, preferably conducted by more than one center or research group.
II-3	Evidence was obtained from multiple-time series with or without the intervention, or dramatic results in uncontrolled experiments.
III	Opinions were gathered from respected authorities on the basis of clinical experience, descriptive studies and case reports, or reports of expert committees.
Strength of the recommendation	
A	There is good evidence to support the recommendation that the condition be considered specifically in a preconception care evaluation.
B	There is fair evidence to support the recommendation that the condition be considered specifically in a preconception care evaluation.
C	There is insufficient evidence to recommend for or against the inclusion of the condition in a preconception care evaluation, but recommendation to include or exclude may be made on other grounds.
D	There is fair evidence to support the recommendation that the condition be excluded in a preconception care evaluation.
E	There is good evidence to support the recommendation that the condition be excluded in a preconception care evaluation.

Source: Jack B, Atrash H, Coonrod D, Moos M, O'Donnell J, Johnson K. The clinical content of preconception care: an overview and preparation of this supplement. Am J Obstet Gynecol 2008;199(6 Suppl 2):S266–79.

TABLE 2. Services included in these recommendations that received a U.S. Preventive Services Task Force (USPSTF) I recommendation

Service/screen	USPSTF recommendation	Why the service is recommended despite a USPSTF I recommendation
Alcohol	I for adolescents	The recommendations are consistent with CDC's recommendations on preconception health and AAP's Bright Futures* guidelines.
Other drugs	I for adolescents and adults	The recommendations are consistent with CDC's recommendations on preconception health and AAP's Bright Futures guidelines.
Clinical breast exam	I for all women	No CDC recommendation exists, but ACOG and ACS recommend conducting clinical breast exams, and the Expert Work Group endorsed the ACOG recommendation.
Chlamydia	I for all males	The recommendations are consistent with CDC's STD treatment guidelines.
Gonorrhea	I for all males	The recommendations are consistent with CDC's STD treatment guidelines.

Source: US Preventive Services Task Force. USPSTF recommendations. Available at http://www.uspreventiveservicestaskforce.org/recommendations.htm.
Abbreviations: AAP = American Academy of Pediatrics; ACS = American Cancer Society; ACOG = American Congress of Obstetricians and Gynecologists; STD = sexually transmitted disease.
* Source: Committee on Practice and Ambulatory Medicine, Bright Futures Periodicity Schedule Workgroup. 2014 recommendations for pediatric preventive health care. Pediatrics 2014;133;568.

TABLE 3. U.S. Preventive Services Task Force (USPSTF) grades, definitions, and suggestions for practice

Grade	Definition	Suggestions for practice
A	USPSTF recommends the service. There is high certainty that the net benefit is substantial.	This service should be offered or provided.
B	USPSTF recommends the service. There is high certainty that the net benefit is moderate, or there is moderate certainty that the net benefit is moderate to substantial.	This service should be offered or provided.
C	Clinicians may provide this service to selected patients depending on individual circumstances. However, for a majority of persons without signs or symptoms there is likely to be only a limited benefit from this service.	This service should be offered or provided only if other considerations support the offering or providing the service in an individual patient.
D	USPSTF recommends against the service. There is moderate or high certainty that the service has no net benefit or that the harms outweigh the benefits.	Use of this service should be discouraged.
I Statement	USPSTF concludes that the current evidence is insufficient to assess the balance of benefits and harms of the service. Evidence is lacking, of poor quality, or conflicting, and the balance of benefits and harms cannot be determined.	The clinical considerations section of USPSTF recommendation statement should be consulted. If the service is offered, patients should be educated about the uncertainty of the balance of benefits and harms.

Source: US Preventive Services Task Force. USPSTF: methods and processes. Available at http://www.uspreventiveservicestaskforce.org/methods.htm.

TABLE 4. Levels of certainty regarding net benefit

Level of certainty*	Description
High	The available evidence usually includes consistent results from well-designed, well-conducted studies in representative primary care populations. These studies assess the effects of the preventive service on health outcomes. This conclusion is therefore unlikely to be strongly affected by the results of future studies.
Moderate	The available evidence is sufficient to determine the effects of the preventive service on health outcomes, but confidence in the estimate is constrained by such factors as • the number, size, or quality of individual studies; • inconsistency of findings across individual studies; • limited generalizability of findings to routine primary care practice; and • lack of coherence in the chain of evidence. As more information becomes available, the magnitude or direction of the observed effect could change, and this change may be large enough to alter the conclusion.
Low	The available evidence is insufficient to assess effects on health outcomes is insufficient because of • the limited number or size of studies, • important flaws in study design or methods, • inconsistency of findings across individual studies, • gaps in the chain of evidence, • findings not generalizable to routine primary care practice, • lack of information on important health outcomes, or • more information required to allow estimation of effects on health outcomes.

Source: US Preventive Services Task Force. USPSTF: methods and processes. Available at http://www.uspreventiveservicestaskforce.org/methods.htm.

* The US Preventive Services Task Force (USPSTF) defines certainty as the likelihood that the USPSTF assessment of the net benefit of a preventive service is correct. The net benefit is defined as benefit minus harm of the preventive service as implemented in a general, primary care population. USPSTF assigns a certainty level on the basis of the nature of the overall evidence available to assess the net benefit of a preventive service.

Appendix B

The Evidence, Potential Consequences, and Rationales for Core Recommendations

Sixteen core recommendations that were considered by the Expert Work Group (EWG) are presented below. Each recommendation is accompanied by a summary of the relevant evidence (full summaries of which will be published separately), a list of potential consequences of implementing the recommendation, and its rationale. When considering the recommendations, the Expert Work Group was divided into two groups (one comprising seven members and the other five members), and each group considered separate recommendations.

Definition of Family Planning Services

Recommendation: Primary care providers should offer the following family planning services: contraceptive services for women and men who want to prevent pregnancy and space births, pregnancy testing and counseling, help for clients who wish to achieve pregnancy, basic infertility services, sexually transmitted disease (STD) services and preconception health services to improve the health of women, men, and infants.

Quality of evidence: A systematic review was not conducted; the recommendation was made on the basis of federal statute and regulation (*1*,*2*), CDC clinical recommendations (*3*–*5*), and expert opinion.

Potential consequences: Adding preconception health services means that more women and men will receive preconception health services. The recommended services also will promote the health of women and men even if they do not have children. The human and financial cost of providing preconception health services might mean that fewer contraceptive and other services can be offered in some settings.

Rationale: Services to prevent and achieve pregnancy are core to the federal government's efforts to promote reproductive health. Adding preconception health as a family planning service is consistent with this mission; it emphasizes achieving a healthy pregnancy and also promotes adult health. Adding preconception health is also consistent with CDC recommendations to integrate preconception health services into primary care platforms (*3*). All seven EWG members agreed to this recommendation.

Preconception Health — Women

Recommendation: Preconception health services for women include the following screening services: reproductive life plan; medical history; sexual health assessment; intimate partner violence, alcohol, and other drug use; tobacco use; immunizations; depression; body mass index (BMI); blood pressure; chlamydia, gonorrhea, syphilis, and HIV/AIDS; and diabetes. All female clients also should be counseled about the need to take a daily supplement of folic acid. When screening results indicate the presence of a health condition, the provider should take steps either to provide or to refer the client for the appropriate further diagnostic testing and or treatment. Services should be provided in a manner that is consistent with established federal and professional medical associations' recommendations to enable clients who need services to receive them and to avoid over-screening.

Quality of evidence: A systematic review was not conducted; the recommendation was made on the basis of CDC's recommendations to improve preconception health and health care (*3*) and a review of preconception health services by an expert panel on preconception care for women (*6*).

Potential consequences: More women will receive specified preconception health services, which will improve the health of infants and women. The evidence base for preconception health is not fully established. There is a potential risk that a client with a positive screen will not be able to afford treatment if the client is uninsured and not eligible for public programs. The human and financial cost of providing preconception health services might mean that fewer contraceptive and other services can be offered.

Rationale: The potential benefits to the health of women and infants were thought by the panel to be greater than the costs, potential harms, and opportunity costs of providing these services. Implementation (e.g., training and monitoring of providers) can address the issues related to providers over-screening and not following the federal and professional medical recommendations. CDC will continue to monitor related research and modify these recommendations, as needed. Health-care reform might make follow-up care more available to low-income clients. All seven EWG members agreed to this recommendation.

Preconception Health — Men

Recommendation: Preconception health services for men include the following screening services: reproductive life plan; medical history; sexual health assessment; alcohol and other drug use; tobacco use; immunizations; depression; BMI; blood pressure; chlamydia, gonorrhea, syphilis, and HIV/AIDS; and diabetes. When screening results indicate the presence of a health condition, the provider should take

steps either to provide or to refer the client for the appropriate further diagnostic testing and or treatment. Services should be provided in a manner that is consistent with established federal and professional medical associations' recommendations to ensure that clients who need services receive them and to avoid over-screening.

Quality of evidence: A systematic review was not conducted; the recommendation was made on the basis of CDC's recommendations to improve preconception health and health care (*3*) and a review of preconception health services for men (*7*).

Potential consequences: More men will receive preconception health services, which might improve infant and men's health. The evidence base for preconception health is not well established and is less than that for women's preconception health. There is a risk of over-screening if recommendations are not followed. There is a potential risk that a client with a positive screen might not be able to afford treatment if the client is uninsured and not eligible for public programs. The human and financial cost of providing preconception health services might mean that fewer contraceptive and other services can be offered.

Rationale: The potential benefits to men and infant health were thought by the panel to be greater than the costs, potential harms, and opportunity costs of not providing these services. Implementation (e.g., training and monitoring of providers) can address the issues related to providers over-screening and not following the federal and professional medical recommendations. CDC will continue to monitor related research and modify these recommendations, as needed. Health-care reform might make follow-up care more available to low-income clients. All seven EWG members agreed to this recommendation.

Contraceptive Services — Contraceptive Counseling Steps

Recommendation: To help a client who is initiating or switching to a new method of contraception, providers should follow these steps, which are in accordance with the key principles for providing quality counseling: 1) establish and maintain rapport with the client; 2) obtain clinical and social information from the client; 3) work with the client interactively to select the most effective and appropriate contraceptive method for her or him; 4) provide a physical assessment related to contraceptive use, when warranted; and 5) provide the contraceptive method along with instructions about correct and consistent use, help the client develop a plan for using the selected method and for follow-up, and confirm understanding.

Quality of evidence: Twenty-two studies were identified that examined the impact of contraceptive counseling in clinical settings and met the inclusion criteria. Of the 16 studies that focused on adults or mixed populations (adolescents and adults) (*8–23*), 11 found a statistically significant positive impact of counseling interventions with low (*11*,*12*,*14–16*,*18–21*), moderate (*8*), or unrated (*22*) intensity on at least one outcome of interest; study designs included two cross-sectional surveys (*14*,*22*), one pre-post study (*21*), one prospective cohort study (*8*), one controlled trial (*15*), and six randomized controlled trials (RCTs) (*11*,*12*,*16*,*18–20*). Six studies examined the impact of contraceptive counseling among adolescents (*24–29*), with four finding a statistically significant positive impact of low-intensity (*27*) or moderate-intensity (*24*,*25*,*29*) counseling interventions on at least one outcome of interest; study designs included two pre-post studies (*24*,*30*), one controlled trial (*29*), and one RCT (*27*). In addition, five studies were identified that examined the impact of reminder system interventions in clinical settings on family planning outcomes and met the inclusion criteria (*31–35*); of these, two found a statistically significant positive impact of reminder systems on perfect oral contraceptive compliance, a retrospective historical nonrandomized controlled trial that examined daily reminder email messages (*31*) and a cohort study that examined use of a small reminder device that emitted a daily audible beep (*34*). In addition, two studies examined the impact of reminder systems among depot medroxyprogesterone acetate users (DMPA) (*33*,*35*) with one, a retrospective cohort study, finding a statistically significant positive impact of receiving a wallet-sized reminder card with the date of the next DMPA injection and a reminder postcard shortly before the next injection appointment on timely DMPA injections. Statements about safety and unnecessary medical examinations and tests are made on the basis of CDC guidelines on contraceptive use (*36*,*37*).

Potential consequences: Fewer clients will use methods that are not safe for them, there will be increased contraceptive use, increased use of more effective methods, increased continuation of method use, increased use of dual methods, increased knowledge, increased satisfaction with services, and increased use of repeat or follow-up services.

Rationale: Making sure that a contraceptive method is safe for an individual client is a fundamental responsibility of all providers of family planning services. Removing medical barriers to contraceptive use is key to increasing access to contraception and helping clients prevent unintended pregnancy. Consistent use of contraceptives is needed to prevent unintended pregnancies, so appropriate counseling is critical to ensure clients make the best possible choice of methods for their unique circumstances, and are supported in continued

use of the chosen method. The principles of quality counseling, from which the steps listed in the recommendations are based, are supported by a substantial body of evidence and expert opinion. Future research to evaluate the five principles will be monitored and the recommendations modified, as needed. All seven EWG members agreed to this recommendation.

Contraceptive Services — Tiered Approach to Counseling

Recommendation: For clients who might want to get pregnant in the future and prefer reversible methods of contraception, providers should use a tiered approach to presenting a broad range of contraceptive methods (including long-acting reversible contraception such as intrauterine devices and contraceptive implants), in which the most effective methods are presented before less effective methods.

Quality of evidence: National surveys have demonstrated low rates of LARC use overall (*38,39*). However, Project CHOICE has demonstrated high uptake of long-acting reversible contraception (approximately two thirds of clients when financial barriers are removed) and a very substantial reduction in rates of unintended pregnancy (*40*). Further, a recent study of postpartum contraceptive use shows that 50% of teen mothers with a recent live birth are using long-acting reversible contraception postpartum in Colorado, which demonstrates high levels of acceptance in the context of a statewide program to remove financial barriers (*41*).

Potential consequences: Use of long-acting reversible contraception has the potential to help many more persons prevent unintended pregnancy because of its ease of use, safety, and effectiveness. Several questions were raised about ethical issues in using a tiered approach to counseling. First, is it ethical to educate about long-acting reversible contraception when the methods are not all available on-site? Second, conversely, is it ethical not to inform clients about the most effective methods? In other health service areas, the standard of care is to inform the client about the most effective treatment (e.g., blood pressure medications), so the client can make a fully informed decision, and this standard should apply in this instance as well. On the basis of historic experiences, there is a need to ensure that methods always are offered on a completely voluntary and noncoercive basis. Health-care reform might make contraceptive services more available to the majority of clients.

Rationale: Providers have an obligation to inform clients about the most effective methods available, even if they cannot provide them. Further, health-care reform will reduce the financial barriers to long-acting reversible contraception for many persons. The potential increase in use of long-acting reversible contraception and other more effective methods is likely to help reduce rates of unintended pregnancy. All seven EWG members agreed to this recommendation.

Contraceptive Services — Broad Range of Methods

Recommendation: A broad range of methods should be available on-site or through referral.

Quality of evidence: Three descriptive studies from the review of quality improvement literature identified contraceptive choice as an important aspect of quality care (*42–44*).

Potential consequences: Clients will be more likely to select a method that they will use consistently and correctly.

Rationale: A central tenet of quality health care is that it be client-centered. Being able to provide a client with a method that best fits her or his unique circumstances is essential for that reason. All seven EWG members agreed to this recommendation.

Contraceptive Services — Education

Recommendation: The content, format, method, and medium for delivering education should be evidence-based.

Quality of evidence: Seventeen studies were identified that met the inclusion criteria for this systematic review. Of these, 15 studies looked at knowledge of correct method use or contraceptive risks and benefits, including side effects and method effectiveness (*45–59*). All but one study (*56*) found a statistically significant positive impact of educational interventions on increased knowledge. These studies included six randomized controlled trials with low risk for bias.

Potential consequences: Clients will make more informed decisions when choosing a contraceptive method. More clients will be satisfied with the process of selecting a contraceptive method.

Rationale: Knowledge obtained through educational activities, as integrated into the larger counseling model, is a critically important precondition for the client's ability to make informed decisions. The techniques described in the recommendations have a well-established evidence base for increasing knowledge and satisfaction with services. This knowledge lays the foundation for further counseling steps that will increase the likelihood of correct and consistent use, and increased satisfaction will increase return visits to the service site, as needed. Four of seven EWG members agreed to this recommendation; three members did not express an opinion.

Contraceptive Services — Confirm Understanding

Recommendation: A check box or written statement should be available in the medical record that can be used to document that the client expressed understanding of the most important information about her/his chosen contraceptive method. The teach-back method may be used to get clients to express the most important points by repeating back messages about risks and benefits and appropriate method use and follow-up. Documentation of understanding using the teach-back method and a check box or written statement can be used in place of a written method-specific informed consent.

Quality of evidence: Two studies from outside the family planning literature (one cohort study and one controlled trial with unclear randomization) (*60,61*) and a strong recommendation by members of the Technical Panel on Counseling and Education were considered.

Potential consequences: More clients will make informed decisions, adherence to contraceptive and treatment plans will improve, and reproductive and other health conditions will be better controlled.

Rationale: Asking providers to document in the record that the client is making an informed decision will increase providers' attention to this task. This recommendation will replace a previous requirement that providers obtain method-specific informed consent from each client (in addition to a general consent form). Six of seven EWG members agreed to this recommendation.

Adolescent Services — Comprehensive Information

Recommendation: Providers should provide comprehensive information to adolescent clients about how to prevent pregnancy and STDs. This should include information about contraception and that avoiding sex (abstinence) is an effective way to prevent pregnancy and STDs.

Quality of evidence: A systematic review was not conducted because other recent reviews were available that have shown a substantial impact of comprehensive sexual health education on reduced adolescent risk behavior (*62–66*). The evidence for abstinence-only education was more limited: CDC's Community Guide concluded that there was insufficient evidence (*67*), but the Department of Health and Human Services' Office of Adolescent Health has identified two abstinence-based programs as having evidence of effectiveness (*68*).

Potential consequences: Teens will make more informed decisions and will delay initiation of sexual intercourse. The absence of harmful effects from comprehensive sexual health education was noted.

Rationale: The benefits of informing adolescents about all ways to prevent pregnancy are substantial. Ultimately, each adolescent should make an informed decision that meets her or his unique circumstances, based on the counseling provided by the provider. Six of seven EWG members agreed to this recommendation.

Adolescent Services — Use of Long-Acting Reversible Contraception

Recommendation: Education about contraceptive methods should include an explanation that long-acting reversible contraception is safe and effective for nulliparous women (women who have not been pregnant or given birth), including adolescents.

Quality of evidence: CDC guidelines on contraceptive use (*37*) provide evidence that long-acting reversible contraception is safe and effective for adolescents and nulliparous women.

Potential consequences: More providers will encourage adolescents to consider long-acting reversible contraception; more adolescents will choose long-acting reversible contraception, resulting in reduced rates of teen pregnancy, including rapid repeat pregnancy.

Rationale: Long-acting reversible contraception is safe for adolescents (*37*). As noted above, providers should inform clients about the most effective methods available. The potential increase in use of long-acting reversible contraception and other more effective methods by adolescents is substantial and is likely to lead to further reductions in teen pregnancy. Three EWG members agreed to this recommendation; two EWG members abstained.

Adolescent Services — Confidential Services

Recommendation: Confidential family planning services should be made available to adolescents, while observing state laws and any legal obligations for reporting.

Quality of evidence: Six descriptive studies documented one or more of the following: that confidentiality is important to adolescents; that many adolescents reported they will not use reproductive health services if confidentiality cannot be assured; and that adolescents might not be honest in discussing reproductive health with providers if confidentiality cannot be assured (*69–74*). One RCT showed a slight reduction in use of services after receiving conditional confidentiality, compared with complete confidentiality (*75*). One study showed a

positive association between confidentiality and intention to use services (*73*).

Potential consequences: Consequences might include an increased intention to use services, increased use of services, and reduced rates of teen pregnancy. However, explaining the need to report under certain circumstances (rape, child abuse) might deter some adolescent clients from using services. Further, some parents/guardians might not agree that adolescents should have access to confidential services.

Rationale: Minors' rights to confidential reproductive health services are consistent with state and federal law. The risks of not providing confidential services to adolescents are great and likely to result in an increased rate of teen pregnancies. Finally, this recommendation is consistent with the recommendations of three professional medical associations that endorse provision of confidential services to adolescents (*76–78*). All seven EWG members agreed to this recommendation.

Adolescent Services — Family-Child Communication

Recommendation: Providers should encourage and promote family-child communication about sexual and reproductive health.

Quality of evidence: From the family planning literature, 16 parental involvement programs (most using an RCT study design) were found to be positively associated with at least one short-term (13 of 16 studies) or medium-term (four of seven studies) outcome (*79–94*). However, only one of these studies was linked to clinical services (*80*); others were implemented in community settings.

Potential consequences: Consequences might include increased parental/guardian involvement and communication, improved knowledge/awareness, increased intentions to use contraceptives, and the adoption of more pro-social norms that support parent-child communication about sexual health.

Rationale: The literature provides strong evidence that increased communication between a child and her/his parent/guardian will lead to safer sexual behavior among teens, and numerous community-based programs have created an evidence base for how to strengthen parents/guardians' ability to hold those conversations. Although less is known about how to do so in a clinical setting, providers can refer their clients to programs in the community, and principles from the community-based approaches can be used to help providers develop appropriate approaches in the clinical setting. Research in this area will be monitored, and the recommendations will be revised, as needed. Four of five EWG members who provided input agreed to this recommendation; one member abstained.

Adolescent Services — Repeat Teen Pregnancy

Recommendation: Providers should refer pregnant and parenting adolescents to home visiting and other programs that have been shown to provide needed support and reduce rates of repeat teen pregnancy.

Quality of evidence: Three of four studies of clinic-based programs (using retrospective case-control cohort, ecological evaluation, and prospective cohort study designs) showed that comprehensive teen pregnancy prevention programs (programs with clinical, school, case management, and community components) were associated with both medium- and long-term outcomes (*95–98*). In addition, several randomized trials of community-based home visiting programs, and an existing systematic review of the home visiting literature, demonstrated a protective impact of these programs on preventing repeat teen pregnancy and other relevant outcomes (*99–103*).

Potential consequences: Consequences might include decreased rapid repeat pregnancy and abortion rates, and increased use of contraceptives.

Rationale: There is sufficient evidence to recommend that providers link pregnant and parenting teens to community and social services that might reduce rates of rapid repeat pregnancy. Three of seven EWG members agreed to an earlier version of this recommendation. Other members wanted to remove a clause about prioritizing the contraceptive needs of pregnant/parenting teens because they felt that all clients should be treated as priority clients. This suggestion was adopted, but the EWG did not have a chance to vote again on the modified recommendation.

Contraceptive Method Availability

Recommendation: Family planning programs should stock and offer a broad a range of FDA-approved contraceptive methods so that the needs of individual clients can be met. These methods are optimally available on-site, but strong referrals can serve to make methods not available on-site real options for clients.

Quality of evidence: No research was identified that explicitly addressed the question of whether having a broad range of methods was associated with short-, medium-, or long-term reproductive health outcomes. However, as noted above, three descriptive studies from the review of quality improvement literature identified contraceptive choice as an important aspect of quality care (*42–44*).

Potential consequences: Consequences might include increased use of contraception and increased use of reproductive

health services. It also was noted that there are sometimes high costs to stocking certain methods (e.g., intrauterine devices and contraceptive implants).

Rationale: Having a broad range of contraceptive methods is central to client-centered care, a core aspect of providing quality services. Individual clients need to have a choice so they can select a method that best fits their particular circumstances. This is likely to result in more correct and consistent use of the chosen methods. The benefits of this recommendation were weighed more heavily than the negative outcomes (e.g., additional cost). All five EWG members agreed to this recommendation.

Youth-Friendly Services

Recommendation: Family planning programs should take steps to make services "youth-friendly."

Quality of evidence: Of 20 studies that were identified, six looked at short-, medium-, or long-term outcomes with mixed designs (one group time series, one cross-sectional, three prospective cohort, and one nonrandomized trial); protective effects were found on long-term (two of three studies), medium-term (three of three), and short-term (three of three) outcomes (*29*,*30*,*104–107*). One of these six studies (*29*), plus 13 other descriptive studies (for a total of 14 studies), presented adolescents' or providers' views on facilitators for adolescent clients in using youth-friendly family planning services. Key factors described were confidentiality (13 of 14), accessibility (11 of 14), peer involvement (three of 14), parental or familial involvement (four of 14), and quality of provider interaction (11 of 14) (*105–121*). Four of these studies (*111*,*112*,*114*,*121*) plus one other descriptive study (*108*) described barriers to clinics adopting and implementing youth-friendly family planning services.

Potential consequences: Consequences might include increased use of reproductive health services by adolescents, improved contraceptive use, use of more effective methods, more consistent use of contraception, and reduced rates of teen pregnancy. It is also likely to lead to improved satisfaction with services and greater knowledge about pregnancy prevention among adolescents. It is possible that there will be higher costs, and some uncertainty regarding the benefits due to a relatively weak evidence base.

Rationale: Existing evidence has demonstrated the importance of specific characteristics to adolescents' attitudes and use of clinical services. The potential benefits of providing youth-friendly services outweigh the potential costs and weak evidence base. All five EWG members agreed to this recommendation. Some thought that it should be cast as an example of comprehensively client-centered care, rather than an end of its own.

Quality Improvement

Recommendation: Family planning programs should have a system for quality improvement, which is designed to review and strengthen the quality of services on an ongoing basis. Family planning programs should select, measure, and assess at least one outcome measure on an ongoing basis, for which the service site can be accountable.

Quality of evidence: A recent systematic review (*122*) was supplemented with 10 articles that provided information related to client and/or provider perspectives regarding what constitutes quality family planning services (*42–44*,*113*,*123–128*). These studies used a qualitative (k = 4) or cross-sectional (k = 6) study design. Ten descriptive studies identified client and provider perspectives on what constitutes quality family planning services, which include stigma and embarrassment reduction (n = 9), client access and convenience (n = 8); confidentiality (n = 3); efficiency and tailoring of services (n = 6); client autonomy and confidence (n = 5); contraceptive access and choice (n = 4); increased time of patient-provider interaction (n = 3); communication and relationship (n = 3); structure and facilities (n = 2); continuity of care (n = 2). Well-established frameworks for guiding quality improvement efforts were referenced (*122*,*129–132*).

Potential consequences: Consequences might include increased use by clients of more effective contraceptive methods, clients might be more likely to return for care, client satisfaction might improve, and there might be reduced rates of teen and unintended pregnancy, and improved spacing of births.

Rationale: Research, albeit limited, has demonstrated that quality services are associated with improved client experience with care and adoption of more protective contraceptive behavior. Further, these recommendations on quality improvement are consistent with those made by national leaders in the quality improvement field. Research is either under way or planned to validate a core set of performance measures, and the recommendations will be updated as new findings emerge. All five EWG members agreed to these recommendations.

References

1. Family Planning Services and Population Research Act of 1970, Title X. Population Research and Voluntary Family Planning Programs Project Grants and Contracts for Family Planning Services Pub. L. No. Public Law 91-572 (1970).
2. Project Grants for Family Planning Services. 42 CFR Part 59, Subpart A, (1971).
3. CDC. Recommendations to improve preconception health and health care—United States. MMWR 2006;55(No. RR-06).

4. CDC. Sexually transmitted diseases treatment guidelines, 2010. MMWR 2010;59(No. RR-12).
5. CDC. Revised recommendations for HIV testing of adults, adolescents and pregnant women in health care settings. MMWR 2006;55(No. RR-14).
6. Jack B, Atrash H, Coonrod D, Moos M, O'Donnell J, Johnson K. The clinical content of preconception care: an overview and preparation of this supplement. Am J Obstet Gynecol 2008;199(Supplement B):S266–79.
7. Frey K, Navarro S, Kotelchuck M, Lu M. The clinical content of preconception care: preconception care for men. Am J Obstet Gynecol 2008;199(6 Suppl 2):S389–95.
8. Adams-Skinner J, Exner T, Pili C, Wallace B, Hoffman S, Leu CS. The development and validation of a tool to assess nurse performance in dual protection counseling. Patient Educ Couns 2009;76:265–71.
9. Bender SS, Geirsson RT. Effectiveness of preabortion counseling on postabortion contraceptive use. Contraception 2004;69:481–7.
10. Boise R, Petersen R, Curtis KM, et al. Reproductive health counseling at pregnancy testing: a pilot study. Contraception 2003;68:377–83.
11. Custo G, Saitto C, Cerza S, Sertoli G. The adjusted contraceptive score (ACS) improves the overall performance of behavioural and barrier contraceptive methods. Adv Contracept Deliv Syst 1987;3:367–73.
12. Gilliam M, Knight S, McCarthy M Jr. Success with oral contraceptives: a pilot study. Contraception 2004;69:413–8.
13. Langston AM, Rosario L, Westhoff CL. Structured contraceptive counseling—a randomized controlled trial. Patient Educ Couns 2010;81:362–7.
14. Lee JK, Parisi SM, Akers AY, Borrero S, Schwarz EB. The impact of contraceptive counseling in primary care on contraceptive use. J Gen Intern Med 2011;26:731–6.
15. Namerow PB, Weatherby N, Williams-Kaye J. The effectiveness of contingency-planning counseling. Fam Plann Perspect 1989;21:115–9.
16. Nobili MP, Piergrossi S, Brusati V, Moja EA. The effect of patient-centered contraceptive counseling in women who undergo a voluntary termination of pregnancy. Patient Educ Couns 2007;65:361–8.
17. Petersen R, Albright J, Garrett JM, Curtis KM. Pregnancy and STD prevention counseling using an adaptation of motivational interviewing: a randomized controlled trial. Perspect Sex Reprod Health 2007;39:21–8.
18. Proctor A, Jenkins TR, Loeb T, Elliot M, Ryan A. Patient satisfaction with 3 methods of postpartum contraceptive counseling: a randomized, prospective trial. J Reprod Med 2006;51:377–82.
19. Schunmann C, Glasier A. Specialist contraceptive counselling and provision after termination of pregnancy improves uptake of long-acting methods but does not prevent repeat abortion: a randomized trial. Hum Reprod 2006;21:2296–303.
20. Shlay JC, Mayhugh B, Foster M, Maravi ME, Baron AE, Douglas JM Jr. Initiating contraception in sexually transmitted disease clinic setting: a randomized trial. Am J Obstet Gynecol 2003;189:473–81.
21. Todres R. Effectiveness of counseling in the transmission of family planning and sexuality knowledge. J Sex Educ Ther 1990;16:279–85.
22. Weisman CS, Maccannon DS, Henderson JT, Shortridge E, Orso CL. Contraceptive counseling in managed care: preventing unintended pregnancy in adults. Womens Health Issues 2002;12:79–95.
23. Yassin AS, Cordwell D. Does dedicated pre-abortion contraception counselling help to improve post-abortion contraception uptake? J Fam Plann Reprod Health Care 2005;31:115–6.
24. Berger DK, Perez G, Kyman W, et al. Influence of family planning counseling in an adolescent clinic on sexual activity and contraceptive use. J Adolesc Health Care 1987;8:436–40.
25. Brindis CD, Geierstanger SP, Wilcox N, McCarter V, Hubbard A. Evaluation of a peer provider reproductive health service model for adolescents. Perspect Sex Reprod Health 2005;37:85–91.
26. Cowley CB, Farley T, Beamis K. 'Well, maybe I'll try the pill for just a few months…': Brief motivational and narrative-based interventions to encourage contraceptive use among adolescents at high risk for early childbearing. Fam Syst Health 2002;20:183–204.
27. Hanna KM. Effect of nurse-client transaction on female adolescents' oral contraceptive adherence. Image J Nurs Sch 1993;25:285–90.
28. Kirby D, Raine T, Thrush G, Yuen C, Sokoloff A, Potter SC. Impact of an intervention to improve contraceptive use through follow-up phone calls to female adolescent clinic patients. Perspect Sex Reprod Health 2010;42:251–7.
29. Winter L, Breckenmaker LC. Tailoring family planning services to the special needs of adolescents. Fam Plann Perspect 1991;23:24–30.
30. Brindis CD, Geierstanger SP, Wilcox N, McCarter V, Hubbard A. Evaluation of a peer provider reproductive health service model for adolescents. Perspect Sex Reprod Health 2005;37:85–91.
31. Fox MC, Creinin MD, Murthy AS, Harwood B, Reid LM. Feasibility study of the use of a daily electronic mail reminder to improve oral contraceptive compliance. Contraception 2003;68:365–71.
32. Hou MY, Hurwitz S, Kavanagh E, Fortin J, Goldberg AB. Using daily text-message reminders to improve adherence with oral contraceptives: a randomized controlled trial. Obstet Gynecol 2010;116:633–40.
33. Keder LM, Rulin MC, Gruss J. Compliance with depot medroxyprogesterone acetate: a randomized, controlled trial of intensive reminders. Am J Obstet Gynecol 1998;179:583–5.
34. Lachowsky M, Levy-Toledano R. Improving compliance in oral contraception: 'the reminder card'. Eur J Contracept Reprod Health Care 2002;7:210–5.
35. Madlon-Kay DJ. The effectiveness of a mail reminder system for depot medroxyprogesterone injections. Arch Fam Med 1996;5:234–6.
36. CDC. U.S. selected practice recommendations for contraceptive use 2013. MMWR 2013;62(No. RR-4).
37. CDC. U.S. medical eligibility criteria for contraceptive use 2010. MMWR 2010;59(No. RR-4).
38. Daniels K, Mosher W, Jones J. Contraceptive methods women have ever used: United States, 1982–2010. National health statistics reports; 2013;62.
39. Martinez G, Copen C, Abma J. Teenagers in the United States: sexual activity, contraceptive use, and childbearing, 2006–2010 National Survey of Family Growth. Vital Health Stat 2011;23(31).
40. Winner B, Peipert J, Zhao Q, et al. Effectiveness of long-acting reversible contraception. N Engl J Med 2012;366:1998–2007.
41. CDC. Vital signs: repeat births among teens—United States, 2007–2010. MMWR 2013;62:249–55.
42. Becker D, Klassen AC, Koenig MA, LaVeist TA, Sonenstein FL, Tsui AO. Women's perspectives on family planning service quality: an exploration of differences by race, ethnicity and language. Perspect Sex Reprod Health 2009;41:158–65.
43. Gilliam ML, Hernandez M. Providing contraceptive care to low-income, African American teens: the experience of urban community health centers. J Community Health 2007;32:231–44.
44. Landry DJ, Wei J, Frost JJ. Public and private providers' involvement in improving their patients' contraceptive use. Contraception 2008;78:42–51.
45. Deijen JB, Kornaat H. The influence of type of information, somatization, and locus of control on attitude, knowledge, and compliance with respect to the triphasic oral contraceptive Tri-Minulet. Contraception 1997;56:31–41.

46. Kirby D, Harvey PD, Claussenius D, Novar M. A direct mailing to teenage males about condom use: its impact on knowledge, attitudes and sexual behavior. Fam Plann Perspect 1989;21:12–8.
47. Little P, Griffin S, Kelly J, Dickson N, Sadler C. Effect of educational leaflets and questions on knowledge of contraception in women taking the combined contraceptive pill: randomised controlled trial. BMJ 1998;316:1948–52.
48. Smith LF, Whitfield MJ. Women's knowledge of taking oral contraceptive pills correctly and of emergency contraception: effect of providing information leaflets in general practice. Br J Gen Pract 1995;45:409–14.
49. Steiner MJ, Dalebout S, Condon S, Dominik R, Trussell J. Understanding risk: a randomized controlled trial of communicating contraceptive effectiveness. Obstet Gynecol 2003;102:709–17.
50. DeLamater J, Wagstaff DA, Havens KK. The impact of a culturally appropriate STD/AIDS education intervention on black male adolescents' sexual and condom use behavior. Health Educ Behav 2000;27:454–70.
51. Mason V, McEwan A, Walker D, Barrett S, James D. The use of video information in obtaining consent for female sterilisation: a randomised study. BJOG 2003;110:1062–71.
52. Schwarz EB, Gerbert B, Gonzales R. Computer-assisted provision of emergency contraception a randomized controlled trial. J Gen Intern Med 2008;23:794–9.
53. Paperny DM, Starn JR. Adolescent pregnancy prevention by health education computer games: computer-assisted instruction of knowledge and attitudes. Pediatrics 1989;83:742–52.
54. Reis J, Tymchyshyn P. A longitudinal evaluation of computer-assisted instruction on contraception for college students. Adolescence 1992;27:803–11.
55. Roberto AJ, Zimmerman RS, Carlyle KE, Abner EL, Cupp PK, Hansen GL. The effects of a computer-based pregnancy, STD, and HIV prevention intervention: a nine-school trial. Health Commun 2007;21:115–24.
56. Lindenberg CS, Solorzano RM, Bear D, Strickland O, Galvis C, Pittman K. Reducing substance use and risky sexual behavior among young, low-income, Mexican-American women: comparison of two interventions. Appl Nurs Res 2002;15:137–48.
57. Chewning B, Mosena P, Wilson D, et al. Evaluation of a computerized contraceptive decision aid for adolescent patients. Patient Educ Couns 1999;38:227–39.
58. O'Donnell L, San Doval A, Duran R, O'Donnell CR. The effectiveness of video-based interventions in promoting condom acquisition among STD clinic patients. Sex Transm Dis 1995;22:97–103.
59. Pedrazzini AE, McGowan H, Lucking L, Johanson RJ. 'The trouble with sex–it always gets in the way': an evaluation of a peer-produced teenage pregnancy video. Br J Fam Plann 2000;26:131–5.
60. McMahon SR, Rimsza ME, Bay RC. Parents can dose liquid medication accurately. Pediatrics 1997;100:330–3.
61. Schillinger D, Piette J, Grumbach K, et al. Closing the loop: physician communication with diabetic patients who have low health literacy. Arch Intern Med 2003;163:83–90.
62. Kirby D. Comprehensive sex education: strong public support and persuasive evidence of impact, but little funding. Arch Pediatr Adolesc Med 2006;160:1182–4.
63. Guide to Community Preventive Services. Preventing HIV/AIDS, other STIs, and teen pregnancy: group-based comprehensive risk reduction interventions for adolescents. Atlanta, GA: US Department of Health and Human Services, CDC; 2009. Available at http://www.thecommunityguide.org/hiv/riskreduction.html.
64. Kirby D, Obasi A, Laris BA. The effectiveness of sex education and HIV education interventions in schools in developing countries. World Health Organ Tech Rep Ser 2006;938:103–50, discussion 317–41.
65. Kirby DB, Laris BA, Rolleri LA. Sex and HIV education programs: their impact on sexual behaviors of young people throughout the world. J Adolesc Health 2007;40:206–17.
66. Oringanje C, Meremikwu MM, Eko H, Esu E, Meremikwu A, Ehiri JE. Interventions for preventing unintended pregnancies among adolescents. Cochrane Database Syst Rev 2009;(4):CD005215.
67. Guide to Community Preventive Services. Preventing HIV/AIDS, other STIs, and teen pregnancy: group-based abstinence education interventions for adolescents. Atlanta, GA: US Department of Health and Human Services, CDC; 2009; Available at http://www.thecommunityguide.org/hiv/abstinence_ed.html.
68. Office of Adolescent Health. Information about effective programs for preventing teen pregnancy. Washington, DC: US Department of Health and Human Services; 2011. Available at www.hhs.gov/ash/oah/oah-initiatives/tpp/programs.html.
69. Ford CA, Thomsen SL, Compton B. Adolescents' interpretations of conditional confidentiality assurances. J Adolesc Health 2001;29:156–9.
70. Garside R, Ayres R, Owen M, Pearson VA, Roizen J. Anonymity and confidentiality: rural teenagers' concerns when accessing sexual health services. J Fam Plann Reprod Health Care 2002;28:23–6.
71. Rainey DY, Brandon DP, Krowchuk DP. Confidential billing accounts for adolescents in private practice. J Adolesc Health 2000;26:389–91.
72. Schuster MA, Bell RM, Petersen LP, Kanouse DE. Communication between adolescents and physicians about sexual behavior and risk prevention. Arch Pediatr Adolesc Med 1996;150:906–13.
73. Thomas N, Murray E, Rogstad KE. Confidentiality is essential if young people are to access sexual health services. Int J STD AIDS 2006;17:525–9.
74. Thrall JS, McCloskey L, Ettner SL, Rothman E, Tighe JE, Emans SJ. Confidentiality and adolescents' use of providers for health information and for pelvic examinations. Arch Pediatr Adolesc Med 2000;154:885–92.
75. Ford CAMS, Halpern-Felsher BL, Irwin CE Jr. Influence of physician confidentiality assurances on adolescents' willingness to disclose information and seek future health care. A randomized controlled trial. JAMA 1997;278:1029–34.
76. American College of Obstetricians and Gynecologists. Confidentiality in adolescent health care: guidelines for adolescent health care. 2nd ed. Washington, DC: American College of Obstetricians and Gynecologists; 2011.
77. American Academy of Pediatrics. Achieving quality health services for adolescents: policy statement of the Committee on Adolescence, American Academy of Pediatrics. Pediatrics 2008;121:1263.
78. Ford C, English A, Sigman G. Confidential health care for adolescents: position paper for the society for adolescent medicine. J Adolesc Health 2004;35:160–7.
79. Anderson NLR, Koniak-Griffin D, Keenan CK, Uman G, Duggal BR, Casey C. Evaluating the outcomes of parent-child family life education [including commentary by Hayman LL]. Sch Inq Nurs Pract 1999;13:211–38.
80. Guilamo-Ramos V, Bouris A, Jaccard J, Gonzalez B, McCoy W, Aranda D. A parent-based intervention to reduce sexual risk behavior in early adolescence: building alliances between physicians, social workers, and parents. J Adolesc Health;48:159–63.
81. Blake SM, Simkin L, Ledsky R, Perkins C, Calabrese JM. Effects of a parent-child communications intervention on young adolescents' risk for early onset of sexual intercourse. Fam Plann Perspect 2001;33:52–61.

82. Brody GH, Murry VM, Gerrard M, et al. The Strong African American Families Program: translating research into prevention programming. Child Dev 2004;75:900–17.
83. Evans WD, Davis KC, Ashley OS, Blitstein J, Koo H, Zhang Y. Efficacy of abstinence promotion media messages: findings from an online randomized trial. J Adolesc Health 2009;45:409–16.
84. Forehand R, Armistead L, Long N, et al. Efficacy of a parent-based sexual-risk prevention program for African American preadolescents: a randomized controlled trial. Arch Pediatr Adolesc Med 2007;161:1123–9.
85. Haggerty KP, Skinner ML, MacKenzie EP, Catalano RF. A randomized trial of Parents Who Care: effects on key outcomes at 24-month follow-up. Prev Sci 2007;8:249–60.
86. Huston RL, Martin LJ, Foulds DM. Effect of a program to facilitate parent-child communication about sex. Clin Pediatr (Phila) 1990;29:626–33.
87. Lederman RP, Chan W, Roberts-Gray C. Sexual risk attitudes and intentions of youth aged 12–14 years: survey comparisons of parent-teen prevention and control groups. Behavioral Medicine (Washington, DC) 2004;29:155–63.
88. Lederman RP, Chan W, Roberts-Gray C. Parent-adolescent relationship education (PARE): program delivery to reduce risks for adolescent pregnancy and STDs. Behav Med 2008;33:137–43.
89. Lefkowitz ES, Sigman M, Au TK. Helping mothers discuss sexuality and AIDS with adolescents. Child Dev 2000;71:1383–94.
90. Miller BC, Norton MC, Jenson GO. Impact Evaluation of Facts and Feelings: a home-based video sex education curriculum. Fam Relat 1993;42:392–400.
91. Murry VM, Berkel C, Brody GH, Gerrard M, Gibbons FX. The Strong African American Families program: longitudinal pathways to sexual risk reduction. J Adolesc Health 2007;41:333–42.
92. Schuster MA, Corona R, Elliott MN, et al. Evaluation of Talking Parents, Healthy Teens, a new worksite based parenting programme to promote parent-adolescent communication about sexual health: randomised controlled trial. BMJ 2008;337:a308.
93. Stanton B, Cole M, Galbraith J, et al. Randomized trial of a parent intervention: parents can make a difference in long-term adolescent risk behaviors, perceptions, and knowledge. Arch Pediatr Adolesc Med 2004;158:947–55.
94. Stanton BF, Li X, Galbraith J, et al. Parental underestimates of adolescent risk behavior: a randomized, controlled trial of a parental monitoring intervention. J Adolesc Health 2000;26:18–26.
95. Elster AB, Lamb ME, Tavare J, Ralston CW. The medical and psychosocial impact of comprehensive care on adolescent pregnancy and parenthood. JAMA 1987;258:1187–92.
96. Omar HA, Fowler A, McClanahan KK. Significant reduction of repeat teen pregnancy in a comprehensive young parent program. J Pediatr Adolesc Gynecol 2008;21:283–7.
97. Rabin JM, Seltzer V, Pollack S. The long term benefits of a comprehensive teenage pregnancy program. Clin Pediatr (Phila) 1991;30:305–9.
98. Stevens-Simon C, Kelly L, Kulick R. A village would be nice but…it takes a long-acting contraceptive to prevent repeat adolescent pregnancies. Am J Prev Med 2001;21:60–5.
99. Eckenrode J, Campa M, Luckey DW, et al. Long-term effects of prenatal and infancy nurse home visitation on the life course of youths: 19-year follow-up of a randomized trial. Arch Pediatr Adolesc Med 2010;164:9–15.
100. Olds D, Henderson CR Jr, Cole R, et al. Long-term effects of nurse home visitation on children's criminal and antisocial behavior: 15-year follow-up of a randomized controlled trial. JAMA 1998;280:1238–44.
101. Olds DL, Eckenrode J, Henderson CR Jr, et al. Long-term effects of home visitation on maternal life course and child abuse and neglect. Fifteen-year follow-up of a randomized trial. JAMA 1997;278:637–43.
102. Black M, Bentley M, Papas M, et al. Delaying second births among adolescent mothers: a randomized, controlled trial of a home-based mentoring program. Pediatrics 2006;118:1087–99.
103. Avellar S, Paulsell D, Sama-Miller E, Del Grosso P. Home visiting evidence of effectiveness review. Washington, DC: US Department of Health and Human Services, Office of Planning, Research and Evaluation, Administration for Children and Families; 2012.
104. Wilson S, Daniel S, Pearson J, Hopton C, Madeley R. An evaluation of a new teenage clinic and its impact on teenage conceptions in Nottingham from 1986 to 1992. Contraception 1994;50:77–86.
105. Gupta S, Hogan R, Kirkman RJ. Experience of the first pelvic examination. Eur J Contracept Reprod Health Care 2001;6:34–8.
106. Morrison A, Mackie CM, Elliott L, Elliott LM, Gruer L, Bigrigg A. The Sexual Health Help Centre: a service for young people. J Public Health Med 1997;19:457–63.
107. Herz EJ, Olson LM, Reis JS. Family planning for teens: strategies for improving outreach and service delivery in public health settings. Public Health Rep 1988;103:422–30.
108. Bethea J, Hippisley-Cox J, Coupland C, Pringle M. General practitioners' attitudes towards the provision of services to young people aged under 16: a cross-sectional survey. Qual Prim Care 2007;15:11–9.
109. Kapphahn CJ, Wilson KM, Klein JD. Adolescent girls' and boys' preferences for provider gender and confidentiality in their health care. J Adolesc Health 1999;25:131–42.
110. Wilson A, Williams R. Sexual health services: what do teenagers want? Ambul Child Health 2000;6:253–60.
111. Alberti PM, Steinberg AB, Hadi EK, Abdullah RB, Bedell JF. Barriers at the frontline: assessing and improving the teen friendliness of South Bronx medical practices. Public Health Rep 2010;125:611–4.
112. Brindis CD, Loo VS, Adler NE, Bolan GA, Wasserheit JN. Service integration and teen friendliness in practice: a program assessment of sexual and reproductive health services for adolescents. J Adolesc Health 2005;37:155–62.
113. Chambers R, Boath E, Chambers S. Young people's and professionals' views about ways to reduce teenage pregnancy rates: to agree or not agree. J Fam Plann Reprod Health Care 2002;28:85–90.
114. Cromer BA, McCarthy M. Family planning services in adolescent pregnancy prevention: the views of key informants in four countries. Fam Plann Perspect 1999;31:287–93.
115. Donovan C, Mellanby AR, Jacobson LD, Taylor B, Tripp JH; The Adolescent Working Group. Teenagers' views on the general practice consultation and provision of contraception. Br J Gen Pract 1997;47:715–8.
116. French RS. The experience of young people with contraceptive consultations and health care workers. Int J Adolesc Med Health 2002;14:131–8.
117. Hayter M. Reaching marginalized young people through sexual health nursing outreach clinics: evaluating service use and the views of service users. Public Health Nurs 2005;22:339–46.
118. Ingram J, Salmon D. 'No worries!' Young people's experiences of nurse-led drop-in sexual health services in South West England. J Res Nurs 2007;12:305–16.

119. Peremans L, Hermann I, Avonts D, Van Royen P, Denekens J. Contraceptive knowledge and expectations by adolescents: an explanation by focus groups. Patient Educ Couns 2000;40:133–41.
120. Perry C, Thurston M. Meeting the sexual health care needs of young people: a model that works? Child Care Health Dev 2008;34:98–103.
121. Russell ST, Lee FCH. Practitioners' perspectives on effective practices for Hispanic teenage pregnancy prevention. Perspect Sex Reprod Health 2004;36:142–9.
122. Becker D, Koenig MA, Kim YM, Cardona K, Sonenstein FL. The quality of family planning services in the United States: findings from a literature review. Perspect Sex Reprod Health 2007;39:206–15.
123. Becker D, Tsui AO. Reproductive health service preferences and perceptions of quality among low-income women: racial, ethnic and language group differences. Perspect Sex Reprod Health 2008;40:202–11.
124. Becker H, Stuifbergen A, Tinkle M. Reproductive health care experiences of women with physical disabilities: a qualitative study. Arch Phys Med Rehabil 1997;78:S26–33.
125. Bender SS. Attitudes of Icelandic young people toward sexual and reproductive health services. Fam Plann Perspect 1999;31:294–301.
126. Dixon-Woods M, Stokes T, Young B, Phelps K, Windridge K, Shukla R. Choosing and using services for sexual health: a qualitative study of women's views. Sex Transm Infect 2001;77:335–9.
127. Khan NS, Kirkman R. Intimate examinations: use of chaperones in community-based family planning clinics. BJOG 2000;107:130–2.
128. Fiddes P, Scott A, Fletcher J, Glasier A. Attitudes towards pelvic examination and chaperones: a questionnaire survey of patients and providers. Contraception 2003;67:313–7.
129. Bruce J. Fundamental elements of the quality of care: a simple framework. Stud Fam Plann 1990;21:61–91.
130. Donabedian A. The quality of care. JAMA 1988;260:1743–8.
131. Institute of Medicine. Crossing the quality chasm: a new health system for the 21st century. Washington, DC: National Academies of Science; 2001.
132. National Quality Forum. ABCs of measurement. Washington, DC: National Quality Forum; 2011. Available at http://www.qualityforum.org/Measuring_Performance/ABCs_of_Measurement.aspx.

Appendix C

Principles for Providing Quality Counseling

Counseling is a process that enables clients to make and follow through on decisions. Education is an integral component of the counseling process that helps clients to make informed decisions. Providing quality counseling is an essential component of client-centered care.

Key principles of providing quality counseling are listed below and may be used when providing family planning services. The model was developed in consultation with the Technical Panel on Contraceptive Counseling and Education and reviewed by the Expert Work Group. Although developed specifically for providing contraceptive counseling, the principles are broad and can be applied to health counseling on other topics. Although the principles are listed here in a particular sequence, counseling is an iterative process, and at every point in the client encounter it is necessary to determine whether it is important to readdress and emphasize a given principle.

Principles of Quality Counseling

Principle 1. Establish and Maintain Rapport with the Client

Establishing and maintaining rapport with a client is vital to the encounter and achieving positive outcomes (*1*). This can begin by creating a welcoming environment and should continue through every stage of the client encounter, including follow-up. The contraceptive counseling literature indicates that counseling models that emphasized the quality of the interaction between client and provider have been associated with decreased teen pregnancy, increased contraceptive use, increased use of more effective methods, increased use of repeat or follow-up services, increased knowledge, and enhanced psychosocial determinants of contraceptive use (*2–5*) .

Principle 2. Assess the Client's Needs and Personalize Discussions Accordingly

Each visit should be tailored to the client's individual circumstances and needs. Clients come to family planning providers for various services and with varying needs. Standardized questions and assessment tools can help providers determine what services are most appropriate for a given visit (*6*). Contraceptive counseling studies that have incorporated standardized assessment tools during the counseling process have resulted in increased contraceptive use, increased correct use of contraceptives, and increased use of more effective methods (*2,7,8*). Contraceptive counseling studies that have personalized discussions to meet the individual needs of clients have been associated with increased contraceptive use, increased correct use of contraceptives, increased use of more effective methods, increased use of dual-method contraceptives to prevent both sexually transmitted diseases (STDs) and pregnancy, increased quality and satisfaction with services, increased knowledge, and enhanced psychosocial determinants of contraceptive use *(4,7,9–12)*.

Principle 3. Work with the Client Interactively to Establish a Plan

Working with a client interactively to establish a plan, including a plan for follow-up, is important. Establishing a plan should include setting goals, discussing possible difficulties with achieving goals, and developing action plans to deal with potential difficulties. The amount of time spent establishing a plan will differ depending on the client's purpose for the visit and health-care needs. A client plan that requires behavioral change should be made on the basis of the client's own goals, interests, and readiness for change (*13–15*). Use of computerized decision aids before the appointment can facilitate this process by providing a structured yet interactive framework for clients to analyze their available options systematically and to consider the personal importance of perceived advantages and disadvantages *(16,17)*. The contraceptive counseling literature indicates that counseling models that incorporated goal setting and development of action plans have been associated with increased contraceptive use, increased correct use of contraceptives, increased use of more effective methods, and increased knowledge *(2,9,18–20)*. Furthermore, contraceptive counseling models that incorporated follow-up contacts resulted in decreased teen pregnancy, increased contraceptive use, increased correct use of contraceptives, increased use of more effective methods, increased continuation of method use, increased use of dual-method contraceptives to prevent both STDs and pregnancy, increased use of repeat or follow-up services, increased knowledge, and enhanced psychosocial determinants of contraceptive use *(2,3,7,11,21,22)* . From the family planning education literature, computerized decision aids have helped clients formulate questions and have been associated with increased knowledge, selection of more effective methods, and increased continuation and compliance (*23–25*).

Principle 4. Provide Information That Can Be Understood and Retained by the Client

Clients need information that is medically accurate, balanced, and nonjudgmental to make informed decisions and follow through on developed plans. When speaking with clients or providing educational materials through any medium (e.g., written, audio/visual, or computer/web-based), the provider must present information in a manner that can be readily understood and retained by the client. Strategies for making information accessible to clients are provided (see Appendix D).

Principle 5. Confirm Client Understanding

It is important to ensure that clients have processed the information provided and discussed. One technique for confirming understanding is to have the client restate the most important messages in her or his own words. This teach-back method can increase the likelihood of the client and provider reaching a shared understanding, and has improved compliance with treatment plans and health outcomes *(26,27)*. Using the teach-back method early in the decision-making process will help ensure that a client has the opportunity to understand her or his options and is making informed choices (*28*).

References

1. Lambert M. Implications of outcome research for psychotherapy integration. In: Norcross J, Goldfind M, eds. Handbook of psychotherapy integration. New York, NY: Basic Books; 1992:94–129.
2. Adams-Skinner J, Exner T, Pili C, Wallace B, Hoffman S, Leu CS. The development and validation of a tool to assess nurse performance in dual protection counseling. Patient Educ Couns 2009;76:265–71.
3. Brindis CD, Geierstanger SP, Wilcox N, McCarter V, Hubbard A. Evaluation of a peer provider reproductive health service model for adolescents. Perspect Sex Reprod Health 2005;37:85–91.
4. Nobili MP, Piergrossi S, Brusati V, Moja EA. The effect of patient-centered contraceptive counseling in women who undergo a voluntary termination of pregnancy. Patient Educ Couns 2007;65:361–8.
5. Proctor A, Jenkins TR, Loeb T, Elliot M, Ryan A. Patient satisfaction with 3 methods of postpartum contraceptive counseling: a randomized, prospective trial. J Reprod Med 2006;51:377–82.
6. Fiore M, Jaén C, Baker T, Bailey W, Benowitz N, Curry S. Treating tobacco use and dependence: 2008 update. Clinical practice guideline. Rockville, MD: US Department of Health and Human Services; 2008. Available at http://www.ncbi.nlm.nih.gov/books/NBK63952.
7. Boise R, Petersen R, Curtis KM, et al. Reproductive health counseling at pregnancy testing: a pilot study. Contraception 2003;68:377–83.
8. Custo G, Saitto C, Cerza S, Sertoli G. The adjusted contraceptive score (ACS) improves the overall performance of behavioural and barrier contraceptive methods. Adv Contracept Deliv Syst 1987;3:367–73.
9. Hanna KM. Effect of nurse-client transaction on female adolescents' oral contraceptive adherence. Image J Nurs Sch 1993;25:285–90.
10. Schunmann C, Glasier A. Specialist contraceptive counselling and provision after termination of pregnancy improves uptake of long-acting methods but does not prevent repeat abortion: a randomized trial. Hum Reprod 2006;21:2296–303.
11. Shlay JC, Mayhugh B, Foster M, Maravi ME, Baron AE, Douglas JM Jr. Initiating contraception in sexually transmitted disease clinic setting: a randomized trial. Am J Obstet Gynecol 2003;189:473–81.
12. Weisman CS, Maccannon DS, Henderson JT, Shortridge E, Orso CL. Contraceptive counseling in managed care: preventing unintended pregnancy in adults. Womens Health Issues 2002;12:79–95.
13. Kaplan D. Family Counseling for all counselors. Greensboro, NC: CAPS Publications; 2003.
14. Nupponen R. What is counseling all about—basics in the counseling of health-related physical activity. Patient Educ Couns 1998; 33(Suppl):S61–7.
15. Whitlock EP, Orleans CT, Pender N, Allan J. Evaluating primary care behavioral counseling interventions: an evidence-based approach. Am J Prev Med 2002;22:267–84.
16. French RS, Wellings K, Cowan F. How can we help people to choose a method of contraception? The case for contraceptive decision aids. J Fam Plann Reprod Health Care 2009;35:219–20.
17. O'Connor AM, Bennett CL, Stacey D, et al. Decision aids for people facing health treatment or screening decisions. Cochrane Database Syst Rev 2009;CD001431.
18. Cowley CB, Farley T, Beamis K. "Well, maybe I'll try the pill for just a few months...": brief motivational and narrative-based interventions to encourage contraceptive use among adolescents at high risk for early childbearing. Fam Syst Health 2002;20:183–204.
19. Gilliam M, Knight S, McCarthy M Jr. Success with oral contraceptives: a pilot study. Contraception 2004;69:413–8.
20. Namerow PB, Weatherby N, Williams-Kaye J. The effectiveness of contingency-planning counseling. Fam Plann Perspect 1989;21:115–9.
21. Berger DK, Perez G, Kyman W, et al. Influence of family planning counseling in an adolescent clinic on sexual activity and contraceptive use. J Adolesc Health Care 1987;8:436–40.
22. Winter L, Breckenmaker LC. Tailoring family planning services to the special needs of adolescents. Fam Plann Perspect 1991;23:24–30.
23. Chewning B, Mosena P, Wilson D, et al. Evaluation of a computerized contraceptive decision aid for adolescent patients. Patient Educ Couns 1999;38:227–39.
24. Garbers S, Meserve A, Kottke M, Hatcher R, Chiasson MA. Tailored health messaging improves contraceptive continuation and adherence: results from a randomized controlled trial. Contraception 2012;86:536–42.
25. Garbers S, Meserve A, Kottke M, Hatcher R, Ventura A, Chiasson MA. Randomized controlled trial of a computer-based module to improve contraceptive method choice. Contraception 2012;86:383–90.
26. McMahon SR, Rimsza ME, Bay RC. Parents can dose liquid medication accurately. Pediatrics 1997;100:330–3.
27. Schillinger D, Piette J, Grumbach K, et al. Closing the loop: physician communication with diabetic patients who have low health literacy. Arch Intern Med 2003;163:83–90.
28. National Quality Forum. Health literacy: a linchpin in achieving national goals for health and healthcare, Issue Brief #13 2009. Available at http://www.qualityforum.org/Publications/2009/03/Health_Literacy__A_Linchpin_in_Achieving_National_Goals_for_Health_and_Healthcare.aspx.

Appendix D

Contraceptive Effectiveness

Providers should counsel clients about the effectiveness of different contraceptive methods. Method effectiveness is measured as the percentage of women experiencing an unintended pregnancy during the first year of use, and is estimated for both typical and perfect use (Table).

TABLE. Percentage of women experiencing an unintended pregnancy during the first year of typical use* and the first year of perfect use† of contraception and the percentage continuing use at the end of the first year — United States

Method	% of women experiencing an unintended pregnancy within the first year of use		% of women continuing use at 1 year§
	Typical use	Perfect use	
No method¶	85.0	85.0	
Spermicides**	28.0	18.0	42.0
Fertility awareness-based methods	24.0		47.0
Standard days method††		5.0	
2-day method††		4.0	
Ovulation method††		3.0	
Symptothermal method		0.4	
Withdrawal	22.0	4.0	46.0
Sponge			36.0
Parous women	24.0	20.0	
Nulliparous women	12.0	9.0	
Condom§§			
Female	21.0	5.0	41.0
Male	18.0	2.0	43.0
Diaphragm¶¶	12.0	6.0	57.0
Combined pill and progestin-only pill	9.0	0.3	67.0
Evra patch	9.0	0.3	67.0
NuvaRing	9.0	0.3	67.0
Depo-Provera	6.0	0.2	56.0
Intrauterine contraceptives			
ParaGard (copper T)	0.8	0.6	78.0
Mirena (LNG)	0.2	0.2	80.0
Implanon	0.05	0.05	84.0
Female sterilization	0.5	0.5	100.0
Male sterilization	0.15	0.1	100.0

Emergency Contraceptives: Emergency contraceptive pills or insertion of a copper intrauterine contraceptive after unprotected intercourse substantially reduces the risk of pregnancy.***
Lactational Amenorrhea Method: LAM is a highly effective, temporary method of contraception.†††
Source: Adapted from Trussell J. Contraceptive efficacy. In: Hatcher RA, Trussell J, Nelson AL, Cates W, Kowal D, Policar M, eds. Contraceptive technology: 20th revised ed. New York, NY: Ardent Media; 2011.

* Among typical couples who initiate use of a method (not necessarily for the first time), the percentage of couples who experience an accidental pregnancy during the first year if they do not stop use for any other reason. Estimates of the probability of pregnancy during the first year of typical use for spermicides and the diaphragm are taken from the 1995 National Survey of Family Growth corrected for underreporting of abortion; estimates for fertility awareness-based methods, withdrawal, the male condom, the pill, and Depo-Provera are taken from the 1995 and 2002 National Survey of Family Growth corrected for underreporting of abortion. See the text for the derivation of estimates for the other methods.

† Among couples who initiate use of a method (not necessarily for the first time) and who use it perfectly (both consistently and correctly), the percentage of couples who experience an accidental pregnancy during the first year if they do not stop use for any other reason. See the text for the derivation of the estimate for each method.

§ Among couples attempting to avoid pregnancy, the percentage of couples who continue to use a method for 1 year.

¶ The percentages becoming pregnant in columns labeled "typical use" and "perfect use" are based on data from populations in which contraception is not used and from women who cease using contraception to become pregnant. Among such populations, approximately 89% become pregnant within 1 year. This estimate was lowered slightly (to 85%) to represent the percentage of women who would become pregnant within 1 year among women now relying on reversible methods of contraception if they abandoned contraception altogether.

** Foams, creams, gels, vaginal suppositories, and vaginal film.

†† The Ovulation and 2-day methods are based on evaluation of cervical mucus. The Standard Days method avoids intercourse on cycle days 8 through 19. The Symptothermal method is a double-check method based on evaluation of cervical mucus to determine the first fertile day and evaluation of cervical mucus and temperature to determine the last fertile day.

§§ Without spermicides.

¶¶ With spermicidal cream or jelly.

*** Ella, Plan B One-Step, and Next Choice are the only dedicated products specifically marketed for emergency contraception. The label for Plan B One-Step (1 dose is 1 white pill) says to take the pill within 72 hours after unprotected intercourse. Research has indicated that all of the brands listed here are effective when used within 120 hours after unprotected intercourse. The label for Next Choice (1 dose is 1 peach pill) says to take one pill within 72 hours after unprotected intercourse and another pill 12 hours later. Research has indicated that that both pills can be taken at the same time with no decrease in efficacy or increase in side effects and that they are effective when used within 120 hours after unprotected intercourse. The Food and Drug Administration has in addition declared the following 19 brands of oral contraceptives to be safe and effective for emergency contraception: Ogestrel (1 dose is 2 white pills), Nordette (1 dose is 4 light-orange pills), Cryselle, Levora, Low-Ogestrel, Lo/Ovral, or Quasence (1 dose is 4 white pills), Jolessa, Portia, Seasonale or Trivora (1 dose is 4 pink pills), Seasonique (1 dose is 4 light-blue-green pills), Enpresse (1 dose is 4 orange pills), Lessina (1 dose is 5 pink pills), Aviane or LoSeasonique (one dose is 5 orange pills), Lutera or Sronyx (1 dose is 5 white pills), and Lybrel (1 dose is 6 yellow pills).

††† However, for effective protection against pregnancy to be maintained, another method of contraception must be used as soon as menstruation resumes, the frequency or duration of breastfeeds is reduced, bottle feeds are introduced, or the baby reaches age 6 months.

Appendix E

Strategies for Providing Information to Clients

The client should receive and understand the information she or he needs to make informed decisions and follow treatment plans. This requires careful attention to how information is communicated. The following strategies can make information more readily comprehensible to clients:

Strategies for Providing Information to Clients

Educational materials should be provided that are clear and easy to understand. Educational materials delivered through any one of a variety of media (for example, written, audio/visual, computer/web-based) need to be presented in a format that is clear and easy to interpret by clients with a 4th to 6th grade reading level (*1*–*3*). Many adults have only a basic ability to obtain, process, and understand health information necessary to make decisions about their health (*4*). Making easy-to-access materials enhances informed decision-making (*1*–*3*). Test all educational materials with the intended audiences for clarity and comprehension before wide-scale use.

The following evidence-based tools provide recommendations for increasing the accessibility of materials through careful consideration of content, organization, formatting, and writing style:

- Health Literacy Universal Precautions Toolkit, provided by the Agency for Healthcare Research and Quality (available at http://www.ahrq.gov/qual/literacy),
- Toolkit for Making Written Material Clear and Effective, provided by the Centers for Medicare and Medicaid Services (available at http://www.cms.gov/WrittenMaterialsToolkit), and
- Health Literacy Online, provided by the Office of Disease Prevention and Health Promotion (available at http://www.health.gov/healthliteracyonline).

Information should be delivered in a manner that is culturally and linguistically appropriate. In presenting information it is important to be sensitive to the client's cultural and linguistic preferences (*5*,*6*). Ideally information should be presented in the client's primary language, but translations and interpretation services should be available when necessary. Information presented must also be culturally appropriate, reflecting the client's beliefs, ethnic background, and cultural practices. Tools for addressing cultural and linguistic differences and preferences include

- Health Literacy Universal Precautions Toolkit, provided by the Agency for Healthcare Research and Quality (available at http://www.ahrq.gov/qual/literacy), and
- Toolkit for Making Written Material Clear and Effective, Part 11; Understanding and using the "Toolkit Guidelines for Culturally Appropriate Translation," provided by the Centers for Medicare and Medicaid Services (available at http://www.cms.gov/outreach-and-education/outreach/writtenmaterialstoolkit/downloads/toolkitpart11.pdf).

The amount of information presented should be limited and emphasize essential points. Providers should focus on needs and knowledge gaps identified during the assessment. Many clients immediately forget or remember incorrectly much of the information provided. This problem is exacerbated as more information is presented (*7*–*9*). Limiting the amount of information presented and highlighting important facts by presenting them first improves comprehension (*10*–*14*).

Numeric quantities should be communicated in a way that is easily understood. Whenever possible, providers should use natural frequencies and common denominators (for example, 85 of 100 sexually active women are likely to get pregnant within 1 year using no contraceptive, as compared with 1 in 100 using an IUD or implant), and display quantities in graphs and visuals. Providers also should avoid using verbal descriptors without numeric quantities (for example, sexually active women using an IUD or implant almost never become pregnant). Finally, they should quantify risk in absolute rather than relative terms (for example, "the chance of unintended pregnancy is reduced from 8 in 100 to 1 in 100 by switching from oral contraceptives to an IUD" versus the chance of unintended pregnancy is reduced by 87%). Numeracy is more highly correlated with health outcomes than the ability to read or listen effectively (*15*). The strategies listed above can help clients interpret numeric quantities correctly (*16*–*28*).

Balanced information on risks and benefits should be presented and messages framed positively. In addition to discussing risks, contraindications, and warnings, providers should discuss the advantages and benefits of contraception. In presenting this information, providers should express risks and benefits in a common format (for example, do not present risks in relative terms and benefits in absolute terms), and frame messages in positive terms (for example "99 out of 100 women find this a safe method with no side effects," versus "1 out of 100 women experience noticeable side effects"). Many clients prefer to receive a balance of information on risks and benefits (*29*), and using a common format avoids bias in presentation of information (*18*,*22*,*26*,*30*). Framing messages positively increases acceptance and comprehension (*18*,*22*,*31*,*32*).

Active client engagement should be encouraged. Providers should use educational materials that encourage active information processing (e.g., questions, quizzes, fill-in-the-blank, web-based games, and activities). In addition, they should be sure the client has an opportunity to discuss the information provided, and when speaking with a client, providers should engage her or him actively. Research has indicated that interactive materials improve knowledge of contraceptive risks, benefits, and correct method use (*33*–*35*). Clients also value spoken information (*29*,*36*); and educational materials, when delivered by a provider, more effectively increase knowledge (*10*,*37*). In particular, presenting information in a question and answer format is more effective than simply presenting the information (*10*,*15*,*37*–*41*).

References

1. Centers for Medicare and Medicaid Services. Toolkit for making written material clear and effective. Baltimore, MD: Centers for Medicare and Medicaid Services; 2011.
2. US Department of Health and Human Services, Office of Disease Prevention and Health Promotion. Health literacy online: A guide to writing and designing easy-to-use health Web sites. Washington, DC: US Department of Health and Human Services; 2010.
3. DeWalt D, Callahan L, Hawk V, et al. Health literacy universal precautions toolkit. AHRQ Publication No. 10–0046-EF. Rockville, MD: Agency for Healthcare Research and Quality; 2010.
4. Kutner M, Greenberg E, Jin Y, Paulsen C. The health literacy of America's adults: results from the 2003 National Assessment of Adult Literacy (NCES 2006–483). Washington, DC: U.S. Department of Education: National Center for Education Statistics; 2006.
5. Olavarria M, Beaulac J, Belanger A, Young M, Aubry T. Organizational cultural competence in community health and social service organizations: how to conduct a self-assessment. J Cult Divers 2009;16:140–50.
6. Tucker C. Reducing health disparities by promoting patient-centered culturally and linguistically sensitive/competent health care. U.S. Department of Health and Human Services Advisory Committee on Minority Health, US Public Health Service; 2009.
7. Anderson JL, Dodman S, Kopelman M, Fleming A. Patient information recall in a rheumatology clinic. Rheumatol Rehabil 1979;18:18–22.
8. Crane JA. Patient comprehension of doctor-patient communication on discharge from the emergency department. J Emerg Med 1997;15:1–7.
9. McGuire LC. Remembering what the doctor said: organization and adults' memory for medical information. Exp Aging Res 1996;22:403–28.
10. Little P, Griffin S, Kelly J, Dickson N, Sadler C. Effect of educational leaflets and questions on knowledge of contraception in women taking the combined contraceptive pill: randomised controlled trial. BMJ 1998;316:1948–52.
11. McGee J. Toolkit for making written material clear and effective: Baltimore, MD: Centers for Medicare and Medicaid Services; 2010. Available at http://www.cms.gov/WrittenMaterialsToolkit.
12. Peters E, Dieckmann N, Dixon A, Hibbard JH, Mertz CK. Less is more in presenting quality information to consumers. Med Care Res Rev 2007;64:169–90.
13. Steiner MJ, Dalebout S, Condon S, Dominik R, Trussell J. Understanding risk: a randomized controlled trial of communicating contraceptive effectiveness. Obstet Gynecol 2003;102:709–17.
14. Berry DC, Michas IC. Rosis F. Evaluating explanations about drug prescriptions: Effects of varying the nature of information about side effects and its relative position in explanations. Psychol Health 1998;13:767–84.
15. Berkman N, Sheridan S, Donahue K, et al. Health literacy interventions and outcomes: an updated systematic review. Evidence Report/ Technology Assesment No. 199. Rockville, MD: Agency for Healthcare Research and Quality; 2011.
16. Berry DC. Informing people about the risks and benefits of medicines: implications for the safe and effective use of medicinal products. Curr Drug Saf 2006;1:121–6.
17. Berry DC, Raynor DK, Knapp P, Bersellini E. Patients' understanding of risk associated with medication use: impact of European Commission guidelines and other risk scales. Drug Saf 2003;26:1–11.
18. Edwards A, Elwyn G, Mulley A. Explaining risks: turning numerical data into meaningful pictures. BMJ 2002;324:827–30.
19. Galesic M, Gigerenzer G, Straubinger N. Natural frequencies help older adults and people with low numeracy to evaluate medical screening tests. Med Decis Making 2009;29:368–71.
20. Garcia-Retamero R, Galesic M. Communicating treatment risk reduction to people with low numeracy skills: a cross-cultural comparison. Am J Public Health 2009;99:2196–202.
21. Garcia-Retamero R, Galesic M, Gigerenzer G. Do icon arrays help reduce denominator neglect? Med Decis Making 2010;30:672–84.
22. Gigerenzer G, Edwards A. Simple tools for understanding risks: from innumeracy to insight. BMJ 2003;327:741–4.
23. Knapp P, Gardner PH, Raynor DK, Woolf E, McMillan B. Perceived risk of tamoxifen side effects: a study of the use of absolute frequencies or frequency bands, with or without verbal descriptors. Patient Educ Couns 2010;79:267–71.
24. Kurz-Milcke E, Gigerenzer G, Martignon L. Transparency in risk communication: graphical and analog tools. Ann N Y Acad Sci 2008;1128:18–28.
25. Lipkus IM. Numeric, verbal, and visual formats of conveying health risks: suggested best practices and future recommendations. Med Decis Making 2007;27:696–713.
26. Paling J. Strategies to help patients understand risks. BMJ 2003; 327:745–8.
27. Skolbekken JA. Communicating the risk reduction achieved by cholesterol reducing drugs. BMJ 1998;316:1956–8.
28. Visschers VH, Meertens RM, Passchier WW, de Vries NN. Probability information in risk communication: a review of the research literature. Risk Anal 2009;29:267–87.
29. Raynor DK, Blenkinsopp A, Knapp P, et al. A systematic review of quantitative and qualitative research on the role and effectiveness of written information available to patients about individual medicines. Health Technol Assess 2007;11:iii, 1–160.
30. Fahey T, Griffiths S, Peters TJ. Evidence based purchasing: understanding results of clinical trials and systematic reviews. BMJ 1995;311:1056–9, discussion 9–60.
31. Armstrong K, Schwartz JS, Fitzgerald G, Putt M, Ubel PA. Effect of framing as gain versus loss on understanding and hypothetical treatment choices: survival and mortality curves. Med Decis Making 2002;22:76–83.
32. Gurm HS, Litaker DG. Framing procedural risks to patients: is 99% safe the same as a risk of 1 in 100? Acad Med 2000;75:840–2.
33. Paperny DM, Starn JR. Adolescent pregnancy prevention by health education computer games: computer-assisted instruction of knowledge and attitudes. Pediatrics 1989;83:742–52.
34. Reis J, Tymchyshyn P. A longitudinal evaluation of computer-assisted instruction on contraception for college students. Adolescence 1992;27:803–11.
35. Roberto AJ, Zimmerman RS, Carlyle KE, Abner EL, Cupp PK, Hansen GL. The effects of a computer-based pregnancy, STD, and HIV prevention intervention: a nine-school trial. Health Commun 2007;21:115–24.
36. Grime J, Blenkinsopp A, Raynor DK, Pollock K, Knapp P. The role and value of written information for patients about individual medicines: a systematic review. Health Expect 2007;10:286–98.

37. DeLamater J, Wagstaff DA, Havens KK. The impact of a culturally appropriate STD/AIDS education intervention on black male adolescents' sexual and condom use behavior. Health Educ Behav 2000;27:454–70.
38. McMahon SR, Rimsza ME, Bay RC. Parents can dose liquid medication accurately. Pediatrics 1997;100:330–3.
39. Belcher L, Kalichman S, Topping M, et al. A randomized trial of a brief HIV risk reduction counseling intervention for women. J Consult Clin Psychol 1998;66:856–61.
40. Eldridge GD, St Lawrence JS, Little CE, et al. Evaluation of the HIV risk reduction intervention for women entering inpatient substance abuse treatment. AIDS Educ Prev 1997;9(Suppl):62–76.
41. Jaccard J. Unlocking the contraceptive conundrum. Washington, DC: The National Campaign to Prevent Teen and Unplanned Pregnancy; 2009. Available at http://thenationalcampaign.org/resource/unlocking-contraception-conundrum.

Appendix F

Screening Services For Which Evidence Does Not Support Screening

The following services have been given a D recommendation from the U.S. Preventive Services Task Force (USPSTF), which indicates that the potential harms of routine screening outweigh the benefits. Providers should not perform these screening services.

The USPSTF has recommended against offering the following services to women and men:

- **Asymptomatic bacteriuria:** USPSTF recommends against screening for asymptomatic bacteriuria in men and nonpregnant women (*1*).
- **Gonorrhea:** USPSTF recommends against routine screening for gonorrhea infection in men and women who are at low risk of infection (*2*).
- **Hepatitis B:** USPSTF recommends against routinely screening the general asymptomatic population for chronic hepatitis B virus infection (*3*).
- **Herpes simplex virus (HSV):** USPSTF recommends against routine serological screening for HSV in asymptomatic adolescents and adults (*4*).
- **Syphilis:** USPSTF recommends against screening of asymptomatic persons who are not at increased risk of syphilis infection (*5*).

The USPSTF has recommended against offering the following services to women:

- **BRCA mutation testing for breast and ovarian cancer susceptibility:** USPSTF recommends against routine referral for genetic counseling or routine breast cancer susceptibility gene (BRCA) testing for women whose family history is not associated with an increased risk of deleterious mutations in breast cancer susceptibility gene 1 (BRCA1) or breast cancer susceptibility gene 2 (BRCA2) (*6*). However, USPSTF continues to recommend that women whose family history is associated with an increased risk of deleterious mutations in BRCA1 or BRCA2 genes be referred for genetic counseling and evaluation for BRCA testing.
- **Breast self-examination:** USPSTF recommends against teaching breast self-examination (*7*).
- **Cervical cytology:** USPSTF recommends against routine screening for cervical cancer with cytology (Pap smear) in the following groups: women aged <21 years, women aged >65 years who have had adequate prior screening and are not otherwise at high risk for cervical cancer, women who have had a hysterectomy with removal of the cervix and who do not have a history of a high-grade precancerous lesion (i.e., cervical intraepithelial neoplasia grade 2 or 3) or cervical cancer. USPSTF recommends against screening for cervical cancer with HPV testing, alone or in combination with cytology, in women aged <30 years (*8*).
- **Ovarian cancer:** USPSTF recommends against routine screening for ovarian cancer (*9*).

The USPSTF has recommended against offering the following services to men:

- **Prostate cancer:** USPSTF recommends against prostate-specific antigen (PSA)-based screening for prostate cancer (*10*).
- **Testicular cancer:** USPSTF recommends against screening for testicular cancer in adolescent or adult males (*11*).

References

1. US Preventive Services Task Force. Screening for asymptomatic bacteriuria in adults. Rockville, MD: US Department of Health and Human Services, Agency for Healthcare Research and Quality; 2008. Available at http://www.uspreventiveservicestaskforce.org/uspstf/uspsbact.htm.
2. US Preventive Services Task Force. Screening for gonorrhea. Rockville, MD: US Department of Health and Human Services, Agency for Healthcare Research and Quality; 2005. Available at http://www.uspreventiveservicestaskforce.org/uspstf/uspsgono.htm.
3. US Preventive Services Task Force. Screening for hepatitis B infection. Rockville, MD: US Department of Health and Human Services, Agency for Healthcare Research and Quality; 2004. Available at http://www.uspreventiveservicestaskforce.org/uspstf/uspshepb.htm.
4. US Preventive Services Task Force. Screening for genital herpes: recommendation statement. Rockville, MD: US Department of Health and Human Services, Agency for Healthcare Research and Quality; 2005. Available at http://www.uspreventiveservicestaskforce.org/uspstf05/herpes/herpesrs.htm.
5. US Preventive Services Task Force. Screening for syphilis infection. Rockville, MD: US Department of Health and Human Services, Agency for Healthcare Research and Quality; 2004. Available at http://www.uspreventiveservicestaskforce.org/uspstf/uspssyph.htm.
6. US Preventive Services Task Force. Risk assessment, genetic counseling, and genetic testing for BRCA-related cancer in women. Rockville, MD: US Department of Health and Human Services, Agency for Healthcare Research and Quality; 2013. Available at http://www.uspreventiveservicestaskforce.org/uspstf/uspsbrgen.htm.
7. US Preventive Services Task Force. Screening for breast cancer. Rockville, MD: US Department of Health and Human Services, Agency for Healthcare Research and Quality; 2009. Available at http://www.uspreventiveservicestaskforce.org/uspstf/uspsbrca.htm.
8. US Preventive Services Task Force. Screening for cervical cancer. Rockville, MD: US Department of Health and Human Services, Agency for Healthcare Research and Quality; 2012. Available at www.uspreventiveservicestaskforce.org/uspstf11/cervcancer/cervcancerrs.htm.
9. US Preventive Services Task Force. Screening for ovarian cancer: U.S. Preventive Services Task Force reaffirmation recommendation statement. Rockville, MD: US Department of Health and Human Services, Agency for Healthcare Research and Quality; 2012. Available at http://www.uspreventiveservicestaskforce.org/uspstf12/ovarian/ovarcancerrs.htm.
10. US Preventive Services Task Force. Screening for prostate cancer. Rockville, MD: US Department of Health and Human Services, Agency for Healthcare Research and Quality; 2012. Available at http://www.uspreventiveservicestaskforce.org/prostatecancerscreening.htm.
11. US Preventive Services Task Force. Screening for testicular cancer. Rockville, MD: US Department of Health and Human Services, Agency for Healthcare Research and Quality; 2011. Available at http://www.uspreventiveservicestaskforce.org/uspstf/uspstest.htm.

Lead Authors

Loretta Gavin, PhD, Division of Reproductive Health, CDC
Susan Moskosky, MS, Office of Population Affairs, CDC

Systematic Review Authors and Presenters

Anna Brittain, MHS, Division of Reproductive Health, CDC
Marion Carter, PhD, Division of Reproductive Health, CDC
Kathryn Curtis, PhD, Division of Reproductive Health, CDC
Emily Godfrey, MD, Division of Reproductive Health, CDC
Arik V. Marcell, MD, The Johns Hopkins University and the Male Training Center
Cassondra Marshall, MPH, Division of Reproductive Health, CDC
Karen Pazol, PhD, Division of Reproductive Health, CDC
Naomi Tepper, MD, Division of Reproductive Health, CDC
Marie Tiller, PhD, MANILA Consulting Group, Inc.
Stephen Tregear, DPhil, MANILA Consulting Group, Inc.
Michelle Tregear, PhD, MANILA Consulting Group, Inc.
Jessica Williams, MPH, MANILA Consulting Group, Inc.
Lauren Zapata, PhD, Division of Reproductive Health, CDC

Expert Work Group

Courtney Benedict, MSN, Marin Community Clinics
Jan Chapin, MPH, American College of Obstetricians and Gynecologists
Clare Coleman, President and CEO, National Family Planning and Reproductive Health Association
Vanessa Cullins, MD, Planned Parenthood Federation of America
Daryn Eikner, MS, Family Planning Council
Jule Hallerdin, MN, Advisor to the Office of Population Affairs
Mark Hathaway, MD, Unity Health Care and Washington Hospital Center
Seiji Hayashi, MD, Bureau of Primary Health Care, Health Resources and Services Administration
Beth Jordan, MD, Association of Reproductive Health Professionals
Ann Loeffler, MSPH, John Snow Research and Training Institute
Arik V. Marcell, MD, The Johns Hopkins University and the Male Training Center
Tom Miller, MD, Alabama Department of Health
Deborah Nucatola, MD, Planned Parenthood Federation of America
Michael Policar, MD, State of California and UCSF Bixby Center
Adrienne Stith-Butler, PhD, Keck Center of the National Academies
Denise Wheeler, ARNP, Iowa Department of Public Health
Gayla Winston, MPH, Indiana Family Health Council
Jacki Witt, MSN, Clinical Training Center for Family Planning, University of Missouri—Kansas City
Jamal Gwathney, MD, Bureau of Primary Health Care, Health Resources and Services Administration

Technical Panel on Women's Clinical Services

Courtney Benedict, MSN, Marin Community Clinics
Janet Chapin, MPH, American College of Obstetricians and Gynecologists
Elizabeth DeSantis, MSN, South Carolina Department of Health and Environmental Control
Linda Dominguez, CNP, Southwest Women's Health
Eileen Dunne, MD, Division of STD Prevention, CDC
Jamal K. Gwathney, MD, Bureau of Primary Health Care, Health Resources and Services Administration
Jule Hallerdin, Consultant Advisor
Mark Hathaway, MD, Washington Hospital Center
Arik V. Marcell, MD, Johns Hopkins University and the Male Training Center
Cheri Moran, University of Illinois Medical Center at Chicago
Deborah Nucatola, MD, Planned Parenthood Federation of America
Michael Policar, MD, Family PACT Program - California State Office of Family Planning
Pablo Rodriguez, MD, Women's Care Inc., Providence Office
Denise Wheeler, ARNP, Iowa Department of Public Health
Jacki Witt, MSN, Clinical Training Center for Family Planning, University of Missouri—Kansas City

Technical on Men's Clinical Services

Linda Creegan, FNP, California STD/HIV Prevention Training Center
Dennis Fortenberry, MD, Indiana University School of Medicine
Emily Godfrey, MD, University of Illinois at Chicago
Wendy Grube, PhD, University of Pennsylvania School of Nursing
Arik V. Marcell, MD, The Johns Hopkins University and the Male Training Center
Elissa Meites, MD, Division of STD Prevention, CDC
Anne Rompalo, MD, Johns Hopkins University
Thomas Walsh, MD, University of Washington Medical Center
Jacki Witt, MSN, Clinical Training Center for Family Planning, University of Missouri—Kansas City
Sandra Wolf, MD, Women's Care Center, Philadelphia

Technical Panel on Adolescents

Claire Brindis, DrPH, University of California, San Francisco
Gale Burstein, MD, SUNY at Buffalo School of Medicine and Biomedical Sciences, Department of Pediatrics
Laura Davis, MA, Advocates for Youth
Patricia J. Dittus, PhD, Division of STD Prevention, CDC
Paula Duncan, MD, University of Vermont College of Medicine
Carol Ford, MD, The Children's Hospital of Philadelphia
Melissa Gilliam, MD, The University of Chicago
Mark Hathaway, MD, Unity Health Care & Washington Hospital Center
Deborah Kaplan, PhD, New York City Department of Health and Mental Hygiene
Arik V. Marcell, MD, The Johns Hopkins University and the Male Training Center
Brent C. Miller, PhD, Utah State University
Elizabeth M. Ozer, PhD, Division of Adolescent Medicine, University of California, San Francisco
John Santelli, MD, Columbia University, Mailman School of Public Health

Technical Panel on Counseling and Education

Beth Barnet, MD, University of Maryland
Betty Chewning, PhD, University of Wisconsin School of Pharmacy
Christine Dehlendorf, MD, University of California, San Francisco
Linda Dominguez, CNP, Southwest Women's Health
Jillian Henderson, PhD, University of California, San Francisco
James Jaccard, PhD, New York University
Beth Jordan, MD, Association of Reproductive Health Professionals—East
David Kaplan, PhD, American Counseling Association
Alicia Luchowski, American Congress of Obstetricians and Gynecologists
Merry-K Moos, FNP, University of North Carolina at Chapel Hill
Patricia Murphy, DrPH, University of Utah College of Nursing
Elizabeth O'Connor, PhD, Kaiser Permanente Center for Health Research
Jeff Peipert, MD, Washington University in St. Louis

Technical Panel on Quality Improvement

Davida Becker, PhD, Bixby Center for Global Reproductive Health University of California, San Francisco
Peter Briss, MD, National Center for Chronic Disease Prevention and Health Promotion, CDC
Denise Dougherty, PhD, Agency for Healthcare Research and Quality
Daryn Eikner, MS, Family Planning Council
Christina I. Fowler, PhD, RTI International
Evelyn Glass, MSPH, Consultant Advisor
Yvonne Hamby, MPH, Regional Quality Improvement and Infertility Prevention Programs
A. Seiji Hayashi, MD, Bureau of Primary Health Care, Health Resources and Services Administration
Michael D. Kogan, PhD, Health Resources and Services Administration /Maternal and Child Health Bureau
Tom Miller, MD, Alabama Department of Health
Sam Posner, PhD, National Center for Chronic Disease Prevention and Health Promotion, CDC
Donna Strobino, PhD, Johns Hopkins University
Amy Tsui, PhD, Johns Hopkins Bloomberg School of Public Health
Reva Winkler, MD, National Quality Forum

Adivsors on Community Outreach and Participation*

Paula Baraitser, MBBS, Kings College Hospital NHS Foundation Trust/Health Protection Agency
Joy Baynes, MPH, Advocates for Youth
Diane Chamberlain, California Family Health Council
Clare Coleman, National Family Planning & Reproductive Health Association
Emily Godfrey, MD, University of North Carolina and Division of Reproductive Health, CDC
Rachel Gold, MPA, Guttmacher Institute
Rachel Kachur, MPH, Division of STD Prevention, CDC
Michelle Kegler, PhD, Rollins School of Public Health, Emory
Eleanor McLellan-Lemal, PhD, Division of HIV/AIDS Prevention, CDC
Paula Parker-Sawyers, National Campaign to Prevent Teen and Unplanned Pregnancy
Denise Wheeler, MS, Iowa Department of Public Health
Gayla Winston, MPH, Indiana Family Health Council, Inc.

CDC and Office of Population Affairs Reviewers

Wanda Barfield, MD, Division of Reproductive Health, CDC
Gail Bolan, MD, Division of STD Prevention, CDC
Linda Dahlberg, PhD, Division of Violence Prevention, CDC
Patricia Dietz, PhD, Division of Reproductive Health, CDC
Sherry Farr, PhD, Division of Reproductive Health, CDC
Evelyn Glass, MSPH, Office of Population Affairs
Tamara Haegerich, PhD, Division of Violence Prevention, CDC
David Johnson, MPH, Office of Population Affairs
Pamela Kania, MS, Office of Population Affairs
Marilyn Keefe, MPH, Deputy Assistant Secretary for Population Affairs
Dmitry Kissin, MD, Division of Reproductive Health, CDC
Nancy Mautone-Smith, MSW, Office of Population Affairs
Jacqueline Miller, MD, Division of Cancer Prevention and Control, CDC
Sam Posner, PhD, National Center for Chronic Disease and Health Promotion, CDC
Cheryl Robbins, PhD, Division of Reproductive Health, CDC
Lance Rodewald, MD, Division of Immunization Services, CDC
Mona Saraiya, MD, Division of Cancer Prevention and Control, CDC
Van Tong, MPH, Division of Reproductive Health, CDC
Lee Warner, PhD, Division of Reproductive Health, CDC
Kim Workowski, MD, Division of STD Prevention, CDC

External Reviewers

Paula Braverman, MD, Department of Pediatrics at the University of Cincinnati
Claire Brindis, DrPH, University of California–San Francisco
Sarah Brown, MPH, National Campaign to Prevent Teen and Unplanned Pregnancy
Marji Gold, MD, Albert Einstein School of Medicine
Milton Kotelchuck, PhD, Massachusetts General Hospital for Children and Harvard Medical School
David Levine, MD, Morehouse School of Medicine
Pamela Murray, MD, West Virginia University School of Medicine

Competing interests for the development of these guidelines were not assessed.

* These persons made important contributions to a discussion about community outreach and participation. A decision was made to narrow the focus of this report to clinical services, so recommendations informed by the input of these persons will be published separately.

The *Morbidity and Mortality Weekly Report (MMWR)* Series is prepared by the Centers for Disease Control and Prevention (CDC) and is available free of charge in electronic format. To receive an electronic copy each week, visit *MMWR*'s free subscription page at *http://www.cdc.gov/mmwr/mmwrsubscribe.html*. Paper copy subscriptions are available through the Superintendent of Documents, U.S. Government Printing Office, Washington, DC 20402; telephone 202-512-1800.

Address all inquiries about the *MMWR* Series, including material to be considered for publication, to Editor, *MMWR* Series, Mailstop E-90, CDC, 1600 Clifton Rd., N.E., Atlanta, GA 30329-4027 or to *mmwrq@cdc.gov.*

ISSN: 1057-5987

Evaluation &
the Health Professions
Volume 30 Number 4
December 2007 303-321

10.1177/0163278707307899
http://ehp.sagepub.com
hosted at
http://online.sagepub.com

Methodologies for Improving Response Rates in Surveys of Physicians

A Systematic Review

Jonathan B. VanGeest
Kennesaw State University, Georgia
Timothy P. Johnson
University of Illinois at Chicago
Verna L. Welch
Morehouse School of Medicine, Atlanta

Although physician surveys are an important tool in health services and policy research, they are often characterized by low response rates. The authors conducted a systematic review of 66 published reports of efforts to improve response rates to physician surveys. Two general strategies were explored in this literature: incentive and design-based approaches. Even small financial incentives were found to be effective in improving physician response. Token nonmonetary incentives were much less effective. In terms of design strategies, postal and telephone strategies have generally been more successful than have fax or Web-based approaches, with evidence also supporting use of mixed-mode surveys in this population. In addition, use of first-class stamps on return envelopes and questionnaires designed to be brief, personalized, and endorsed by legitimizing professional associations were also more likely to be successful. Researchers should continue to implement design strategies that have been documented to improve the survey response of physicians.

Keywords: *physicians; surveys; research methods; response rates*

Physician surveys are an important tool in health services and policy research, providing cost-effective sources of information on physicians' attitudes, knowledge, and practices related to care delivery. Surveys have been used to assess a range of issues, from more routine subjects like knowledge of and/or compliance with evidence-based practice recommendations (Mosca et al., 2005; Schroy et al., 2001; Webster, Courtney, Huang, Matz, & Christiani, 2005) to highly sensitive topics such as substance abuse among

physicians (Hughes et al., 1999; McAuliffe et al., 1986), physician attitudes toward euthanasia (Emanuel et al., 2000; Farber et al., 2006; Meier et al., 1998), and physician manipulation of reimbursement rules for patients (Wynia, Cummins, VanGeest, & Wilson, 2000). Despite their importance, however, physician surveys are characterized by low response rates, raising concerns about the validity and generalizability of their findings (Asch, Connor, Hamilton, & Fox, 2000; Asch, Jedrziewski, & Christakis, 1997; Berk, 1985; Cartwright, 1978; Cull, O'Connor, Sharp, & Tang, 2005; Cummings, Savitz, & Konrad, 2001; Kellerman & Herold, 2001). Specifically, low response rates raise concerns about nonresponse bias or the likelihood that nonresponding physicians will be systematically different from the population under study. This concern is supported by research showing modest differences between responders and nonresponders and between early and late respondents on demographic and/or practice-related characteristics (Cartwright, 1978; Cockburn, Campbell, Gordon, & Sanson-Fisher, 1988; Cull et al., 2005; Goodman & Jensen, 1981; McFarlane, Olmsted, Murphy, & Hill, 2006; Myerson, 1993; Parsons, Warnecke, Czaja, Barnsley, & Kaluzny, 1994; Stocks & Gunnell, 2000; Tambor et al., 1993; Templeton, Deehan, Taylor, Drummond, & Strang, 1997; Thran and Gonzalez, 1999). As a result, researchers have investigated why physicians are less likely to respond to surveys and implemented strategies for improving physician participation.

Why Physicians Do Not Respond

In a seminal article, Seymour Sudman (1985) identified a number of reasons why professionals (e.g., physicians) might refuse to participate in surveys. Arguably the most important reason for nonresponse is lack of time. Physicians are busy and time spent completing a survey is time that could be spent seeing patients or used to attend to other—more important—tasks. A second and related issue involves the perceived salience of the study. Like other professionals, physicians will not take the time to complete a survey if the value of the study is not clear or is clear but perceived to be low. Third, physicians will generally not complete a survey when they have concerns about the confidentiality of the results. Finally, the likelihood of nonresponse is greater in cases where individual questions may appear biased or not allow the respondent a full range of choices on the subject. Lack of time is compounded by the increasing volume and length of surveys physicians are asked to respond to (Kaner, Haighton, & McAvoy, 1998; MacPherson & Bisset, 1995; McAvoy & Kaner, 1996). Researchers have also identified the

private practice office setting (with its various gatekeepers) as an additional barrier to physician participation (Berry & Kanouse, 1987; Heywood, Mudge, Ring, & Sanson-Fisher, 1995; Moore & An, 2001; Parsons et al., 1994).

Strategies to Encourage Physician Participation

Numerous strategies have been devised to increase physician response to surveys (Field et al., 2002; Kellerman & Herold, 2001; Sudman, 1985). These strategies generally fall into two categories: incentive-based interventions (both monetary and nonmonetary) and design-based approaches (e.g., personalized mailings, design-friendly questionnaires, sponsorship, etc.). Previous reviews have found token monetary incentives to be effective at improving physician participation (Field et al., 2002; Kellerman & Herold, 2001). However, questions remain regarding how much of an incentive is most cost-effective (Field et al., 2002; Halpern, Ubel, Berlin, & Asch, 2002; VanGeest, Wynia, Cummins, & Wilson, 2001). Less is know about the efficacy of non-monetary incentives, although a review of the literature suggests mixed results in surveys of physicians (Thran & Berk, 1993). Design-based approaches have also been shown to increase physician cooperation (Cummings et al., 2001). Again, however, there is little consensus on the efficacy of the full range of techniques purported to increase response rates of physicians. In this article, we conducted a systematic review to determine the extent to which incentive- and designed-based strategies have been found to be effective in improving physician response to surveys.

Method

Experimental studies examining methods to improve physician response to mail surveys were identified through keyword searches of the MEDLINE, Scopus, Sociological Abstracts, and PsychINFO databases from 1975 to 2006. Searches by author using the same databases were also conducted for investigators with identified relevant articles. Finally, several seed sources (e.g., *Medical Care*, *Public Opinion Quarterly*, *Evaluation and the Health Professions*) were also referenced manually in an effort to establish a comprehensive set of studies to be included in the analyses. Further relevant articles and books were selected from the reference listings of the primary journal articles. Where appropriate, odds ratios (ORs) were calculated for individual studies as a measure of effect size for the different interventions identified

(Tu, 2003). In addition, weighted overall ORs were calculated for groups of studies analyzing like interventions.

Results

Impact of Incentives on Response Rates Among Physicians

Monetary incentives. A total of 21 articles (1981 to 2006) were identified that examined the effects of token monetary incentives on physician response to surveys. Incentive amounts ranged $1 to $50 and included both cash payments and charitable donations. Incentives also included opportunities to win cash lottery prizes. Selected studies comparing incentives to no-incentive controls are presented in Table 1. Taken as a whole, the weighted overall effect size reflected an association between monetary incentives and physician response (OR 2.13; 95% confidence interval [CI] 1.7–2.6). Unweighted average effect sizes for different incentive levels (minimum of 2 studies per level) are presented in Figure 1. Generally, even modest $1 incentives were associated with higher response rates among physicians (average OR across the relevant studies was 2.11) when compared with physicians receiving no incentive (Berk, Edwards, & Gay, 1993; Deehan, Templeton, Taylor, Drummond, & Strang, 1997; Donaldson et al., 1999, Easton, Price, Telljohann, & Boehm, 1997; Everett, Price, Bedell, & Telljohann, 1997; Gunn & Rhodes, 1981; Kasprzyk, Montano, St. Lawrence, & Phillips, 2001; Leung, Ho, Chan, Johnston, & Wong, 2002; Mizes, Fleece, & Roos, 1984; Moore & An, 2001; Robertson, Walkom, & McGettigan, 2005). The only exception was a small $1 donation to charity (Olson, Schneiderman, & Armstrong, 1993). With regard to larger incentives, results are mixed. As illustrated in Figure 1, there are little differences in serial increments over $1. This is supported by studies (not shown) which tested for and found no or nonsignificant differences between incentive levels (Gunn & Rhodes, 1981; Kasprzyk et al., 2001; Mizes et al., 1984; VanGeest et al., 2001). The only exceptions were studies by Asch, Christakis, & Ubel (1998) and Halpern et al. (2002), although their results may be compromised by the uniqueness of the $2 bill option employed. Comparative studies indicate that cash payments are more effective compared with charity inducements (Deehan et al., 1997), monetary donations to their alma mater (Gattellari & Ward, 2001), nonmonetary incentives (Easton et al., 1997; Tambor et al., 1993), or opportunities to win a cash lottery prize (Leung et al. 2002; Tamayo-Sarver & Baker, 2004). Prepaid monetary incentives are also superior to promised incentives (Berry & Kanouse, 1987;

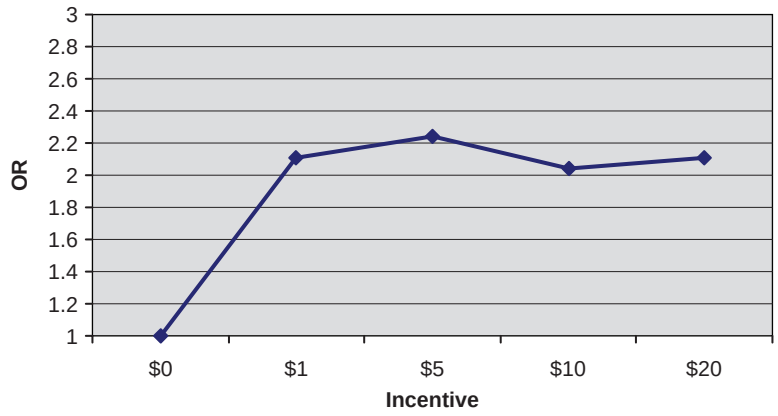
Table 1
Selected Studies Examining Monetary and Nonmonetary Incentives on Physician Response Rates

Monetary Incentives	Intervention	OR	95% CI
Gunn & Rhodes (1981)	$25 vs. no incentive	1.59	0.98–2.59
	$50 vs. no incentive	2.46	1.47–4.12
Mizes et al. (1984)	$1 vs. no incentive	2.66	1.03–6.86
	$5 vs. no incentive	2.66	1.03–6.86
Berry & Kanouse (1987)	Prepayment vs. postpayment	1.83	1.50–2.23
Berk et al. (1993)	$10 vs. no incentive	2.01	1.15–3.50
Everett et al. (1997)	$1 vs. no incentive	2.07	1.46–2.93
Deehan et al. (1997)	£5 vs. no incentive	2.07	1.55–2.76
	£10 vs. no incentive	3.03	2.30–3.99
Easton et al. (1997)	$1 vs. booklet	2.12	1.47–3.04
Donaldson et al. (1999)	$5 vs. no incentive	1.62	1.09–2.41
Moore & An (2001)	$10 vs. no incentive	1.98	1.37–2.87
Kasprzyk et al. (2001)	$15 vs. no incentive	6.38	3.36–12.12
	$25 vs. no incentive	6.06	3.20–11.47
Leung et al. (2002)	HKD$10	1.07	0.52–2.23
	HKD$20	2.09	1.13–3.88
	HKD$40	2.52	1.38–4.58
Delveno et al. (2004)	Prepayment vs. postpayment	1.81	1.42–2.30
Leung et al. (2004)	Prepayment vs. postpayment	1.81	1.32–2.48
Burt & Woodwell (2005)	$50 vs. no incentive	1.00	0.74–1.35
Robertson et al. (2005)	AU$2 lottery	1.48	1.00–2.18
Nonmonetary Incentives	**Intervention**	**OR**	**95% CI**
Sallis et al. (1984)			
2nd mailing	Pencil	2.30	0.81–6.54
3rd mailing	Pencil	0.91	0.41–2.03
Mullen et al. (1987)	Sticker	1.11	0.75–1.66
Bonito et al. (1997)	Risk disk	1.05	0.80–1.36
Ward et al. (1998)	Pen	0.96	0.72–1.28
Baron et al. (2001)	Prize draw	1.29	1.00–1.67
Clark et al. (2001a)	Pen	0.96	0.77–1.19
Halpern et al. (2002)	Candy	0.62	0.49–0.79
Moses & Clark (2004)	Prize draw	1.09	0.88–1.35
Burt & Woodwell (2005)	Candy	0.79	0.58–1.06

Note: OR = odds ratio; CI = confidence interval. Table includes only those studies where sufficient information was available to calculate odds ratio measures of effect size.

Delnevo, Abatemarco, & Steinberg, 2004; Leung et al., 2004). Collectively, when compared to promised incentives, prepaid incentives have a weighted overall effect size reflecting an association with improved physician response (OR 1.82; 95% CI 1.6–2.1).

Figure 1
Average Effect Size (OR) by Monetary Incentive Level



Nonmonetary incentives. Several studies also assessed the effectiveness of token nonmonetary incentives on physician participation, including stickers (Mullen, Easling, Nixon, Koester, & Biddle, 1987), pencils (Sallis, Fortmann, Solomon, & Farquhar, 1984), pens (Clark, Khan, & Gupta, 2001a; Ward, Bruce, Holt, D'Este, & Sladden, 1998), informational brochures (Easton et al., 1997), risk-assessment computer programs (Bonito, Samsa, Akin, & Matchar, 1997), and candy (Burt & Woodwell, 2005; Halpern et al., 2002; see Table 1). The effects of more substantial incentives (e.g., prize draws for a weekend trip or a personal digital assistant [PDA]) were also explored (Baron et al., 2001; Moses & Clark, 2004). Generally, when compared to physicians receiving no incentive, token nonmonetary incentives appear to have little or no impact on response rates (Bonito et al., 1997; Burt & Woodwell, 2005; Clark et al., 2001a; Easton et al., 1997; Halpern et al., 2002; Mullen et al., 1987; Ward et al., 1998). This is supported in the present analyses in which nonmonetary incentives had a weighted overall effect size (OR 0.97; 95% CI 0.82–1.14) reflecting a nonsignificant impact on physician response. There are, however, a couple of exceptions. In one study, the opportunity to receive continuing medical education (CME) credits was deemed to be an effective motivation for physician participation in a mailed questionnaire (McDermott et al., 2003). However, the CME credits were offered in conjunction with a small ($5) monetary incentive, making

it impossible to determine the independent effects of the CME credit on physician participation. The impact of CME credit is also not consistent, with a similar study concluding that CME credit was not as effective as a monetary incentive for inducing physician response (Tambor et al., 1993). In the other exception, the inclusion of a pencil in a second mailing resulted in an increased response (Sallis et al., 1984). However, when the same questionnaire was sent to another sample, inclusion of a pencil in the third mailing had no impact. Studies examining more substantial nonfinancial inducements also had mixed results, with only the opportunity to win a weekend trip for two resulting in a small but significant increase in physician response (OR 1.29; 95% CI 1.00–1.67; Baron et al., 2001). One study also explored the effect of magnitude of the prize draw—one big prize versus many small prizes—on physician response, finding the larger prize to be more effective despite lower odds of winning (Thomson, Paterson-Brown, Russell, McCaldin, & Russell, 2004).

Impact of Design-Based Strategies

Questionnaire design. Nine studies examined the impact of questionnaire design (e.g., length of questionnaire, paper size/quality, questionnaire format) on physician response. Intuitively, length of questionnaire would be of particular interest given that time constraints so prominently figure in physician participation. Only four studies, however, were identified that examined the effect of questionnaire length on physician response. One relatively small study found nonsignificant differences in response rates related to length of the survey (Marin & Howe, 1984). Other studies, however, suggest that shorter questionnaires result in higher cooperation rates (Cartwright & Ward, 1968; Hing, Schappert, Burt, & Shimizu, 2005; Jepson, Asch, Hershey, & Ubel, 2005). A simple average of individual ORs across the latter three studies was 2.33 (Table 2), with the weighted overall effect size reflecting an association between shorter questionnaire length and physician response (OR 2.0; 95% CI 1.1–3.7). Evidence from Jepson et al. (2005) even suggests that under certain conditions, physician participation will be sensitive to relatively small (under 1,000 vs. more than 1,000 word) differences in questionnaire length (OR 2.348; 95% CI 1.20–4.61). Studies have also examined print format, paper size, and paper quality on physician cooperation rates, with mixed results. For example, a recent study comparing single- versus double-sided printing found no differences in physician cooperation rates (Brehaut, Graham, Visentin, & Stiell, 2006). Other studies found the use of an attractive business letter format (Gullen & Garrison, 1973) and standard-sized (8.5 in. × 11 in.)

Table 2
Selected Studies Examining Design-Based Interventions on Physician Cooperation

Questionnaire Design	Intervention	OR	95% CI
Cartwright & Ward (1968)	Short form	3.33	1.70–6.54
Hing et al. (2005)	Short form	1.30	0.89–1.90
Jepson et al.(2005)	Short form	3.35	1.20–4.61
Personalization and Sponsorship			
Maheux et al. (1989)	Handwritten letter	1.90	1.27–2.83
	Personalized mailout	1.52	1.05–2.19
Bostic et al. (1992)	Physician contact	5.92	3.54–9.91
Asch & Christakis (1994)	Sponsorship	1.35	1.03–1.77
Ward & Wain (1994)	Telephone prompt	2.36	1.25–4.44
Heywood et al. (1995)	Physician contact	3.27	1.17–9.15
Osborn et al. (1996)	Advance contact	1.88	1.19–2.98
Temple-Smith et al. (1998)	Advance contact	1.35	0.80–2.27
Ward et al. (1998)	Physician contact	1.46	1.04–2.03
Bhandari et al. (2003)	Sponsorship	0.62	0.41–0.92
McKenzie-McHarg et al. (2005)	Handwritten signature	1.06	0.91–1.24
Leece et al. (2006)	Personalized letter	2.07	1.19–3.60
Type of Mailing			
Gullen & Garrison (1973)	First-class mail	2.02	1.69–2.42
Shiono & Klebanoff (1991)	First-class stamps	1.15	1.06–1.25
Urban et al. (1993)	First-class stamps	2.04	1.22–3.43
Del Valle et al. (1997)	Certified mail	2.09	1.35–3.22
Kasprzyk et al. (2001)	FedEx	1.44	0.90–2.33
Streiff et al. (2001)	First-class stamps	1.30	1.12–1.51

Note: OR = odds ratio; CI = confidence interval. Table includes only those studies where sufficient information was available to calculate odds ratio measures of effect size.

questionnaire booklets (Johnson, Parsons, Warnecke, & Kaluzny, 1993) to be associated with higher response rates. Paper quality, on the other hand, was not associated with increased physician cooperation (Clark, Khan, & Gupta, 2001b). Finally, one study was identified that examined open- versus closed-ended questionnaire formats on physician response (Griffith, Cook, Guyatt, & Charles, 1999). The closed-ended questionnaire format resulted in a 22% higher cooperation rate compared with the open-ended format.

Personalization and sponsorship. Several studies assessed the impact of a personalized cover letter on physician participation (Table 2). Three studies

found that a personalized cover letter and/or the inclusion of a handwritten note resulted in significantly higher response rates (Leece et al., 2006; Maheux, Legault, & Lambert, 1989; Olson et al., 1993). Another, however, found no difference in response rate between those sent a hand-signed letter and those sent a letter with a scanned signature (McKenzie-McHarg, Tully, Gates, Ayers, & Brocklehurst, 2005). Collectively, personalized cover letters and/or mailout packages had a weighted overall effect size (OR 1.51; 95% CI 1.1–2.2) reflecting an association with higher physician response. Direct contact (e.g., prenotification calls and/or letters in advance of a survey and follow-up contact) is another mechanism to personalize a survey that often results in improved physician response (Bostick, Pirie, Luepker, & Kofron, 1992; Heywood et al., 1995; Osborn, Ward, & Boyle, 1996; Ward et al., 1998; Ward & Wain, 1994). This includes contact by a medical peer, which has been found in some studies to increase physician participation (Bostick et al., 1992; Haywood et al., 1995). Exceptions include a study by Temple-Smith, Mulvey, & Doyle (1998) in which a medical researcher was able to contact a higher proportion of cases (80% vs. 69%), without increasing overall response. Taken as a whole, direct contact had a weighted overall effect size denoting a relationship with increased physician response (OR 2.3; 95% CI 1.42–3.64). Finally, attempts have been made to personalize surveys using endorsements by opinion leaders and/or professional associations, with mixed results (Asch & Christakis, 1994; Bhandari et al., 2003; Olson et al., 1993). Although organizational sponsorship generally improved participation, a study supported by expert surgeons actually resulted in a lower response rate, suggesting possible "limits of leadership" related to collegial sponsorship (Bhandari et al., 2003).

Type of mailing. Six studies were identified that compared physician response by type of mail and/or return mail employed (Table 2). Two studies were identified that examined the impact of the initial mailing on physician participation. In one study, certified mail resulted in a 16.5% increase in participation (OR 2.085; 95% CI 1.35–3.22) compared with first-class mail (Del Valle, Morgenstern, Rogstad, Albright, & Vickrey, 1997). In the other, FedEx resulted in an 8% increase over first-class mail (OR 1.444; 95% CI 0.90–2.33; Kasprzyk et al., 2001). With regard to the return mailing, studies have consistently found that return envelopes with first-class stamps result in higher physician response compared with franked or business reply return envelopes (Shiono & Klebanoff, 1991; Streiff, Dundes, & Spivak, 2001; Urban, Anderson, & Tseng, 1993). The weighted overall effect size across these three studies reflected an association between the use of first-class stamps and physician response (OR 1.3; 95% CI 1.1–1.5). Additionally, one

study compared the influence of multiple return mailing strategies on physician response to mailed questionnaires (Gullen & Garrison, 1973). Although this study identified a postcard reply as having the lowest participation rate compared to bulk and first-class mail, the different strategies were assessed in conjunction with other changes in the mail out package (e.g., different cover letters), making it impossible to determine the independent effects of the return mailing on physician cooperation. With regard to follow-up mailings, research suggests that the inclusion of a replacement questionnaire with the follow-up contact will improve participation (Ogborne, Rush, & Fondacaro, 1986; Vogel, Nowacek, Harlan, Tribble, & Thorup, 1983). One study was also identified that examined envelope size (initial mailing) on physician cooperation (Halpern et al., 2002). This study found no differences in the response rates of general internists and family practitioners to a study received in either a large or small envelope. Although the interventions differ, collectively the weighted overall effect size across studies examining postage/mailing strategies reflected an association between use of these strategies and physician response (OR 1.4; 95% CI 1.11–1.69).

Survey mode. Finally, several studies have examined survey mode as one potential mechanism to improve physician participation. For example, studies have examined response rates for telephone versus mailed surveys, with mixed results. Although some studies found mail surveys to have higher response rates compared with telephone surveys (Hocking, Lim, Read, & Hellard, 2006; Ogborne et al., 1986), others suggest that telephone interviews result in higher response rates (Parsons et al., 1994; Sibbald, Addington-Hall, Brenneman, & Freeling, 1994; Thran & Hixson, 2000). Parsons et al. (1994) also found evidence of mode preference across different medical specialties, with family practitioners more likely to select the mail option than surgeons. Use of e-mail and fax technologies has also been explored. In direct comparisons with mailed questionnaires, e-mail resulted in significantly lower physician response rates (Akl, Maroun, Klocke, Montori, & Schunemann, 2005; Raziano, Jayadevappa, Valenzula, Weiner, & Lavizzo-Mourey, 2001; Seguin, MacDonald, Godwin, & McCall, 2004; VanDenKerkhof, Parlow, Goldstein, & Milne, 2004). With regard to fax technology, there is evidence suggesting that, when incorporated within a mixed-method design, it may be a cost-effective method of increasing physician participation. In one study where pediatricians were offered a choice of three response modes, 26% responded by e-mail, 47% by fax, and 41% by mail (McMahon et al., 2003). In another, a larger percentage of respondents requested to be surveyed by fax compared with telephone or mail (Lensing et al., 2000). Finally, researchers have also

begun to explore the utility of the Internet in surveying physicians (see Braithwaite, Emery, de Lusignan, & Sutton, 2003). In one controlled comparison, an Internet-based survey resulted in a significantly lower response rate compared with a traditional mail survey (Leece et al., 2004). Other mixed mode (Internet vs. mail) studies of American urologists identified similar problems (Hollowell, Patel, Bales, & Gerber, 2000; Kim et al., 2000). There is also evidence that Internet surveys may present methodological issues related to sample representativeness (Braithwaite et al., 2003).

Discussion

Despite the importance of physician surveys in health services and policy research, and the ongoing concern over potential biases associated with low response rates to these surveys, there are relatively few randomized trials examining potential strategies to improve physician cooperation. Our review of available studies, however, does suggest a number of promising strategies for enhancing physician cooperation. Financial incentives, in particular, were shown to be effective in improving physician response to surveys. Surprisingly, even a small $1 incentive significantly improved participation by 18% to 21% (Everett et al., 1997; Mizes et al. 1984). In fact, the combined evidence regarding appropriate levels of incentive suggests that the steepest part of the incentive/response curve may be between $0 and $1, with diminishing returns to serial increments above that amount (Halpern et al., 2002; VanGeest & Johnson, 2001). These results mirror general population studies in which the use of token financial incentives averaged nearly a 20% increase in survey participation (Church, 1993; Fox, Clark, & Kim, 1988; Heberlein & Baumgartner, 1978; Yammarino, Skinner, & Childress, 1991). They are also consistent with studies examining the use of monetary incentives in surveys of nurses and allied health professionals (Paul, Walsh, & Tzelepis, 2005; Ulrich et al., 2005). In contrast, token nonmonetary incentives were much less effective in improving physician cooperation, with the cumulative evidence suggesting that nonmonetary inducements work only if physicians value them.

A number of design-based strategies were also identified as being potentially effective in improving physician response rates. For example, survey mode clearly had an impact on response, with postal and telephone surveys resulting in higher average return rates across the studies reviewed. Existing evidence also supports the use of mixed-mode formats that include fax and possibly e-mail options, as these give physicians more alternatives by which to respond to a survey in their busy schedules (Lensing et al., 2000; Parsons

et al., 1994). Choice of survey mode, however, often entails significant cost considerations and more studies are needed to identify the most cost-effective methodologies in surveys of physicians.

Although only a limited number of studies examined the impact of questionnaire design on physician cooperation, existing evidence does suggest that researchers should be succinct when designing physician questionnaires. Again, this is no surprise, given previous research on the association between questionnaire length and survey response (Dillman, Sinclair, & Clark, 1993; Heberlein & Baumgartner, 1978; Nakash, Hutton, Jorstad-Stein, Gates, & Lamb, 2006; Olmsted, Murphy, McFarlane, & Hill, 2005). Previous qualitative research with physicians also identified questionnaire length as a major factor in their willingness to participate (Kasprzyk et al., 2001). With regard to item response format, caution is necessary when drawing conclusions from the single study supporting the use of closed- versus open-item formats, as previous reviews have identified professionals as being more resistant to closed-ended questions compared with the general public (Deutscher, 1956; Sudman, 1985).

Findings related to personalization, sponsorship, and even mailing strategies are consistent with Sudman's (1985) recommendation concerning the need to establish relevance as a means of improving response. In a meta-analysis of population-based studies, use of prenotification letters and sponsorship had the largest effect sizes for increasing response rates (Fox et al., 1988). Similarly, endorsements by local, state, or national organizations also typically result in improved physician participation (Asch & Christakis, 1994). Use of certified mail and/or courier companies such as FedEx also enhances the importance of the mail out package, increasing the likelihood of physician receipt and cooperation. Finally, a note with regard to the use of replacement questionnaires in follow-up mailings: decisions must be carefully balanced against cost considerations, as previous reviews have found little evidence supporting the use of replacement questionnaires in promoting participation generally (Heberlein & Baumgartner, 1981).

Although not addressed explicitly in this review, number of contacts/length of field periods may be one of the most important determinants of physician response. Research suggests that number of contacts may explain up to 40% of the variance in response rates in surveys of the general public (Heberlein & Baumgartner, 1978). Similarly, there is growing consensus in the literature that lengthy field periods may be necessary in maximizing physician participation (Goodman & Jensen, 1981; Parsons et al., 1994; Sudman, 1985; Thran, Olson, & Strouse, 1987). For example, in one study, more than 30% of the completed surveys were obtained after 1 contact, but another 20% required

11 or more contact attempts (Parsons et al., 1994). This same study highlighted differences in number of contact attempts by mode of data collection, with only 6% of telephone interviews completed after 1 contact attempt compared with 60% finalized mail surveys in one mailing of the questionnaire.

Ultimately, a decision regarding what methodologies to employ to improve physician cooperation are embedded within cost-quality trade offs (Olmsted et al., 2005). The good news for those working in limited resource environments is that previous reviews identified smaller-than-anticipated differences between physician respondents and nonrespondents and between early and late responders (Field et al., 2002; Kellerman & Herold, 2001; McFarlane et al., 2006), suggesting low rates of nonresponse bias. This is often because of the homogeneity of physicians with regard to knowledge, training, attitudes, and behavior. Although changing, physicians remain a relatively homogeneous population compared to the general population. This suggests that researchers can still be strategic in employing any of the strategies identified in this review, especially when conducting a survey on a tight budget. That said, researchers should still make every effort to improve access to their target population by implementing design strategies demonstrated to improve physician participation rates, thereby increasing the legitimacy and credibility of their results.

References

Akl, E. A., Maroun, N., Klocke, R. A., Montori, V., & Schunemann, H. J. (2005). Electronic mail was not better than postal mail for surveying residents and faculty. *Journal of Clinical Epidemiology*, *58*, 425-429.

Asch, D. A., & Christakis, N. A. (1994). Different response rates in a trial of two envelop styles in mail survey research. *Epidemiology*, *5*, 364-365.

Asch, D. A., Christakis, N. A., & Ubel, P. A. (1998). Conducting physician mail surveys on a limited budget: A randomized trial comparing $2 bill versus $5 bill incentives. *Medical Care*, *36*, 95-99.

Asch, D. A., Connor, S. E., Hamilton, E. G., & Fox, S. A. (2000). Problems in recruiting community-based physicians for health services research. *Journal of General Internal Medicine*, *15*, 591-599.

Asch, D. A., Jedrziewski, M. K., & Christakis, N. A. (1997). Response rates to mail surveys published in medical journals. *Journal of Clinical Epidemiology*, *50*, 1129-1136.

Baron, G., De Wals, P., & Milord, F. (2001). Cost effectiveness of a lottery for increasing physicians' responses to a mail survey. *Evaluation and the Health Professions*, *24*, 47-52.

Berk, M. L. (1985). Interviewing physicians: The effect of improved response rate. *American Journal of Public Health*, *75*, 1338-1340.

Berk, M. L., Edwards, W. S., & Gay, N. L. (1993). The use of a prepaid incentive to convert nonresponders on a survey of physicians. *Evaluation and the Health Professions*, *16*, 239-245.

Berry, S. H., & Kanouse, D. E. (1987). Physician response to a mailed survey: An experiment in timing of payment. *Public Opinion Quarterly*, *51*, 102-114.

Bhandari, M., Devereaux, P. J., Swiontkowski, M. F., Schemitsch, E. H., Shankardass, K., Sprague, S., et al. (2003). A randomized trial of opinion leader endorsement in a survey of orthopaedic surgeons: Effect on primary response rate. *International Journal of Epidemiology*, *32*, 634-636.

Bonito, A. J., Samsa, G. P., Akin, D. R., & Matchar, D. B. (1997). Use of a non-monetary incentive to improve physician responses to a mail survey. *Academic Medicine*, *72*, 73.

Bostick, R. M., Pirie, P., Luepker, R. V., & Kofron, P. M. (1992). Using physician caller follow-ups to improve the response rate to a physician telephone survey: Its impact and implications. *Evaluation and the Health Professions*, *15*, 420-433.

Braithwaite, D., Emery, J., de Lusignan, S., & Sutton, S. (2003). Using the Internet to conduct surveys of health professionals: A valid alternative? *Family Practice*, *20*, 545-551.

Brehaut J. C., Graham, I. D., Visentin, L., & Stiell, I. G. (2006). Print format and sender recognition were related to survey completion rate. *Journal of Clinical Epidemiology*, *59*, 635-641.

Burt, C. W., & Woodwell, D. (2005, November). *Tests of methods to improve response to physician surveys*. Paper presented at the 2005 Federal Committee on Statistical Methodology, Arlington, VA.

Cartwright, A. (1978). Professionals as responders: Variations in and effects of response rates to questionnaires, 1961-77. *British Medical Journal*, *2*, 1419-1421.

Cartwright A., & Ward, A. W. (1968). Variations in general practitioners' response to postal questionnaires. *British Journal of Preventive and Social Medicine*, *22*, 199-205.

Church, A. H. (1993). Estimating the effect of incentives on mail survey response rates: A metal-analysis. *Public Opinion Quarterly*, *57*, 62-79.

Clark, T. J., Khan, K. S., & Gupta, J. K. (2001a). Provision of pen along with questionnaire does not increase the response rate to a postal survey: A randomized controlled trial. *Journal of Epidemiology and Community Health*, *55*, 595-596.

Clark, T. J., Khan, K. S., & Gupta, J. K. (2001b). Effect of paper quality on the response rate to a postal survey: A randomized controlled trial. *BMC Medical Research Methodology*, *1*, 12.

Cockburn, J., Campbell, E., Gordon, J. J., & Sanson-Fisher, R. W. (1988). Response bias in a study of general practice. *Family Practice*, *5*, 18-23.

Cull, W. L., O'Connor, K. G., Sharp, S., & Tang, S. S. (2005). Response rates and response bias for 50 surveys of pediatricians. *Health Services Research*, *40*, 213-226.

Cummings, S. M., Savitz, L. A., & Konrad, T. R. (2001). Reported response rates to mailed physician questionnaires. *Health Services Research*, *35*, 1347-1355.

Deehan, A., Templeton, L., Taylor, C., Drummond, C., & Strang, J. (1997). The effect of cash and other financial inducements on the response rate of general practitioners in a national postal survey. *British Journal of General Practice*, *47*, 87-90.

Delnevo, C. D., Abatemarco, D. J., & Steinberg, M. B. (2004). Physician response rates to a mail survey by specialty and timing of incentive. *American Journal of Preventive Medicine*, 26, 234-236.

Del Valle, M., Morgenstern, H., Rogstad, T. L., Albright, C., & Vickrey, B. G. (1997). A randomized trial of the impact of certified mail on response rate to a physician survey, and a cost-effectiveness analysis. *Evaluation and the Health Professions*, *20*, 389-406.

Deutscher, I. (1956). Physician reactions to a mailed questionnaire: A study in "resistentialism." *Public Opinion Quarterly*, *20*, 599-604.

Dillman, D. A., Sinclair, M. D., & Clark, J. R. (1993). Effects of questionnaire length, respondent-friendly design, and a difficult question on response rates for occupant-addressed census mail surveys. *Public Opinion Quarterly*, *57*, 289-304.

Donaldson, G. W., Moinpour, C. M., Bush, N. E., Chapko, M., Jocom, J., Siadak, M., et al. (1999). Physician participation in research surveys: A randomized study of inducements to return mailed research questionnaires. *Evaluation and the Health Professions*, *22*, 427-441.

Easton, A. N., Price, J. H., Telljohann, S. K., & Boehm, K. (1997). An informational versus monetary incentive in increasing physicians' response rates. *Psychological Reports*, *81*, 968-970.

Emanuel, E. J., Fairclough, D., Clarridge, B. C., Blum, D., Bruera, E., Penley, W.C., et al. (2000). Attitudes and practices of U.S. oncologists regarding euthanasia and physician-assisted suicide. *Annals of Internal Medicine*, *133*, 527-532.

Everett, S. A., Price, J. H., Bedell, A. W., & Telljohann, S. K. (1997). The effect of a monetary incentive in increasing the return rate of a survey to family physicians. *Evaluation and the Health Professions*, *20*, 207-214.

Farber N. J., Simpson, P., Salam, T., Collier, V. U., Weiner, J., & Boyer, E. G. (2006). Physicians' decisions to withhold and withdraw life-sustaining treatment. *Archives of Internal Medicine*, *166*, 560-564.

Field, T. S., Cadoret, C. A., Brown, M. L., Ford, M., Greene, S. M., Hill, D., et al. (2002). Surveying physicians: Do components of the "Total Design Approach" to optimizing survey response rates apply to physicians? *Medical Care*, *40*, 596-606.

Fox, R. J., Clark, M. R., & Kim, J. (1988). Mail survey response rate: A meta-analysis of selected techniques for inducing response. *Public Opinion Quarterly*, *52*, 467-491.

Gattellari, M., & Ward, J. E. (2001). Will donations to their learned college increase surgeons' participation in surveys? A randomized trial. *Journal of Clinical Epidemiology*, *54*, 645-650.

Goodman, L., & Jensen, L. (1981). Economic surveys of medical practice: AMA's periodic survey of physicians, 1966-1978. In Sudman S (Ed.), *Proceedings of the Third Biennial Conference on Health Services Research* (pp. 56-65). Rockville, MD: National Center for Health Services Research.

Griffith, L. E., Cook, D. J., Guyatt, G. H., & Charles C. A. (1999). Comparison of open and closed questionnaire formats in obtaining demographic information from Canadian general internists. *Journal of Clinical Epidemiology*, *52*, 977-1005.

Gullen, W. H., & Garrison, G. E. (1973). Factors influencing physicians' response to mailed questionnaires. *Health Services Reports*, *88*, 510-514.

Gunn, W. J., & Rhodes, I. N. (1981). Physician response rates to a telephone survey: Effects of monetary incentive level. *Public Opinion Quarterly*, *45*, 109-115.

Halpern, S. D., Ubel, P. A., Berlin, J. A., & Asch, D. A. (2002). Randomized trail of $5 versus $10 monetary incentives, envelope size, and candy to increase physician response rates to mailed questionnaires. *Medical Care*, *40*, 834-839.

Heberlein, T. A., & Baumgartner, R. (1978). Factors affecting response rates to mailed questionnaires: A quantitative analysis of the published literature. *American Sociological Review*, *43*, 447-462.

Heberlein, T. A., & Baumgartner, R. (1981). Is a questionnaire necessary in a second mailing? *Public Opinion Quarterly*, *45*, 102-108.

Heywood, A., Mudge, P., Ring, I., & Sanson-Fisher, R. (1995). Reducing systematic bias in studies of general practitioners: The use of a medical peer in the recruitment of general practitioners in research. *Family Practice*, *12*, 227-231.

Hing, E., Schappert, S. M., Burt, C. W., & Shimizu, I. M. (2005). Effects of form length and item format on response patterns and estimates of physician office and hospital outpatient department visits. National Ambulatory Medical Care Survey and National Hospital Ambulatory Medical Care Survey. *Vital Health Statistics*, *2*, 139, 1-32.

Hocking, J. S., Lim, M. S., Read, T., & Hellard, M. (2006). Postal surveys of physicians gave superior response rates over telephone interviews in a randomized trial. *Journal of Clinical Epidemiology*, *59*, 521-524.

Hollowell, C. M. P., Patel, R. V., Bales, G. T., & Gerber, G. S. (2000). Internet and postal survey of endourologic practice patterns among American urologists. *The Journal of Urology*, *163*, 1779-1782.

Hughes, P. H., Storr, C. L., Brandenburg, N. A., Baldwin, D.C., Jr., Anthony, J. C., & Sheehan, D. V. (1999). Physician substance use by medical specialty. *Journal of Addictive Diseases*, *18*, 23-37.

Jepson, C., Asch, D. A., Hershey, J. C., & Ubel, P. A. (2005). In a mailed physician survey, questionnaire length had a threshold effect on response rate. *Journal of Clinical Epidemiology*, *58*, 103-105.

Johnson T. P., Parsons, J. A., Warnecke, R. B., & Kaluzny, A. D. (1993). Dimensions of mail questionnaires and response quality. *Sociological Focus*, *26*, 271-274.

Kaner, E. F., Haighton, C. A., & McAvoy, B. R. (1998). "So much post, so busy with practice—so, no time!": A telephone survey of general practitioners' reasons for not participating in postal questionnaire surveys. *British Journal of General Practice*, *48*, 1067-1069.

Kasprzyk, D., Montano, D. E., St. Lawrence, J. S., & Phillips, W. R. (2001). The effects of variations in mode of delivery and monetary incentive on physicians' responses to a mailed survey assessing STD practice patterns. *Evaluation and the Health Professions*, *24*, 3-17.

Kellerman, S. E., & Herold, J. (2001). Physician response to surveys: A review of the literature. *American Journal of Preventive Medicine*, *20*, 61-67.

Kim, H. L., Hollowell, C. M. P., Patel, R. V., Bales, G. T., Clayman, R. V., & Gerber, G. S. (2000). Use of new technology in endourology and laparoscopy by American urologists: Internet and postal survey. *Urology*, *56*, 760-765.

Leece, P., Bhandari, M., Sprague, S., Swiontkowski, M. F., Schemitsch, E. H., & Tornetta, P. (2006). Does flattery work? A comparison of 2 different cover letters for an international survey of orthopedic surgeons. *Canadian Journal of Surgery*, *49*, 90-95.

Leece, P., Bhandari, M., Sprague, S., Swiontkowski, M. F., Schemitsch, E. H., Tornetta, P., et al. (2004). Internet versus mailed questionnaires: A controlled comparison. *Journal of Medical Internet Research*, 6, e39.

Lensing, S. Y., Gillaspy, S. R., Simpson, P. M., Jones, S. M., James, J. M., & Smith, J. M. (2000). Encouraging physicians to respond to surveys through the use of fax technology. *Evaluation and the Health Professions*, *23*, 348-359.

Leung, G. M., Ho, L. M., Chan, M. F., Johnston, J. M., & Wong, F. K. (2002). The effects of cash and lottery incentives on mailed surveys to physicians: A randomized trial. *Journal of Clinical Epidemiology*, *55*, 801-807.

Leung, G. M., Johnston, J. M., Saing, H., Tin, K. Y. K., Wong, I. O. L., & Ho, L. (2004). Prepayment was superior to postpayment cash incentives in a randomized postal survey among physicians. *Journal of Clinical Epidemiology*, *57*, 777-784.

MacPherson, I., & Bisset, A. (1995). Not another questionnaire!: Eliciting the views of general practitioners. *Family Practice*, *12*, 335-338.

Maheux, B., Legault, C., & Lambert, J. (1989). Increasing response rates in physicians' mail surveys: An experimental study. *American Journal of Public Health*, *79*, 638-639.

Marin, J., & Howe, H. L. (1984). Physicians' attitudes toward breast self-examination: A pilot study. *Evaluation and the Health Professions*, *7*, 193.

McAuliffe, W. E., Rohman, M., Santangelo, S., Fieldman, B., Magnuson, E., Sobol, A., et al. (1986). Psychoactive drug use among practicing physicians and medical students. *New England Journal of Medicine*, *315*, 805-810.

McAvoy, B. R., & Kaner, E. F. S. (1996). General practice postal surveys: A questionnaire too far? *British Medical Journal*, *313*, 732-733.

McDermott, M. M., Greenland, P., Hahn, E.A., Brogan, D., Cella, D., Ockene, J., et al. (2003). The effects of continuing medical education credits on physician response rates to a mailed questionnaire. *Health Marketing Quarterly*, *20*, 27-42.

McFarlane, E., Olmsted, M. G., Murphy, J., & Hill, C. A. (2006, May). *Nonresponse bias in a mail survey of physicians*. Paper presented at the annual conference of the American Association for Public Opinion Research, Montreal, Quebec.

McKenzie-McHarg, K., Tully, L., Gates, S., Ayers, S., & Brocklehurst, P. (2005). Effect on survey response rate of hand written versus printed signature on a covering letter: Randomised controlled trial. *BMC Health Services Research*, *5*, 52.

McMahon, S. R., Iwamoto, M., Massoudi, M. S., Yusuf, H. R., Stevenson, J. M., David, F., et al. (2003). Comparison of e-mail, fax and postal surveys of pediatricians. *Pediatrics*, *111*, e299-e303.

Meier, D. E., Emmons, C. A., Wallenstein, S., Quill, T., Morrison, R. S., & Cassel, C. K. (1998). A national survey of physician-assisted suicide and euthanasia in the United States. *New England Journal of Medicine*, *338*, 1193-1201.

Mizes, S., Fleece, L., & Roos, C. (1984). Incentives for increasing return rates: Magnitude levels, response bias, and format. *Public Opinion Quarterly*, *48*, 794-800.

Moore, D., & An, L. (2001, August). *The effect of repetitive token incentives and priority mail on response to physician surveys*. Proceedings of the annual meeting of the American Statistical Association, Atlanta, GA.

Mosca, L., Linfante, A. H., Benjamin, E. J., Berra, K., Hayes, S. N., Walsh, B. W., et al. (2005). National study of physician awareness and adherence to cardiovascular disease prevention guidelines. *Circulation*, *111*, 499-510.

Moses, S. H., & Clark, T. J. (2004). Effect of prize draw incentive on the response rate to a postal survey of obstetricians and gynecologists: A randomized controlled trial. *BMC Health Services Research*, 4, 14.

Mullen, P. D., Easling, I., Nixon, S. A., Koester, D. R., & Biddle, A. K. (1987). The cost-effectiveness of randomized incentive and follow-up contacts in a national mail survey of family physicians. *Evaluation and the Health Professions*, *10*, 232-245.

Myerson, S. (1993). Improving the response rates in primary care research. Some methods used in a survey on stress in general practice since the new contract (1990). *Family Practice*, *10*, 342-346.

Nakash, R. A., Hutton, J. L., Jorstad-Stein, E. C., Gates, S., & Lamb, S. E. (2006). Maximising response to postal questionnaires: A systematic review of randomized trials in health research. *BMC Medical Research Methodology*, *6*, 5.

Ogborne, A. C., Rush, R., & Fondacaro, R. (1986). Dealing the nonrespondents in a mail survey of professionals. *Evaluation and the Health Professions*, *9*, 121-128.

Olmsted, M. G., Murphy, J., McFarlane, E., & Hill, C. (2005, May). *Evaluating methods for increasing physician survey cooperation*. Paper presented at the annual conference of the American Association for Public Opinion Research, Miami Beach, FL.

Olson, L., Schneiderman, M., & Armstrong, R. V. (1993). Increasing physician survey response rates without biasing survey results. In *Proceedings of the section on survey research methods of the American Statistical Association* (pp. 1036-1041) Alexandria, VA: American Statistical Association.

Osborn, M., Ward, J., & Boyle, C. (1996). Effectiveness of telephone prompts when surveying general practitioners: A randomized trial. *Australian Family Physician*, *25*, S41-S43.

Parsons, J. A., Warnecke, R. B., Czaja, R. F., Barnsley, J., & Kaluzny, A. (1994). Factors associated with response rates in a national survey of primary care physicians. *Evaluation Review*, *18*, 756-766.

Paul, C. L., Walsh, R. A., & Tzelepis, F. (2005). A monetary incentive increases postal survey response rates for pharmacists. *Journal of Epidemiology and Community Health*, *59*, 1099-1101.

Raziano, D. B., Jayadevappa, R., Valenzula, D., Weiner, M., & Lavizzo-Mourey, R. (2001). E-mail versus conventional postal mail survey of geriatric chiefs. *Gerontologist*, *41*, 799-804.

Robertson, J, Walkom, E. J., & McGettigan, P. (2005). Response rates and representativeness: A lottery incentive improves physician survey return rates. *Pharmacoepidemiology and Drug Safety*, *14*, 571-577.

Sallis, J. F., Fortmann, S. P., Solomon, D. S., & Farquhar, J. F. (1984). Increasing returns of physician surveys. *American Journal of Public Health*, *74*, 1043.

Schroy, P. C., 3rd, Geller, A. C., Crosier Wood, M., Page, M., Sutherland, L., Holm, L.J., et al. (2001). Utilization of colorectal cancer screening tests: A 1997 survey of Massachusetts internists. *Preventive Medicine*, *33*, 381-391.

Seguin, R., MacDonald, S., Godwin, M., & McCall, M. (2004). E-mail or snail mail? Randomized controlled trial on which works better for surveys. *Canadian Family Physician*, *50*, 414-419.

Shiono, P. H., & Klebanoff, M. A. (1991). The effect of two mailing strategies on the response to a survey of physicians. *American Journal of Epidemiology*, *134*, 539-542.

Sibbald, B., Addington-Hall, J., Brenneman, D., & Freeling, P. (1994). Telephone versus postal surveys of general practitioners: Methodological considerations. *British Journal of General Practice*, *44*, 297-300.

Stocks N., & Gunnell, D. (2000). What are the characteristics of GPs who routinely do not return postal questionnaires: A cross sectional study. *Journal of Epidemiology and Community Health*, *54*, 940-941.

Streiff, M. B., Dundes, L., & Spivak, J. L. (2001). A mail survey of United States hematologists and oncologists: A comparison of business reply versus stamped return envelopes. *Journal of Clinical Epidemiology*, *54*, 430-432.

Sudman, S. (1985). Mail surveys of reluctant professionals. *Evaluation Review*, *9*, 349-360.

Tamayo-Sarver, J. H., & Baker, D. W. (2004). Comparison of responses to a $2 bill versus a chance to win $250 in a mail survey of emergency physicians. *Academic Emergency Medicine*, *11*, 888-892.

Tambor, E. S., Chase, G. A., Faden, R. R., Geller, G., Hofman, K. J., & Holtzman, N. A. (1993). Improving response rates through incentive and follow-up: The effect on a survey of physicians' knowledge of genetics. *American Journal of Public Health*, *83*, 1599-1603.

Temple-Smith, M., Mulvey, G., & Doyle, W. (1998). Maximizing response rates in a survey of general practitioners: Lessons from a Victorian survey on sexually transmissible diseases. *Australian Family Physician*, *27*(Suppl. 1), S15-S18.

Templeton, L., Deehan, A., Taylor, C., Drummond, C., & Strang, J. (1997). Surveying general practitioners: Does a low response rate matter? *British Journal of General Practice*, *47*, 91-94.

Thomson C. E., Paterson-Brown, S., Russell, D., McCaldin, D., & Russell, I. T. (2004). Short report: Encouraging GPs to complete postal questionnaires—one big prize or many small prizes? A randomized controlled trial. *Family Practice*, *21*, 697-698.

Thran, S. L., & Berk M. L. (1993). Survey of physicians: An overview. In *Proceedings of the International Conference on Establishment Surveys* (pp. 83-92). Alexandria, VA: American Statistical Association.

Thran, S. L., & Gonzalez, M. L. (1999). Characteristics of unlocatable physicians. In *Proceedings of the section on survey research methods of the American Statistical Association* (pp. 529-532). Alexandria, VA: American Statistical Association.

Thran, S. L., & Hixson, J. S. (2000). Physician surveys: Recent difficulties and proposed solutions. In *Proceedings of the section on survey research methods of the American Statistical Association* (pp. 233-237). Alexandria, VA: American Statistical Association.

Thran, S. L., Olson, L., & Strouse, R. (1987). The effectiveness and costs of special data collection efforts in a telephone survey of physicians. In *Proceedings of the survey research section of the American Statistical Association* (pp. 761-766). Alexandria, VA: American Statistical Association.

Tu, S. (2003). Developmental epidemiology: A review of three key measures of effect. *Journal of Clinical Child & Adolescent Psychology*, *32*(2), 187-192.

Ulrich, C. M., Danis, M., Koziol, D., Garrett-Mayer, E., Hubbard, R., & Grady, C. (2005). Does it pay to play? A randomized trial of prepaid financial incentives and lottery incentives in surveys of nonphysician healthcare professionals. *Nursing Research*, *54*, 178-183.

Urban, N., Anderson, G. L., & Tseng, A. (1993). Effects on response rates and cost of stamps vs. business reply in a mail survey of physicians. *Journal of Clinical Epidemiology*, *46*, 455-459.

VanDenKerkhof, E. G., Parlow, J. L., Goldstein, D. H., & Milne, B. (2004). In Canada, anesthesiologists are less likely to respond to an electronic, compared to a paper questionnaire. *Canadian Journal of Anesthesia*, *51*, 449-454.

VanGeest, J. B., & Johnson, T. P. (2001, October). *Methodologies for improving response rates in surveys of physicians*. Paper presented at the annual meeting of the American Public Health Association, Atlanta, GA.

VanGeest, J. B., Wynia, M. K., Cummins, D. S., & Wilson, I. B. (2001). Effects of different monetary incentives on the return rate of a national survey of physicians. *Medical Care*, *39*, 197-201.

Vogel, L. L., Nowacek, G., Harlan, J. F., Tribble, A., & Thorup, O. A. (1983). Impact of replacement questionnaire on the response rate of practicing physicians to mail questionnaire. *Journal of Medical Education*, *58*, 905.

Ward, J., Bruce, T., Holt, P., D'Este, K., & Sladden, M. (1998). Labour-saving strategies to maintain survey response rates: A randomized trial. *Australian New Zealand Journal of Public Health*, *22*(Suppl.), 394-396.

Ward, J., & Wain, G. (1994). Increasing response rates of gynecologists to a survey: A randomized trial of telephone prompts. *Australian Journal of Public Health*, *18*, 332-334.

Webster, B. S., Courtney, T. K., Huang, Y. H., Matz, S., & Christiani, D. C. (2005). Physicians' initial management of acute low back pain versus evidence-based guidelines. Influence of Sciatica. *Journal of General Internal Medicine*, *20*, 1132-1135.

Wynia, M. K., Cummins, D. S., VanGeest, J. B., & Wilson, I. B. (2000). Physician manipulation of reimbursement rules for patients: Between a rock and a hard place. *Journal of the American Medical Association*, *283*, 1858-1865.

Yammarino, F. J., Skinner, S. J., & Childress, T. L. (1991). Understanding mail survey response behavior: A meta-analysis. *Public Opinion Quarterly*, *55*, 613-639.

ELSEVIER

Contraception 75 (2007) 177–184

Contraception

Original research article

Intrauterine contraception: evaluation of clinician practice patterns in Kaiser Permanente Northern California

Debbie Postlethwaite[a,*], Ruth Shaber[a], Victoria Mancuso[a], Jean Flores[b], Mary Anne Armstrong[c]

[a]*Kaiser Permanente Women's Health Research Institute, Oakland, CA 94612, USA*
[b]*Kaiser Permanente Quality and Operations Support, Oakland, CA 94612, USA*
[c]*Kaiser Permanente Division of Research, Oakland, CA 94612, USA*

Received 28 July 2006; revised 23 October 2006; accepted 24 October 2006

Abstract

Background: Despite the medical evidence, few women of reproductive age in the United States use intrauterine contraception (IUC) in comparison with women worldwide. To reduce cost as a barrier, Kaiser Permanente removed the cost to the patient for IUC throughout California in 2002. The goal of this study was to evaluate whether providing evidence-based information about IUC would result in changes in the knowledge, attitudes and practice patterns of clinicians and in greater IUC utilization as compared with removing cost alone.
Study Design: A comprehensive education intervention was conducted in half of Kaiser Permanente Northern California ob-gyn departments. To make comparisons between the intervention and comparison sites, we surveyed clinicians in both groups before and after the intervention about their IUC knowledge, attitudes as well as practice patterns and collected utilization data for 27 months.
Results: Statistically significant changes in attitudes and practice patterns were reported by the intervention group as compared with the usual care comparison group. By the end of the study, change in IUC utilization was significantly greater in the intervention group (utilization rate=9.57/1000) as compared with the comparison group (utilization rate=7.35/1000) ($p=.02$).
Conclusion: A multifaceted approach to providing evidence-based clinician and patient education resulted in statistically significant reported changes in attitudes and practice patterns and in greater IUC utilization as compared with usual practice.

Keywords: IUC; LNG-20 IUC; Cu-T 380A; Academic detailing

1. Introduction

Much concern has been voiced in recent years about the lag time from research to the integration of the medical evidence into clinical practice toward improved patient outcomes. It was recently stated in *Harvard Business Review* that it takes on average 17 years for emerging research to be translated into standard practice [1]. In recent years, there has been much research devoted to studying the gap that exists between research and evidence-based practice [2–6].

The story about intrauterine contraception (IUC) use in the United States is a perfect example of the delay from acquiring clinical evidence to changing clinician practice. Worldwide, IUC is used by approximately 15% of women of reproductive age overall, 9% of those in developed countries and 15% of those in underdeveloped countries [7,8]. Although there has been more than a decade of evidence presented about the effectiveness and cost-effectiveness of IUC [9,10], its safety [11–18] and the high reported satisfaction [19] among women who use it, IUC is still used by fewer than 2% of women of reproductive age in the United States [7,8]. Experts in the reproductive field continue to pose the following questions: (1) What will it take to change clinician practice and consumer attitudes regarding intrauterine contraception? and (2) What will it take to shift couples toward use of more cost-effective contraception? There have been numerous efforts over the last decade to share the medical evidence about IUC regarding its safety and efficacy among health care professionals and consumers. However, it is remarkable how little has changed in attitudes toward IUC and in its utilization across the United States.

In January 2002, Kaiser Permanente Northern California (KPNC) removed copayment (cost to the patient) for the

* Corresponding author. Tel.: +1 510 987 3257; fax: +1 510 873 5089.
E-mail address: Debbie.A.Postlethwaite@kp.org (D. Postlethwaite).

0010-7824/$ – see front matter © 2007 Elsevier Inc. All rights reserved.
doi:10.1016/j.contraception.2006.10.010

most cost-effective contraceptives (intrauterine, injectable, implantable and emergency). This change was made throughout the region, affecting all KPNC health plan members regardless of the plan they purchased. Additional operational changes were made throughout the region, which made it easier for patients to obtain IUC directly from their medical providers, such as stocking devices in the medical offices instead of the pharmacies. The investigators hypothesized that offering current and consistent evidence-based information about IUC to clinicians and patients after removal of cost as a barrier would result in changes in knowledge and attitudes and would have a more rapid impact on changing practice patterns and IUC device insertion rates.

2. Materials and methods

The KPNC Women's Health Research Institute conducted a study approved by the KPNC Institutional Review Board to evaluate ob-gyn clinician IUC knowledge, attitudes and practice patterns, to provide a multifaceted educational intervention and to track IUC utilization over a 27-month period. The findings were based on changes in IUC device insertion rates in the medical offices that participated in the educational intervention as compared with usual practice sites. Before the study began, KPNC was divided into six medical service areas that comprised 17 large medical centers and multiple satellite medical offices. Each service area was assigned to either the intervention group or the comparison group based on matching preintervention IUC device insertion rates between the six medical service areas. IUC device insertion rates were obtained for each medical center in all six medical service areas from outpatient service clinical records (OSCRs) for calendar quarters 3 and 4 of 2001. There was a wide variation in IUC device insertion rates in some of the medical centers within and across medical service areas. To control for these outliers, we matched service areas that had high and low insertion rates for them to be equally divided between the intervention group and the control group. It was necessary to keep medical centers within the same service area in the same research group to control for contamination due to greater interaction between clinicians in the same medical service area. Across the KPNC region, the female member population is widely varied. Overall, female members between 20 and 44 years old are primarily employed (89%), have a middle income and are educated (38% with some college education or higher); their approximate distribution by race and ethnicity is as follows: 55% White, 7% African American, 16% Latina, 19% Asian, 1.3% Native American and less than 1% Pacific Islander [19]. Specific demographics by medical center are more difficult to determine. For this study, it was not practical to randomly assign clinicians or facilities within each service area to strict experimental or control grouping because of the existing interaction between clinicians across medical centers within the same geographic service area.

The evidence-based education with academic detailing started in the second quarter of 2002 and ended in December 2002. The study team recruited four enthusiastic physicians who had their clinical practice at one of the intervention sites to be IUC champions. The IUC champions and study investigators developed the evidence-based clinician education program. The study team, the Regional Health Education and the IUC champions developed new or revised existing patient education materials about IUC use that paralleled the evidence-based clinician education component. The patient education materials were proactively placed in the intervention sites but were also made available for purchase to the comparison sites in their usual manner. A KPNC quarterly newsletter mailed to all health plan members in the intervention sites included an article about IUC and featured comments by the local IUC champion. The article clarified common concerns about safety, alerted women that IUC was available at no cost and invited appropriate candidates to discuss IUC use with their clinician. Peer-to-peer clinician education and academic detailing activities were conducted by the IUC champions at the intervention sites, including continuing medical education (CME)-approved grand rounds and IUC device insertion training sessions. The comparison sites were exposed to the usual offsite clinician educational opportunities.

2.1. Preintervention survey

The preintervention survey aimed at measuring baseline knowledge, attitudes and practice patterns regarding IUC use and was administered to all KPNC ob-gyn clinicians (306 physicians and 180 nurse practitioners). The survey developed was based on some of the questions from two published surveys reviewed in the literature [20,21] and additional questions that address experience with use of the levonorgestrel 20-containing (LNG-20) IUC device. The survey was pretested on clinician investigators and the IUC champions who developed and conducted the evidence-based peer-to-peer education. The voluntary survey was sent through interoffice mail in early March 2002 to eligible clinicians in a sealed envelope containing an informed consent cover letter. A second mailing was done, in the same manner, to all nonrespondents 2 weeks later to increase the response rate. Of the 486 eligible providers surveyed, 304 responded, giving a 63% response rate.

2.2. Intervention

A multifaceted intervention was delivered to the three intervention service areas over a 9-month period. The intervention included evidence-based CME-approved education, patient education as well as outreach and peer-to-peer academic detailing (Table 1).

2.3. Postintervention survey

The postintervention survey compared changes in the intervention group and usual care comparison group from

Table 1
Peer-to-peer education and academic detailing activities

Type	Activity	Exposure
Peer-to-peer clinician education	CME-approved grand rounds	10 sessions; 17 facilities; more than 350 clinicians from all departments
	IUC champion consultation service IUC device insertion training sessions	30 IUC device insertion training sessions
Patient outreach	*Member News* features about IUC, featuring IUC champions	1.5 million KPNC members in three service areas exposed to the intervention
Reinforcement: Clinicians and staff	Case presentations; question-and-answer fora; barrier reduction	17 facilities; ob-gyn department meetings
	Proactive distribution of IUC tip sheets	1500 pads of 50 tip sheets in English, Spanish and Chinese
	IUC champion consultation service	Four IUD champions for nine intervention sites
	Clinician/staff incentives: post-it note pads and coffee mugs with slogan	1500 mugs and 3000 post-it note pads ("Give Her the Choice To Change Her Mind, Intrauterine Contraception")
	Final distribution of IUC tip sheet, December 2002	

preintervention to postintervention. The survey was distributed 1 year after the CME-approved education and 7 months after the end of the full intervention to 533 clinicians using the same inclusion and exclusion criteria as those observed for the baseline survey. Questions in the postintervention survey were added or modified from the baseline survey to reflect possible exposure to the intervention. All modifications were approved by the KPNC Institutional Review Board. Of the 533 eligible providers surveyed, 334 responded, giving a 63% response rate.

2.4. Survey analysis

Survey data were analyzed using SAS version 8 software (SAS Institute, Cary, NC). Unless otherwise noted, Pearson χ^2 tests were used to detect statistically significant differences in demographic characteristics among survey respondents and nonrespondents and among the intervention and comparison groups. Pearson χ^2 tests were also used to detect statistically significant differences between the intervention and control groups' responses to selected postsurvey questions. In cases in which the number of observations in survey response categories was very low (i.e., <5), Fisher's exact test was used.

Several questions in the baseline and postintervention surveys used a four-point Likert scale to capture an ordinal range of responses to assess the providers' comfort level with various scenarios around recommending and inserting an IUC device (e.g., 1=*strongly disagree*; 2=*somewhat disagree*; 3=*agree*; 4=*strongly agree*). To compare the changes in the intervention and comparison groups' comfort level over the intervention period with the different scenarios, we calculated the number and percentage of respondents indicating a higher degree of agreement on the postsurvey as compared with the presurvey. Pearson χ^2 tests were used to determine if the number of providers indicating a higher level of comfort in the intervention group was significantly different from that in the comparison group.

2.5. IUC utilization

There were nine medical centers in the intervention group and eight medical centers in the comparison group. Documentation of IUC device insertion data was collected from the third quarter of 2001 through the third quarter of 2003 for each medical center using KPNC OSCRs. Each of the three study periods consisted of three calendar quarters of data: preintervention (third quarter of 2001 to first quarter of 2002); intervention (second quarter of 2002 to fourth quarter of 2002); and postintervention (first quarter of 2003 to third quarter of 2003). For the analysis, the 9-month rate was used because full-year data were not available for each study period.

Rates were determined based on the number of IUC device insertions for each calendar quarter per 1000 women between 15 and 44 years old. The denominator for the rate was based on all female KPNC members between 15 and 44 years old who had been an active member (paid medical coverage) anytime during the quarter for each quarter that data were collected. Assignment of a member to a service area was determined using an algorithm based on medical center utilization, location of the primary care provider and the home zip code, known as the PARFU (population at risk for utilization). The OSCR IUC device insertion code did not distinguish which type of device was inserted.

2.6. IUC utilization rate analysis

To calculate the 9-month rate, we first summed the number of IUC device insertions during the 9-month period and then divided the result by the average number of female health plan members (PARFU) aged between 15 and 44 years during the 9-month period for each medical center. The 9-month rates by study period and by group were then calculated. Independent-samples *t* tests were calculated to see if the IUC device insertion rates (number of IUC device insertions per 1000 women aged between 15 and 44 years)

Table 2
Demographic data on the intervention versus comparison groups and respondents versus nonrespondents

Variable	Intervention [n=114; n (%)]	Comparison [n=98; n (%)]	p	Respondents [n=212; n (%)]	Nonrespondents [n=208; n (%)]	p
Age ≤47 years[a]	53/110 (48)	42/91 (46)	.77	95/201 (47)	82/198 (41)	.24
Female[a]	89/113 (79)	73/92 (79)	.92	162/205 (79)	115/202 (57)	<.001
Race[a]						
White	83/111 (75)	68/91 (75)	.70[b]	151/202 (75)	142/198 (72)	.10[c]
Asian	18/111 (16)	12/91 (13)		30/202 (15)	35/198 (18)	
Hispanic	7/111 (6)	8/91 (9)		15/202 (7)	8/198 (4)	
Black	3/111 (3)	2/91 (2)		5/202 (2)	13/198 (7)	
Native American	0/111 (0)	1/91 (1)		1/202 (<1)	0/198 (0)	
Physicians (vs. nurse practitioners)	70 (61)	56 (57)	.53	126 (59)	144 (69)	.04
Years as licensed practitioner <15	60 (53)	52 (53)	.95	–	–	–

[a] Some observations are missing; the number of providers in each group for whom data are available is cited.
[b] The Black and Native American subjects were not included in the χ^2 analysis due to their low cell counts.
[c] The Native American subjects were not included in the χ^2 analysis due to their low cell counts.

were different in the two study groups. Changes over time were evaluated using paired t tests to see if changes were statistically significant.

3. Results

3.1. Demographics of respondents

There were 420 presurvey and postsurvey questionnaire recipients. Fifty percent (n=212) of the preintervention and postintervention survey questionnaire recipients responded to both surveys. Respondents and nonrespondents were similar in demographic characteristics, except that females were more likely than males to respond (p<.001) and nurse practitioners were more likely than physicians to respond (p=.04) (Table 2). Among the survey respondents, demographic comparisons between the intervention and comparison groups showed no statistically significant difference in age, sex, race, provider type and years licensed.

3.2. Knowledge changes

Both groups reported attendance at IUC-related conferences, departmental meetings about IUC and device insertion training sessions. However, respondents from the

Table 3
Postintervention IUC knowledge and attitude clinician survey responses

	Intervention group [n (%) indicating *yes*]	Comparison group [n (%) indicating *yes*]	p
Knowledge			
Attended KPNC-sponsored CME grand rounds about IUC[a]	33/114 (29)	7/98 (7)	<.001
Increased familiarity with LNG-20 IUC device[b]	32/108 (30)	44/98 (45)	.02
Attitudes			
Positive attitude about Cu-T 380A IUC device currently[a]	91/114 (82)	84/98 (88)	.20
Positive attitude about LNG-20 IUC device currently[a]	102/114 (92)	71/98 (75)	<.001
Improved attitude about Cu-T 380A IUC device from 1 year ago[a]	13/114 (12)	9/98 (9)	.59
Improved attitude about LNG-20 IUC device from 1 year ago[a]	57/114 (51)	32/98 (34)	.02
I am more likely to recommend IUC to women who are considering permanent sterilization as compared with 1 year ago[a]	70/114 (64)	46/98 (49)	.03
Greater comfort in recommending IUC for women with diabetes[b]	36/107 (34)	20/95 (21)	.05
Greater comfort in recommending an LNG-20 IUC device to a woman with a history of menorrhagia instead of endometrial ablation[b]	25/102 (25)	9/92 (10)	.007
Greater comfort in offering an LNG-20 IUC device to a woman with dysmenorrhea[b]	29/105 (28)	12/91 (13)	.01
Greater comfort in inserting an IUC device when a woman is not on her menses as long as pregnancy can be ruled out[b]	27/107 (25)	8/92 (9)	.002
Greater comfort in inserting an LNG IUC device as progesterone replacement or supplementation for perimenopausal or menopausal women[b]	35/101 (35)	13/88 (15)	.002
Greater comfort in inserting an LNG-20 IUC device in a patient with a history of ectopic pregnancy[b]	39/103 (38)	14/85 (16)	.001

[a] Provider responses to questions in the postintervention survey.
[b] Providers indicating higher degree of agreement/comfort in their response to a question at postintervention as compared with their response to the same question at preintervention.

Table 4
Postintervention practice pattern clinician survey responses

Practice pattern	Intervention group [n (%) indicating *yes*]	Comparison group [n (%) indicating *yes*]	p
Likely to recommend IUC for birth control at			
Tubal sterilization consultation	88/106 (83)	62/91 (68)	.01
Routine visit	100/110 (91)	76/95 (80)	.03
Postabortion visit	70/104 (67)	49/95 (52)	.02
I am more likely to recommend IUC to a woman who desires long-term birth control compared with 1 year ago	54/112 (48)	33/96 (34)	.04
I am more likely to do the insertion of an IUC device myself compared with 1 year ago	42/107 (39)	25/95 (26)	.05
Information about the safety and efficacy of IUC I received from KPNC education has affected my frequency of recommending this method			
Not at all	22/112 (20)	37/95 (39)	.01
Somewhat	28/112 (25)	25/95 (26)	
Moderately	23/112 (21)	14/95 (15)	
A great deal	32/112 (29)	13/95 (14)	
No additional information	7/112 (6)	6/95 (6)	
I require a woman to be on her menses for IUC device insertion[a]	18/98 (18)	8/93 (9)	.05
IUC device insertion training I received from KPNC affected the frequency of IUC device insertions I perform			
Not at all	36/109 (33)	45/94 (48)	.047
Somewhat	23/109 (21)	17/94 (18)	
Moderately	29/109 (27)	15/94 (16)	
A great deal	13/109 (12)	5/94 (5)	
No additional training	8/109 (7)	12/94 (13)	
Compared with 1 year ago I have			
More experience inserting the LNG-20 IUC device	85/104 (82)	61/90 (68)	.02
The same amount of experience inserting the LNG-20 IUC device	19/104 (18)	29/90 (32)	
The fact that IUC is now a covered benefit (reduced cost to patient) has affected my frequency of recommending this method			
Not at all	38/112 (34)	46/95 (48)	.12
Somewhat	31/112 (28)	26/95 (27)	
Moderately	24/112 (21)	13/95 (14)	
A great deal	19/112 (17)	10/95 (11)	

Data presented are providers' responses to questions in the postintervention survey unless otherwise indicated.
[a] Providers who changed their response from *yes* in the preintervention survey to *no* in the postintervention survey.

intervention group reported a higher frequency of attending KPNC-developed IUC CME-approved grand rounds as compared with respondents from the comparison group ($p<.001$) (Table 3). Familiarity with the copper T (Cu-T 380A) IUC device was reported as "very high" in both groups at baseline and postintervention. Reported familiarity with the LNG-20 IUC device was higher in the intervention group as compared with the comparison group at baseline ("very familiar": intervention=56% vs. comparison=35%) and remained higher at postintervention ("very familiar": intervention=74% vs. comparison=59%), but the comparison group actually reported greater gains in familiarity with the LNG-20 IUC device as compared with the intervention group ($p=.02$) (Table 3).

3.3. Attitude changes

At baseline, survey respondents from both groups reported a positive attitude toward the Cu-T 380A, which remained positive after the intervention. After the intervention, respondents from the intervention group reported a more positive attitude toward the LNG-20 IUC device as compared with those from the comparison group (Table 3).

At postintervention, the intervention group reported a greater likelihood than the comparison group to recommend IUC to women considering permanent sterilization. The comfort level reported by the intervention group improved significantly more as compared with that reported by the comparison group over the course of the intervention period with respect to recommending IUC to women with diabetes, menorrhagia, dysmenorrhea or a history of ectopic pregnancy or to perimenopausal/menopausal women as progestin replacement/supplement. Neither group reported reluctance in recommending IUC due to a history of pelvic inflammatory disease. Only a small percentage of survey respondents in both groups reported reluctance to recommend IUC to women desiring a future pregnancy or because of medical liability concerns or beliefs that IUC acts as an abortifacient (Table 3).

Table 5
IUC device insertion rates by group and by period

Period	Intervention group (IUC device insertions per 1000 women aged 15–44 years)	Comparison group (IUC device insertions per 1000 women aged 15–44 years)	p
Preintervention	6.31	5.46	.30
Intervention	8.04	6.30	.08
Postintervention	9.57	7.35	.02

Table 6
IUC device insertion rate changes by study group

Group	Preintervention rate per 1000 women aged 15–44 years	Intervention rate per 1000 women aged 15–44 years	Postintervention rate per 1000 women aged 15–44 years	Preintervention to intervention rate change	Intervention to postintervention rate change	Preintervention to postintervention rate change
Intervention	6.31	8.04	9.57	1.73 (p=.0005)	1.53 (p=.002)	3.26 (p<.0001)
Comparison	5.46	6.30	7.35	0.84 (p=.04)	1.05 (p=.005)	1.89 (p=.003)

3.4. Practice pattern changes

The intervention group was more likely than the comparison group to report that the information they had received in 2002 about the safety and effectiveness of IUC had affected their frequency of recommending it and that the IUC device insertion training they received had affected the number of insertions they performed themselves, especially with experience in inserting the LNG-20 IUC device. The intervention group also reported a greater likelihood to recommend IUC for patients who desire a long-term method of birth control at sterilization consults, routine visits and at postabortion visits and to allow IUC device insertion when women were not on their menses (Table 4).

3.5. IUC utilization results

IUC device insertion rates were determined for the intervention and comparison groups for each 9-month study period (Table 5). The preintervention insertion rate was 6.31 per 1000 women for the intervention group and was 5.46 per 1000 women for the comparison group. The differences in preintervention rates are not statistically significant (p=.30) between the two groups. During the intervention period, the IUC device insertion rate increased to 8.04 per 1000 for the intervention group and to 6.30 per 1000 for the comparison group. The difference in rates during the intervention period is not statistically significant between the two groups (p=.08). Finally, the postintervention insertion rate was 9.57 per 1000 for the intervention group and was 7.35 per 1000 for the comparison group. The difference in these two rates is statistically significant (p=.02) (Table 5).

Overall, the IUC device insertion rate increased from 5.91 to 8.53 per 1000 women aged between 15 and 44 years. The intervention and comparison groups showed an upward trend. The increases from preintervention to intervention, intervention to postintervention and preintervention to postintervention were statistically significant for both groups (Table 6).

Rate increases were not significantly different between the intervention and comparison groups from preintervention to intervention (p=.07) and from intervention to postintervention (p=.28); however, the rate increase was significantly different between the two groups from preintervention to postintervention (p=.02). The intervention group demonstrated a rate increase of 3.3 per 1000 women aged between 15 and 44 years, as compared with an increase of 1.9 per 1000 women aged between 15 and 44 years for the comparison group (Table 7).

4. Discussion

4.1. Clinician knowledge, attitudes and practice pattern changes

It was evident from the results of the preintervention survey that general IUC familiarity and knowledge levels were high among the KPNC respondents and that attitudes toward IUC were generally positive. This presented additional challenge to demonstrate an increase in knowledge and more positive attitudes toward IUC. For example, at preintervention, 86% of the intervention group and 90% of the comparison group reported being "very familiar" with the Cu-T 380A IUC device and 93% of the intervention group and 89% of the comparison group indicated that they would be comfortable recommending IUC to a woman younger than 25 years if she were parous and monogamous. Similarly, at preintervention, 81% of the intervention group and 80% of the comparison group agreed with a statement about feeling comfortable with recommending IUC to a woman who had a sexually transmitted infection 2 years before her current monogamous relationship and the birth of her children.

At postintervention, when asked about the frequency of their IUC device insertion, 20% of the comparison group versus 25% of the intervention group reported IUC device insertions being performed more than once per week. This reflects increases of 11% and 12% from the preintervention levels of 9% and 13%, respectively. This change may be a reflection of the removal of copayment, however.

Reported changes in attitudes and practice patterns in the intervention group that were significantly greater in magnitude than those in the comparison group from preintervention to postintervention included an increased frequency of recommending IUC due to information received about the safety and efficacy of IUC, greater likelihood to insert IUC

Table 7
Comparisons of 9-month IUC device insertion rate changes by study group

Nine-month rate changes	Intervention group	Comparison group	p
Preintervention to intervention	1.73	0.84	.07
Intervention to postintervention	1.53	1.05	.28
Preintervention to postintervention	3.26	1.89	.02

devices themselves and greater likelihood to recommend IUC for birth control at routine medical office visits (e.g., annual examination and postabortion), especially for women who desire long-term contraception or permanent sterilization (tubal sterilization consults). The intervention group also demonstrated an increased likelihood over the course of the intervention to insert an IUC device without requiring a woman to be menstruating, to recommend IUC to women with chronic diseases such as diabetes and to use IUC for some of the noncontraceptive benefits of the LNG-20 IUC device. Based on the medical evidence, the intervention group was also less likely to avoid recommending an LNG-20 IUC device to women with a history of ectopic pregnancy as compared with control subjects.

4.2. Did reported changes in attitudes and practice patterns lead to changes in IUC utilization?

IUC utilization increased over the 27-month period in all KPNC sites. This overall trend may be attributed to the removal of cost as a barrier for IUC. The IUC utilization trends in the sites exposed to the peer-to-peer education and academic detailing showed a more consistent upward trend, reaching statistical significance by the postintervention period.

4.3. Peer-to-peer education

The concept of an IUC champion participating in formal clinician education, onsite consultation and academic detailing reinforcement activities in the intervention sites added value to the intervention. The IUC champions reported that they were tapped to assist with difficult insertions and consultations about the use of IUC in nontraditional candidates (e.g., nulliparity, history of pelvic inflammatory disease and previous ectopic pregnancy) and in the use of progestin IUC for noncontraceptive benefits. Although the usual practice comparison sites possibly received the very same evidence about IUC from offsite conferences and meetings sponsored by professional affiliations, academic institutions or industry, the peer-to-peer educational approach employed in this study may have been more effective in changing clinician practice.

4.4. Transitioning research into practice

Despite more than a decade of medical evidence showing the safety and cost-effectiveness of IUC, we hypothesized that a comprehensive approach was needed to supplement the removal of cost as a barrier to promote change in clinician practice. The literature shows that CME alone (passive dissemination) is relatively ineffective in promoting evidence-based changes in practice [5,6]. Strategies that have been shown to facilitate the transition from research to practice include common themes, such as the following:

- strong evidence of the need for change (although safe and cost-effective, IUC is used by <2% of American women of reproductive age);
- appropriate identification of the problem (safety myths prevail about IUC);
- stakeholders and strong opinion leaders (IUC champions and academic detailing);
- comprehensive and multidisciplinary strategies to effect the change and diffusion of information (multifaceted approach to clinician and patient education); and
- strong organizational commitment (removal of cost for IUC as a barrier).

This study employed these strategies and demonstrated an impact on IUC utilization. Multifaceted interventions have demonstrated a higher probability for positive changes in health care outcomes as compared with single or double interventions [5,6].

Removing the cost of IUC for the patient occurred before the onset of the study, affecting the intervention and comparison sites equally. Further studies might investigate the difference in rates of all methods of contraception use between sites with education alone and those with removal of cost alone. Additional studies could also look into whether health plan providers, such as KPNC, that assume the costs for IUC and other cost-effective reversible methods of contraception have reduced unintended pregnancies, improved patient outcomes and led to greater cost savings.

4.5. Study limitations

One limitation of this study was its inability to identify what method of contraception was used by each woman before the insertion of an IUC device during the study period. A second limitation was the use of a 9-month utilization rate rather than an annual rate. A 9-month intervention was feasible, and full-year rate information was not available for the preintervention period. Another limitation of the study was its inability to accurately document the types of IUC devices inserted for comparisons between the Cu-T 380A and the LNG-20. This limitation was due to the documentation of insertion through *ICD-9* procedure codes, which do not account for product type. A fourth limitation of this study was its inability to randomize all medical centers to the intervention or usual care comparison group. There was a wide variation in IUC device insertion rates within each medical service area, but clinicians within a service area would have a greater chance for interaction. This increased the risk for contamination if facilities within each service area were randomized. There is a potential for bias in the results because the sites were not chosen randomly. It was also not in the scope of the study to include follow-up data to track patient satisfaction, complication rates or IUC device removals after insertion.

5. Conclusion

This study demonstrated a comprehensive effort that resulted in a change in reported attitudes and practice

patterns and in a statistically significant increase in IUC utilization in the intervention group within a large health plan organization. It is an example of how a multifaceted approach can rapidly effect change in practice based on medical evidence. Several components of the intervention (IUC champion, IUC clinician education, academic detailing of health education materials and reminder incentives for clinicians and staff) are being replicated in the comparison sites. All materials created in conjunction with this project can be shared with the health care community. (Contact Debbie Postlethwaite through Debbie.a.postlethwaite@kp.org.)

Acknowledgments

We thank Berlex Lab for providing an unrestricted research grant to conduct the study; Kaiser Permanente Women's Health Research Institute, Quality and Operations Support and Division of Research for data collection and analysis; and Felicia Stewart, MD (posthumously), for her critical manuscript review.

References

[1] Porter M, Teisberg EO. Redefining competition in health care. Harv Bus Rev 2004;82:64–76,136.
[2] Berwick DM. Disseminating innovations in health care. JAMA 2003; 289:1969–75.
[3] Dopson S, Fitzgerald L, Ferlie E, et al. No magic targets! Changing clinical practice to become more evidence based. Health Care Manage Rev 2002;27:35–47.
[4] Gruman J, Follock M. Putting evidence into practice. Office of Behavioral and Social Science Research, NIH 1997. Available from: www.obssr.od.nih.gov [accessed 3/6/06].
[5] Davis DA, Thompson MA, Oxman AD, et al. Changing physician performance: a systematic review of the effect of continuing medical education strategies. JAMA 1995;274:700–5.
[6] Bero LA, Grilli R, Grimshaw JM, et al. Getting research findings into practice: closing the gap between research and practice: an overview of systematic reviews of interventions to promote the implementation of research findings. BMJ 1998;317:465–8.
[7] Family Planning Worldwide Data Sheet, 2002. Population Reference Bureau. Available at: www.prb.org [accessed 3/6/06].
[8] Mosher WD, Martinez GM, Chandra A. et al. Use of contraception and use of family planning services in the United States: 1982–2002. Available at: http://www.cdc.gov/nchs/nsfg.htm [accsessed 6/18/06]. Advance data from Vital and Health Statistics, USDHHS, CDC, 350;2004:1–35.
[9] Trussell J, Leveque JA, Koenig JD, et al. The economic value of contraception; a comparison of 15 methods. Am J Public Health 1995; 85:494–503.
[10] Hatcher RA, Trussell J, Stewart F, et al. Contraceptive technology. 18th ed. New York: NY Ardent Media Inc; 2004.
[11] Summary of changes from the first edition of WHO medical eligibility criteria for contraceptive use. Contracep Rept 2001;12:12–4.
[12] Hicks D. What risk of infection with IUD use? Lancet 1998;351: 1222–3.
[13] Darney P. Time to pardon the IUD? N Engl J Med 2001;345:608.
[14] Duenas JL, Albert A, Carrasco F. Intrauterine contraception in nulligravid vs parous women. Contraception 1996;53:23–4.
[15] Farley T, Rosenberg M, Rowe PJ, et al. Intrauterine devices and pelvic inflammatory disease: an international perspective. Lancet 1992;339: 785–8.
[16] Sivin I, Stern J. Health during prolonged use of levonorgestrel 20 μg/d and the copper TCu 380Ag intrauterine contraceptive devices: a multicenter study. Fertil Steril 1994;61:70–7.
[17] Toivonen J, Luukkainen T, Allonen H. Protective effect of intrauterine release of levonorgestrel on pelvic infection: three years' comparative experience of levonorgestrel- and copper-releasing intrauterine devices. Obstet Gynecol 1991;77:261–4.
[18] Forrest JD. U.S. women's perception of and attitudes about the IUD. Obstet Gynecol Surv 1996;51:S30–4.
[19] Gordon NP. Kaiser Permanente Member Health Survey, 2002. http://dor-ent1.kaiser.org/dor/mhsnet/pdf_02/mhs02reg_e.pdf [accessed 6/01/06].
[20] Kooiker CH, Scutchfield FD. Barriers to prescribing the copper T 380A intrauterine device by physicians. West J Med 1990;153:279–82.
[21] Stanwood N, Garrett J, Konrad T. Obstetrician-gynecologists and the intrauterine device: a survey of attitudes and practice. Obstet Gynecol 2002;99:275–80.

Challenges in Translating Evidence to Practice

The Provision of Intrauterine Contraception

Cynthia C. Harper, PhD, Maya Blum, MPH, Heike Thiel de Bocanegra, PhD, MPH, Philip D. Darney, MD, MSc, J. Joseph Speidel, MD, MPH, Michael Policar, MD, and Eleanor A. Drey, MD, EdM

OBJECTIVE: Intrauterine contraception is used by many women worldwide, however, it is rarely used in the United States. Although available at no cost from the state family planning program for low-income women in California, only 1.3% of female patients obtain intrauterine contraceptives annually. This study assessed knowledge and practice patterns of practitioners regarding intrauterine contraception.

METHODS: We conducted a survey among physicians, nurse practitioners, and physician assistants (n=1,246) serving more than 100 contraceptive patients per year in the California State family planning program. The response rate was 65% (N=816). We used multiple logistic regression to measure the association of knowledge with clinical practice among different provider types.

RESULTS: Forty percent of providers did not offer intrauterine contraception to contraceptive patients, and 36% infrequently provided counseling, although 92% thought their patients were receptive to learning about the method. Regression analyses showed younger physicians and those trained in residency were more likely to offer insertions. Fewer than half of clinicians considered nulliparous women (46%) and postabortion women (39%) to be appropriate candidates. Evidence-based views of the types of patients who could be safely provided with intrauterine contraception were associated with more counseling and method provision, as well as with knowledge of bleeding patterns for the levonorgestrel-releasing intrauterine system and copper devices.

CONCLUSION: Prescribing practices reflected the erroneous belief that intrauterine contraceptives are appropriate only for a restricted set of women. The scientific literature shows intrauterine contraceptives can be used safely by many women, including postabortion patients. Results revealed a need for training on updated insertion guidelines and method-specific side effects, including differences between hormonal and nonhormonal devices.

(Obstet Gynecol 2008;111:1359–69)

LEVEL OF EVIDENCE: III

Of the 6 million pregnancies among U.S. women each year, nearly half are unintended, and the rate is increasing among low-income women.[1] Intrauterine contraception is safe and highly effective, with a failure rate of less than 1%.[2,3] However, it is infrequently prescribed by health care providers in the United States. Intrauterine contraception does not depend on individual compliance to be highly effective, but it does depend on health care providers for insertion and removal, and provider practices vary widely. In Europe, intrauterine contraception use ranges from 20–26% in certain countries (France 20%, Norway 24%, Finland 26%). In other parts of the world, use is even higher, for example, Israel (30%), China (34%), Egypt (37%), Korea (49%), and Uzbekistan (52%). In the United States, by contrast, intrauterine contraception use is negligible at about 1%.[4]

Specific–and unfounded–concerns related to intrauterine contraception safety include a correlation

From the Bixby Center for Reproductive Health Research & Policy, Department of Obstetrics, Gynecology and Reproductive Sciences, University of California, San Francisco, California.

Supported by funds from the State of California, Department of Health Services, contract 05-45122, and the William and Flora Hewlett Foundation. All analyses, interpretations, or conclusions are those of the authors and not of the funders.

The authors thank Debbie Postlethwaite, RNP, MPH, for her detailed review of the survey and Debbie Weiss, MPH, for her contributions to the survey and provider interviews.

Corresponding author: Cynthia C. Harper, PhD, Assistant Professor, Department of Obstetrics, Gynecology and Reproductive Sciences, Box 0744 UCSF, 3333 California St., Ste. 335, San Francisco, CA 94118; e-mail: harperc@obgyn.ucsf.edu.

Financial Disclosure
The authors have no potential conflicts of interest to disclose.

ISSN: 0029-7844/08

between intrauterine contraception and ectopic pregnancy,[3] which was disproved in the 1980s,[5] and a heightened risk of pelvic inflammatory disease (PID) and future infertility.[6,7] The Dalkon Shield, a poorly designed and tested device, was associated with an eightfold increase in PID risk and was removed from the market in the 1970s.[7,8] Although these events occurred three decades ago, they continue to influence providers' perceptions of intrauterine contraception, and the fear of litigation has been found to be associated with low intrauterine contraception provision in practice.[9] However, the forms of intrauterine contraception currently on the market do not pose similar risks.[6,8,10,11] In addition, concern that copper intrauterine contraceptives increase the risk of infertility in nulliparous women also has been refuted.[12]

Newly improved devices have been developed and widely used throughout the world; two methods currently on the market in the United States are the levonorgestrel-releasing intrauterine system (Mirena, Bayer HealthCare Pharmaceuticals, Wayne, NJ) and the Copper T 380A (ParaGard, Barr Pharmaceuticals, Montvale, NJ). A limited number of studies have examined barriers that health care providers face in the United States in offering newer devices to women. Barriers to provision generally are related to lack of clinical training and limited knowledge. A national survey of fellows of the American College of Obstetricians and Gynecologists (ACOG) found that 95% of respondents considered the Copper T 380A to be safe. However, nearly one third felt that there was a causal relationship between intrauterine contraception use and PID, and having this belief was significantly associated with lower provision. Although only 16% agreed that intrauterine contraception use leads to litigation, there was also an association with fewer insertions.[9] This study was not able to collect specific data on the levonorgestrel-releasing system, as it had just become available. A more recent training and educational intervention in a Northern California HMO including physicians and nurse practitioners (N=212) showed an increase in positive attitudes about the newer levonorgestrel-releasing intrauterine contraceptive and a greater comfort in recommending it to patients.[13] This intervention is promising and leads to the research question of what knowledge and content is most important for providers to gain to be proficient in intrauterine contraception provision.

This study builds on the new and growing literature in this area by examining the specific method characteristics of each of the available devices, including benefits and side effects. We used multivariable modeling to assess knowledge and practice patterns of physicians and advance practice clinicians.

MATERIALS AND METHODS

As part of an evaluation program of the California State family planning program for low-income populations, Family PACT, we conducted a study on intrauterine contraception practices among health care providers. In this program, 2,834 clinician providers served more than 1.5 million clients in 2003–2004.[14] Women eligible for Family PACT can obtain contraception at no cost, but only 1.3% of female patients received an intrauterine contraceptive in 2003, a level that has not varied since program inception. Claims data showed that fewer than half of providers received reimbursement for any intrauterine contraception-related procedure in 2003.

We collected data in 2006 through a small set of in-depth interviews and then a large-scale self-administered written survey of 1,246 clinicians, including physicians, nurse practitioners and physician assistants. Responses to the in-depth interviews generated questions for the survey, and we also included survey items from published provider surveys on intrauterine contraception.[9,13] The survey was pretested among clinician researchers. Two weeks before the initial survey mailing, a letter was sent to prospective participants to inform them of the survey. The survey was mailed with a cover letter and instructions that the survey was to be completed by a physician, nurse practitioner, or physician assistant who offers contraceptive services. Providers then were mailed a reminder postcard later that week, and a second survey was mailed to nonrespondents 4 weeks later. Providers were telephoned a maximum of four times, and data collection ended 12 weeks after the initial mailing. The survey was conducted on the entire population of Family PACT providers, serving more than 100 female contraceptive patients per year. A Family PACT provider refers to a public or private health care facility where contraceptive services are provided by clinicians.

The survey had 816 respondents, including 399 physicians and 402 nonphysicians, for a response rate of 65%. The number of respondents was more than sufficient to show a difference in proportion of physician and nonphysician intrauterine contraception insertions of .65 and .55, respectively, with alpha equal to 0.05 and power of 80% with a two-tailed test. The regression models adjusting for covariates require a slightly larger size to account for correlation between covariates, although we still have 80% power

for a one-tailed test with a squared multiple correlation coefficient of the predictor provider type compared with other covariates to be up to 0.2. The study was approved by the University of California, San Francisco Committee on Human Research.

The survey included items on demographic (age, gender, race/ethnicity), professional (physician, nurse practitioner, or physician assistant, specialty), and practice characteristics (number of female contraceptive patients per year, urban or rural location, public or private practice). Claims data were used for the practice characteristic variables. For items directly related to intrauterine contraception, the survey included training during residency or core training (number of insertions), counseling (frequency of counseling and content), provider views of the safety and risks of the intrauterine contraceptive, provider perceptions about which women make suitable intrauterine contraception candidates, requirements and protocols for insertions, knowledge of appropriate practices for intrauterine contraception in general and specifically by intrauterine contraceptive type–the Copper T 380A or the levonorgestrel-releasing system–and availability of intrauterine contraception in each clinician's practice.

To assess providers' knowledge of correct practices and basic method characteristics, including benefits and side effects, we used a series of scale variables. These variables measure knowledge of bleeding patterns, knowledge of hormonal side effects, and general knowledge. All scales were created using Cronbach's alpha to assess scale reliability and with tests for validity with associations with professional and practice variables as well as outcome variables. The knowledge of bleeding patterns scale was created from 10 survey items that asked about the levonorgestrel-releasing intrauterine system and the Copper T 380A separately. The items in the bleeding scale on the levonorgestrel-releasing intrauterine system included appropriate counseling about the system for patients with dysmenorrhea, patients with menorrhagia, and patients with iron-deficiency anemia, with an emphasis in counseling on spotting, amenorrhea, and irregular bleeding. The items for Copper T 380A were emphasis in counseling for copper intrauterine contraception on menorrhagia, dysmenorrhea, irregular bleeding, and anemia. The scale reliability coefficient for the provider practices for bleeding was 0.80.

The scale of hormonal implications included nine items on the accuracy of the counseling providers give to their patients on the effect of the levonorgestrel-releasing intrauterine system on breast tenderness, headaches, mood changes, acne, and smoking. The scale also captures the accuracy of what providers tell their patients about Copper T 380A for headaches, mood changes, acne, and breast tenderness. Cronbach's alpha gave a scale reliability coefficient of 0.87. For the general knowledge variable, we included 12 items that contributed to a cohesive scale: accurate counseling information on the levonorgestrel-releasing intrauterine system on spotting, amenorrhea, irregular bleeding, pain with intercourse, headaches, mood changes, and acne; and accurate Copper T 380A counseling on menorrhagia, dysmenorrhea, irregular bleeding, anemia, and pain with intercourse. The scale reliability coefficient is 0.86.

For provider views on which women are suitable intrauterine contraception candidates, we asked about consideration of the following nine items: nulliparity, immediate postpartum usage, immediate postabortion usage, teenager, history of ectopic pregnancy, STD in the past 2 years, PID in the past 5 years, current bacterial vaginosis, and human immunodeficiency virus (HIV) positivity. The scale reliability coefficient, measured by Cronbach's alpha, for potential intrauterine contraception candidates was 0.77.

We created a scale variable with provider concerns about risks that affect their willingness to recommend intrauterine contraception with these seven items: sexually transmitted diseases, PID, infertility, ectopic pregnancy, expulsion, uterine perforation at insertions, and other risks Cronbach's alpha gave a scale reliability coefficient of 0.88 for provider concern of risks.

The two outcome variables we assessed on intrauterine contraception provision were counseling contraceptive patients and availability of method at the provider's practice (n=812). Of the 816 survey respondents, we limited analyses to the 812 respondents with data on whether intrauterine contraception was provided at their practices. For analysis, the counseling variable was coded dichotomously to measure a general practice of usually counseling (always/mostly) or not (sometimes/rarely). We performed multiple logistic regression analysis to measure the association of demographic, professional, and practice factors with the outcome variables of provision of counseling and insertions. The models included demographic (age, gender), professional (clinician type, specialty, level of intrauterine contraception training), and practice (private/public, patient volume, urban/rural) characteristics. Additional models included provider perceptions of safety and risk, perceptions about which women make suitable intrauterine contraception candidates, and provider knowledge of the method. The variables that were significant in univariable analyses for either the counseling or provision outcome were included in the multivariable models, as

well as variables shown in the literature to be associated with intrauterine contraception counseling and provision. Data were analyzed with Stata 8.2 (StataCorp LP, College Station, TX) and significance levels reported at $P \leq .05$.

RESULTS

Table 1 presents demographic, professional, and practice characteristics of respondents. Almost half of respondents were physicians (49%), 36% were nurse practitioners, and 15% were physician assistants. By specialty, they were largely family practice (37%) and ob-gyn (35%), although there were also women's health specialists (12%) and general practice (8%). Within the ob-gyn specialty, 54% were physicians and 46% advance practice clinicians. Within the family practice specialty, 46% were physicians and 54% advance practice clinicians. However, in the women's health specialty, almost all (98%) were advance practice clinicians. More than half of providers were in private practice (56%), and the rest were in public/nonprofit. Patient volume for practice ranged from 100 to more than 10,000 female contraceptive patients per year, with a mean number of female contraceptive patients of 1,113 per year. The mean age of the providers was 49 years (standard deviation 10.5), and the sample was diverse, with 47% white, 21% Asian, 20% Latino, 6% African American. In comparing claims data on the respondents and nonrespondents, we found no differences between respondents and nonrespondents by provider type, urban area, contraceptive patients, or intrauterine contraception patients served. The respondents are similar to the full population surveyed: 56% of respondents were in private practice compared with 57% in the population surveyed; 80% in urban areas compared with 82%; 1.5% intrauterine contraception clients compared with 1.5% intrauterine contraception clients; 1,113 mean female contraceptive clients per year compared with 1,027 mean clients.

Sixty-nine percent of these contraceptive providers had received training in intrauterine contraceptive insertions during residency or their core training, although 44% reported inserting fewer than 20 intrauterine contraceptives in training. Among ob-gyn physicians, only 4% were not trained; but among other physicians, 32% were not trained, and among the mid-level practitioners, 41% were not trained. Younger age is significantly associated with a higher level of training (*t* test; $P = .006$). Although most ob-gyn physicians had received training, only 74% of them provided intrauterine contraception at their practices, as did 43% of other physicians. Clinicians reported substantial intrauterine contraception provision by mid-level practitioners in practices where the method was available (nurse practitioner 59%, physician assistant 32%), although the greatest proportion was by physicians (81%). Thirty-six percent of contraceptive

Table 1. Respondent Characteristics and Intrauterine Contraception Practices

Demographic		
Age, mean y (SD)	48.6	10.5
Race/ethnicity, n (%)		
White	373	46.8
Latino	161	20.2
Asian/Pacific Islander	166	20.8
African American	48	6.0
Multi-racial/other	49	6.1
Professional		
Professional title, n (%)		
Physician	389	49.4
Nurse practitioner	118	15.0
Physician assistant	281	35.7
Specialty, n (%)		
Obstetrician-gynecologist	285	35.1
Family practice	302	37.2
Internal medicine, pediatrics, adolescent medicine	47	5.8
Women's health	101	12.4
General practice/other	77	9.5
Trained in IUC insertions n (%)	548	69.0
Practice		
Private practice (vs public), n (%)	450	55.6
Urban, n (%)	644	79.6
Number female contraceptive patients per year, n (%)		
100–500 patients	389	47.8
501–1,000 patients	185	22.8
More than 1,000 patients	239	29.4
IUC counseling		
Frequently discuss IUC with patients seeking contraception	515	64.1
Sufficient time to counsel patients on contraceptive options	680	85.3
Patients receptive to learning about IUC	730	91.9
Enough experience to counsel patients on Copper T 380A	657	82.0
Enough experience to counsel patients on LNG system	590	74.0
IUC provision		
IUCs available at practice	495	61.0
Practitioners inserting IUC at your practice		
Physicians	342	67
Nurse-practitioners	291	59
Physician assistants	158	32
Very comfortable in inserting Copper T 380A	476	59.8
Very comfortable in inserting LNG system	310	39.5
Offered IUC in past 5 years but stopped (n=305)	106	34.7

SD, standard deviation; IUC, intrauterine contraception; LNG, levonorgestrel-releasing intrauterine system.
Not all numbers add up to 812 due to missing data on individual items.

providers counseled their patients infrequently on the method, although 85% reported that they had sufficient time to counsel patients on contraceptive options, and a full 92% considered their patients to be receptive to learning about intrauterine contraception. The majority of providers (55%) considered fewer than one quarter of their contraceptive patients to be intrauterine contraception candidates.

Of the providers offering intrauterine contraception at their practices, most (72%) had both the Copper T 380A and the levonorgestrel-releasing system available. Twenty-three percent of providers with intrauterine contraception offered just the Copper T 380, and 5% offered just the levonorgestrel system. Of the practices not offering intrauterine contraception to contraceptive patients, the main reasons cited included inadequate reimbursement (47%), lack of training (40%), low patient interest (32%), and concerns about procedure risks (28%). Twenty-three percent cited litigation concerns, and only 10% of providers cited few intrauterine contraception candidates as a reason for ceasing to offer services. Thirty-five percent of practices not offering intrauterine contraception had offered it in the previous 5 years but then stopped.

Provider knowledge as well as providers' perceptions of the safety and specific risks involved in intrauterine contraception are presented in Table 2. Almost all clinicians agreed that intrauterine contraception is safe (94%). However, they had many concerns that kept them from recommending intrauterine contraception to their patients. Sexually transmitted diseases and PID were top concerns affecting the willingness of providers to recommend intrauterine contraception, followed by ectopic pregnancy. Providers also showed extremely restrictive views of the women they were willing to consider intrauterine contraception candidates. Fewer than half of providers considered nulliparous, postpartum (immediate), postabortion (immediate), teenagers, history of ectopic pregnancy, PID, or HIV-positive women as candidates for intrauterine contraception, contrary to the World Health Organization Medical Eligibility Criteria.[15]

Basic knowledge about intrauterine contraception was inadequate. Roughly 20% of providers emphasized hormonal side effects, such as mood change, headache, acne, and breast tenderness, when counseling patients about ParaGard®—a copper T device that contains no hormones. Providers also were confused about the hormone content in the levonorgestrel-releasing system; the proportion who would insert a levonorgestrel-releasing intrauterine system for a patient who smoked was only 34%, but levonorgestrel is not contraindicated for smokers.[15] Contraceptive pro-

Table 2. Provider Attitudes and Knowledge

	n	%
Provider perceptions of IUC safety		
Agrees IUC is safe	759	94.2
Concerns affecting IUC recommendation scale (scale reliability coefficient 0.88)		
Sexually transmitted diseases	219	27.8
Pelvic inflammatory disease	227	28.7
Ectopic pregnancy	144	18.2
Infertility	75	9.5
Expulsion	86	10.9
Uterine perforation at insertion	81	10.3
IUC candidate scale (scale reliability coefficient 0.76)		
Nulliparous	362	45.9
Immediately postpartum	263	33.1
Immediately postabortion	313	39.4
Teenager	311	39.1
History of ectopic pregnancy	247	31.1
Sexually transmitted disease in past 2 years	486	61.1
PID in past 5 years	383	48.4
Current bacterial vaginosis	337	42.2
HIV positive	340	42.9
Knowledge of hormonal side effects scale (scale reliability coefficient 0.87)		
LNG system		
Willing to insert for menorrhagic patients who smoke	264	33.7
Breast tenderness	362	48.5
Headache	381	50.8
Mood changes	396	53.0
Acne	324	43.4
Copper T 380A		
Headaches	561	72.0
Mood changes	599	77.1
Acne	639	82.1
Breast tenderness	604	77.6
General IUC knowledge scale (scale reliability coefficient 0.86)		
LNG system		
Appropriate emphasis in counseling on		
Spotting	533	70.4
Amenorrhea	595	79.2
Irregular bleeding	629	83.4
Pain with intercourse	407	54.3
Headache	381	50.8
Mood change	396	53.0
Acne	324	43.4
Copper T 380A		
Appropriate emphasis in counseling on		
Anemia	555	71.0
Irregular bleeding	400	51.5
Dysmenorrhea	551	70.6
Menorrhagia	612	78.5
Pain with intercourse	461	59.3

IUC, intrauterine contraception; PID, pelvic inflammatory disease; HIV, human immunodeficiency virus; LNG, levonorgestrel-releasing intrauterine system.

viders in general are not yet fully informed about the benefits of the levonorgestrel-releasing system, nor of all of the important implications on bleeding patterns of both devices. Only 33% of providers had ever recommended the levonorgestrel-releasing intrauterine system for noncontraceptive benefits. Only 39% reported they would insert a levonorgestrel-releasing intrauterine system for a patient with dysmenorrhea if she were interested, and half (51%) for a woman with menorrhagia. Only 45% of providers responded they would insert a levonorgestrel-releasing intrauterine system in an iron-deficient anemic patient if she were interested. Just as bleeding patterns were often forgotten as a potential benefit of the levonorgestrel-releasing intrauterine system, they were also omitted in counseling on the Copper T 380, but in this case as a possible drawback: when discussing the Copper T 380 with patients, more than 25% of providers did not emphasize dysmenorrhea or menorrhagia, which can occur.

In other areas of general knowledge, 25% of the contraceptive providers responded erroneously that antibiotics should be given routinely at the time of intrauterine contraceptive insertion to prevent infection. A similar proportion (24%) responded that a woman with diabetes should not have an intrauterine contraceptive, although it is an appropriate method for this population.[15] Providers were unlikely to mention the Copper T 380A for use as emergency contraception; 85% had never mentioned it to patients.

Multivariable analyses of the factors associated with counseling contraceptive patients about intrauterine contraception are presented in Table 3 . The first model, with socio-demographic, professional, and practice characteristics, shows that there is no difference between physicians and advance practice clinicians (ie, nurse practitioners and physicians assistants) in the frequency of intrauterine contraception counseling. However, practitioners in ob-gyn prac-

Table 3. Counseling on Intrauterine Contraceptives to Female Contraceptive Patients: Multivariable Logistic Regression Results

	Model 1		Model 2		Model 3	
Frequently Counsel Patients on IUC	Odds Ratio	95% CI	Odds Ratio	95% CI	Odds Ratio	95% CI
Demographic						
Age (y)	0.96	0.98–1.01	1.00	0.98–1.02	1.00	0.98–1.02
Gender						
Male (reference)						
Female	1.50*	1.01–2.21	1.28	0.84–1.95	1.26	0.83–1.92
Professional and practice						
Title						
Mid-level NP/PA (reference)						
Physician	1.15	0.76–1.72	1.22	0.79–1.89	1.18	0.76–1.81
Specialty						
Family practice (reference)						
Ob-gyn	1.66†	1.13–2.46	1.51*	1.00–2.28	1.45	0.95–2.21
Women's health	0.96	0.56–1.63	0.83	0.48–1.44	0.81	0.46–1.41
Other (pediatrics/adolescent, GP, internist)	0.64	0.40–1.01	0.83	0.50–1.39	0.84	0.50–1.39
Trained in IUC insertions	1.60†	1.13–2.25	1.52*	1.05–2.20	1.51*	1.05–2.17
Female contraceptive patients (#/y)	0.98*	0.97–1.00	0.98‡	0.96–0.99	0.98‡	0.96–0.99
Provider type						
Public (reference)						
Private	0.70	0.47–1.04	0.84	0.55–1.29	0.87*	0.56–1.33
Urban location	1.24	0.82–1.87	1.05	0.67–1.65	1.08	0.69–1.69
Perceptions and knowledge of IUC						
Consider IUC to be safe	–	–	6.19‡	2.68–14.3	5.68‡	2.56–12.6
Low perception of risks	–	–	1.21	0.96–1.54	1.17	0.93–1.48
Expansive view of IUC candidates	–	–	1.89‡	1.36–2.64	1.85‡	1.32–2.59
High-level knowledge	–	–	1.59†	1.21–2.08	–	–
Knowledge of bleeding patterns	–	–	–	–	1.45*	1.06–1.98
Knowledge of hormonal side effects	–	–	–	–	0.84	0.66–1.08
Number of observations	801		783		792	
Likelihood ratio χ^2 (degrees of freedom)	46.5 (14)		112 (19)		111 (20)	

IUC, intrauterine contraception; CI, confidence interval; NP, nurse practitioner; PA, physician assistant; GP, general practitioner.
* $P \leq .050$.
† $P \leq .010$.
‡ $P \leq .001$.

tices, whether physicians or advance practice clinicians, are significantly more likely to counsel contraceptive patients on the intrauterine contraception than clinicians in other specialties (odds ratio [OR]=1.7). Likewise, healthcare providers who have received training in intrauterine contraception insertion are 1.6 times as likely to counsel patients, but as the number of female contraceptive patients increases, the frequency of intrauterine contraception counseling declines, perhaps due to time constraints. We assessed the contribution of providers' perceptions and knowledge of intrauterine contraception to their likelihood of counseling in Model 2, and we found that those who consider intrauterine contraception to be a safe method have far higher odds (OR=6.2) of counseling their patients frequently about the method than those who do not consider it to be safe. Providers who consider many different types of women eligible for intrauterine contraception and those with high knowledge levels of basic method characteristics and contraindications are also more likely to counsel frequently on the method. However, providers' reports of how concerned they are about the potential risks of insertion (eg, expulsions, perforation) were not associated with counseling. In the final model tested (Model 3), we included specific knowledge scales for whether providers were familiar with bleeding patterns of each device and with the hormonal side effects, and we found better knowledge of bleeding patterns to be associated with frequent counseling.

The multivariable logistic results of provision of the method in the practice showed associations with somewhat different factors than with counseling (Table 4). Model 1, with the socio-demographic, professional, and practice characteristics, showed that younger providers are significantly more likely to offer the method than

Table 4. Provision of Intrauterine Contraceptives to Female Contraceptive Patients: Multivariable Logistic Regression Results

	Model 1		Model 2		Model 3	
Provide IUC in Practice	Odds Ratio	95% CI	Odds Ratio	95% CI	Odds Ratio	95% CI
Demographic						
Age (y)	0.96*	0.94–0.98	0.97†	0.95–0.99	0.97†	0.95–0.99
Gender						
Male (reference)						
Female	1.29	0.82–2.03	1.09	0.68–1.76	0.90	0.55–1.47
Professional and practice						
Title						
Mid-level NP/PA (reference)						
Physician	2.26*	1.40–3.64	2.45*	1.48–4.05	2.53*	1.51–4.23
Specialty						
Family practice (reference)						
Ob-gyn	4.67*	2.98–7.32	4.52*	2.82–7.24	3.31	2.03–5.39
Women's health	3.85*	1.90–7.82	3.79*	1.81–7.97	2.64‡	1.25–5.56
Other (pediatrics/adolescent, GP, internist)	0.36*	0.20–0.62	0.45†	0.25–0.83	0.45‡	0.24–0.83
Trained in IUC insertions	1.82†	1.21–2.74	1.66‡	1.08–2.56	1.44	0.93–2.25
Female contraceptive patients (#/y)	1.05*	1.02–1.07	1.03†	1.01–1.06	1.04*	1.01–1.05
Provider type						
Public (reference)						
Private	0.18*	0.11–0.28	0.16*	0.10–0.27	0.20*	0.12–0.33
Urban location	1.43	0.43–1.13	0.58	0.34–0.99	0.56	0.32–0.97
Perceptions and knowledge of IUC						
Consider IUC to be safe	–	–	5.57*	2.10–14.8	3.36‡	1.29–8.74
Low perception of risks	–	–	1.08	0.82–1.41	0.96	0.73–1.27
Expansive view of IUC candidates	–	–	1.63†	1.13–2.35	1.40	0.96–2.05
High-level knowledge	–	–	1.68†	1.23–2.31		
Knowledge of bleeding patterns	–	–	–	–	3.24*	2.23–4.70
Knowledge of hormonal side effects	–	–	–	–	1.53†	1.15–2.03
Number of observations	809		785		794	
Likelihood ratio χ^2 (degree of freedom)	298.2 (14)		324 (19)		360 (20)	

IUC, intrauterine contraception; CI, confidence interval; NP, nurse practitioner; PA, physician assistant; GP, general practitioner.
* $P \leq .001$.
† $P \leq .010$.
‡ $P \leq .050$.

older providers and that physicians have elevated odds (OR=2.3) of offering intrauterine contraception compared with advance practice clinicians. Ob-gyn specialists, whether physicians or advance practice clinicians, are 4.6 times as likely as family practice clinicians to offer intrauterine contraception, and women's health specialists are 3.8 times as likely. Because these multivariable models are additive, the elevated odds for both physicians and ob-gyn specialists show that ob-gyn physicians have the greatest odds of providing intrauterine contraception. Providers in private practice had greatly reduced odds for offering intrauterine contraception to their patients (OR=0.18). Whereas clinicians in practices with greater numbers of contraceptive patients were less likely to counsel on intrauterine contraception, they were significantly more likely to offer the method to their patients at their practices. As with counseling, training is also significantly associated with increased odds of intrauterine contraception provision (OR=1.8).

In Model 2, we assessed how much of the elevated odds of ob-gyns and other physicians providing intrauterine contraception resulted from their more favorable perceptions or more informed practices. Results showed that the differences in age as well as the differences in professional and practice characteristics remain important, as do intrauterine contraception-specific attitudes and knowledge. As with the counseling results, the intrauterine contraception provision results show that considering the method to be safe was a key factor in its availability at the practice (OR=5.6), and that expansive views of who might be considered as an intrauterine contraception candidate as well as knowledge about each method were also significantly associated with method provision. We assessed the different aspects of knowledge in Model 3 and found that knowledge of bleeding patterns, as well as of hormonal side effects, were strongly associated with provision of the method. We also found that the impact of training was reduced, showing that much of the significant effect of training (seen in Models 1 and 2) lies in the ability to provide patients with accurate information and care about the bleeding they might experience with each method.

Because the physicians differed significantly from the nonphysicians in provision of intrauterine contraception, we also estimated the models separately for both groups and found that the results for physicians were the same as the results for all respondents. For the mid-level practitioners, the estimated coefficients were in the same direction, but a few variables lost strength and did not reach significance level, namely age and training. The specialty of the practice for mid-level practitioners retained a robust association with intrauterine contraception provision and is perhaps more important than core training for nurse practitioners and physician assistants.

DISCUSSION

Prescribing practices of providers in the United States reflect erroneous beliefs that intrauterine contraception is appropriate for an extremely limited segment of women seeking contraception. Our results showed that contraceptive providers who are open to the possibility that the method can be used by many different types of women are more likely to counsel their patients on intrauterine contraception and to have it available in their practices. Recent research has shown that the levonorgestrel-releasing intrauterine system can be used for nulliparous women, although clinicians do not generally provide it and the patient information on the product recommends that the woman have a child.[9,16,17] This study showed that fewer than half of providers would offer intrauterine contraception to nulliparous women, although ACOG has concluded that the method is appropriate for this population as long as the patient is at low risk for sexually transmitted diseases.[3] Likewise, nearly 70% believed that women with previous ectopic pregnancies were not eligible for intrauterine contraception, also contrary to ACOG and WHO recommendations.[3,15]

Only one third viewed immediate postpartum patients as potential intrauterine contraception candidates. Immediate postpartum insertions are done within 48 hours of delivery but usually directly after the placenta is out. Providers have been reluctant to insert intrauterine contraception in postpartum women for fears of increased risk of perforation or expulsion. However, women are often highly motivated for contraception at the time of birth, and the discomfort of insertion is diminished postpartum. Research has documented unintended pregnancies during the waiting period for interim insertions, showing that up to 40% of women requesting intrauterine contraception are lost in this period, sometimes because providers discourage them from intrauterine contraceptive use.[18,19] Postplacental insertion, that is within 10 minutes of placental expulsion, has been shown to be safe and acceptable to women and is a promising area for future research and practice.[20] In this study, only 39% of providers considered postabortion patients to be intrauterine contraception candidates, although the demonstrated need for contraception at that time is clear; half of the women in the United States having abortions are having repeat abortions.[21] A large World Health Organization study of

immediate post first trimester abortion intrauterine contraception insertions showed no increased risk of infection and low expulsion rates, and a Cochrane review of postabortion insertions found them to be safe and practical.[22,23]

Research also has indicated that even women at risk of infections and those who are already HIV positive may be able to benefit from intrauterine contraception, although most providers would not consider them as candidates. A small body of research has begun to inquire about the use of intrauterine contraception in HIV-positive women, even in developing country settings, and has found promising results.[24–27] One study found that providers in Africa (Zimbabwe) might be open to the use of intrauterine contraception for women at high risk of HIV,[28] although training efforts and other interventions to increase insertions have not yet been launched. Further intrauterine contraception research is needed to better understand client eligibility, particularly among immediate postcesarean or vaginal delivery and post second trimester abortion patients, and use by HIV-infected women.

A limitation with these results is that they may not apply to all contraceptive clinicians in the publicly funded program, because survey respondents could have different approaches to intrauterine contraception provision than nonrespondents (although they did have similar numbers of intrauterine contraception clients). Another limitation of this study is that the data were collected from a survey administered at one point in time, so that we cannot measure causality, only association. It is likely that causality does not only go in one direction; that is, the providers who are able to offer accurate counseling and evidence-based services are those who have more experience. However, the one survey item that does point to a temporal effect is training in insertions during residency and subsequently increased provision in practice. Whereas this study showed ob-gyn physicians were likely to be trained, family practice physicians and advance practice clinicians had far less training. A promising note was that younger physicians were more likely to be trained and to offer intrauterine contraception at their practices.

Training, not only in insertions but in basic method characteristics, is necessary to improve intrauterine contraception services. Incorrect knowledge about method benefits, contraindications, side effects, and appropriate candidates may deter providers from recommending the method to patients or may cause them to give faulty information. Although more than 90% of these providers thought their patients were receptive to learning about intrauterine contraception, far fewer had integrated it into their contraceptive services. As with providers, misconceptions about intrauterine contraception limit acceptance by patients.[29,30] Results show that even among these high-volume contraceptive providers, many were not familiar with the overall decrease in blood loss and improved dysmenorrhea associated with the levonorgestrel-releasing intrauterine system. Similarly, a significant minority did not emphasize the increased bleeding that can occur with the Copper T 380A. Among U.S. women who do choose intrauterine contraception, a main reason for discontinuation is increased menstrual bleeding.[29] Accurate information about the bleeding patterns associated with the different intrauterine contraceptives would help providers improve their recommendations and would also help women in their selections.

Improved intrauterine contraception provision requires medical education as well as training.[13,31] Specifically, provider education should involve evidence-based guidelines that emphasize safety and insertion techniques and should include not only ob-gyns and women's health care providers, but all providers offering family planning counseling and services.[32–34] In addition, less restrictive, evidence-based criteria for intrauterine contraception candidate selection should be developed and promoted.[9] Patient counseling should ensure proper knowledge and expectations of the method to increase adherence.[35] Clinical training is also necessary, especially to alleviate concerns about perforations from postpartum insertions or expulsions from incorrect placement in postabortion insertions.[19,22] Results from an intervention to use a checklist with the new medical eligibility criteria of the World Health Organization showed that a checklist was not sufficient to change providers' reliance on outdated knowledge about the intrauterine contraception.[36] A randomized trial likewise showed that provider education, without hands-on training, was insufficient to change practice.[37]

Finally, the issue of insurance coverage and reimbursements is a large obstacle for those health care providers who were actually trained and had experience offering intrauterine contraception but then stopped. Coverage of all contraceptive methods is a health policy need in the United States, but particularly for those methods that are expensive to pay out-of-pocket but confer many years of protection. The abortion rate in the United States is approximately three times higher than that of Western European countries. To prevent unintended pregnancy,

improved use of effective contraception is needed. Intrauterine contraception is an extremely effective and safe method that is far underutilized. It is also the most cost-effective method of reversible contraception.[38] Unfortunately, our study showed health care provider knowledge and practices continue to reflect erroneous views and unrealistic risk perceptions; current practice does not reflect the body of scientific evidence. By addressing these deficiencies in provider perceptions and practices, we can offer women in the United States greater protection against unintended pregnancy, similar to that of women in other industrialized countries where intrauterine contraception use is more frequent.

REFERENCES

1. Finer LB, Henshaw SK. Disparities in rates of unintended pregnancy in the United States, 1994 and 2001. Perspect Sex Reprod Health 2006;38:90–6.
2. Trussell J. The essentials of contraception: efficacy, safety, and personal considerations. In: Hatcher RA, Trussell J, Nelson AL, Cates C, Guest F, Kowal D, eds. Contraceptive Technology. 18 ed. New York (NY): Ardent Media; 2004:221–52.
3. Clinical management guidelines for obstetrician-gynecologists. Intrauterine device. ACOG practice bulletin No. 59. American College of Obstetricians and Gynecologists. *Obstet Gynecol* 2005;105:223–32.
4. United Nations Department of Economic and Social Affairs Population Division. World contraceptive use 2005. Available at: http://www.un.org/esa/population/publications/contraceptive2005/2005_World_Contraceptive_files/WallChart_WCU2005.pdf. Retrieved June 19, 2007.
5. Sivin I. IUDs and ectopic pregnancy [French]. Contracept Fertil Sex (Paris) 1983 Dec;11:1287–94.
6. Grimes D. Intrauterine device and upper-genital-tract infection. Lancet 2000;356:1013–9.
7. Kimble-Haas SL. The intrauterine device: dispelling the myths. Nurse Pract 1998;23:58, 63–9, 73.
8. Darney PD. Time to pardon the IUD? N Engl J Med 2001; 345:608–10.
9. Stanwood NL, Garrett JM, Konrad TR. Obstetrician-gynecologists and the intrauterine device: a survey of attitudes and practice. Obstet Gynecol 2002;99:275–80.
10. Lee NC, Rubin GL, Ory HW, Burkman RT. Type of intrauterine device and the risk of pelvic inflammatory disease. Obstet Gynecol 1983;62:1–6.
11. Shelton JD. Risk of clinical pelvic inflammatory disease attributable to an intrauterine device. Lancet 2001;357:443.
12. Hubacher D, Lara-Ricalde R, Taylor DJ, Guerra-Infante F, Guzman-Rodriguez R. Use of copper intrauterine devices and the risk of tubal infertility among nulligravid women. N Engl J Med 2001; 345:561–7.
13. Postlethwaite D, Shaber R, Mancuso V, Flores J, Armstrong MA. Intrauterine contraception: evaluation of clinician practice patterns in Kaiser Permanente Northern California. Contraception 2007;75:177–84.
14. UCSF Bixby Center for Reproductive Health Research & Policy. Family PACT Program Report Fiscal Year 03/04. San Francisco, CA, 2005. Available at http://www.familypract.org/_resources/documents/0203.pdf. Retrieved April 8, 2008.
15. World Health Organization. Medical eligibility criteria. Geneva: WHO; 2004.
16. Prager S, Darney PD. The levonorgestrel intrauterine system in nulliparous women. Contraception 2007;75:S12–5.
17. Suhonen S, Haukkamaa M, Jakobsson T, Rauramo I. Clinical performance of a levonorgestrel-releasing intrauterine system and oral contraceptives in young nulliparous women: a comparative study. Contraception 2004;69:407–12.
18. Ogburn J, Espey E, Stonehocker J. Barriers to intrauterine device insertion in postpartum women. Contraception 72; 2005:426–9.
19. Grimes D, Schulz K, Van Vliet H, Stanwood N, Lopez L. Immediate post-partum insertion if intrauterine devices. Cochrane Database of Systematic Reviews 2001; Art. No.: CD003036. DOI: 10.1002/14651858.CD003036.
20. Celen S, Möröy P, Sucak A, Aktulay A, Danisman N. Clinical outcomes of early postplacental insertion of intrauterine contraceptive devices. Contraception 2004;69:279–82.
21. Jones R, Singh S, Finer L, Forhwirth L. Repeat abortion in the United States: Guttmacher Institute; 2006. Available at http://www.guttmacher.org/pubs/2006/11/21/or29.pdf. Retrieved April 4, 2008.
22. Grimes D, Lopez L, Schulz K, Stanwood N. Immediate postabortal insertion of intrauterine devices. Cochrane Database of Systematic Reviews 2004; Art. No.: CD001777. DOI: 10.1002/14651858.CD01777.pub2.
23. Li CF, Lee SS, Pun TC. A pilot study on the acceptability of levonorgestrel-releasing intrauterine device by young, single, nulliparous Chinese females following surgical abortion. Contraception 2004;69:247–50.
24. Richardson BA, Morrison CS, Sekadde-Kigondu C, Sinei SK, Overbaugh J, Panteleeff DD, et al. Effect of intrauterine device use on cervical shedding in HIV-1 DNA. AIDS 1999;13:2091–7.
25. Morrison CS, Sekadde-Kigondu C, Sinei SK, Weiner DH, Kwok C, Dokonya D. Is the intrauterine device appropriate contraception for HIV-1-infected women? Br J Obstet Gynecol 2001;108:784–90.
26. Heikinheimo O, Lehtovirta P, Suni J, Paavonen J. The levonorgestrel-releasing intrauterine system (LNG-IUS) in HIV-infected women-effects on bleeding patterns, ovarian function and genital shedding of HIV. Hum Reprod 2006;21:2857–61.
27. Lehtovirta P, Paavonen J, Heikinheimo O. Experience with the levonorgestrel-releasing intrauterine system among HIV-infected women. Contraception 2007;75:37–9.
28. Gaffikin L, Phiri A, McGrath J, Zinanga A, Blumenthal PD. Provider attitudes toward IUC provision in Zimbabwe: perception of HIV risk and training implications. Adv Contracept 1998;14:27–39.
29. Mishell DR Sulak PJ. The IUD: dispelling the myths and assessing the potential. Dialogues Contracept 1997;5:1–4.
30. Cheng D. The intrauterine device: still misunderstood after all these years. South Med J 2000;93:859–64.
31. Kooiker CH, Scutchfield FD. Barriers to prescribing the Copper T 380A intrauterine device by physicians. West J Med 1990;153:279–82.
32. Espey E, Ogburn T, Espey D, Etsitty V. IUD-related knowledge, attitudes and practices among Navajo Area Indian Health Service providers. Perspect Sex Reprod Health 2003; 35:169–73.

33. Rivera R, Best K. Current opinion: consensus statement on intrauterine contraception. Contraception 2002;65: 385–8.

34. Farr G, Rivera R, Amatya R. Non-physician insertion of IUDs: clinical outcomes among TCu380A insertions in three developing-country clinics. Adv Contracept 1998;14: 45–57.

35. Freeman S. Nondaily hormonal contraception: considerations in contraceptive choice and patient counseling. J Am Acad Nurse Pract 2004;16:226–38.

36. Wesson J, Gmach R, Gazi R, Ashraf A, Méndez JF, Olenja J, et al. Provider views on the acceptability of an IUD checklist screening tool. Contraception 2006;74:382–8.

37. Hubacher D, Vilchez R, Gmach R, Jarquin C, Medrano J, Gadea A, et al. The impact of clinician education on IUD uptake, knowledge and attitudes: results of a randomized trial. Contraception 2006;73:628–33.

38. Chiou CF, Trussell J, Reyes E, Knight K, Wallace J, Udani J, et al. Economic analysis of contraceptives for women. Contraception 2003;68:3–10.

Published in final edited form as:
Contraception. 2010 February ; 81(2): 112. doi:10.1016/j.contraception.2009.08.002.

Intrauterine contraception in Saint Louis: A Survey of Obstetrician and Gynecologists' knowledge and attitudes

Tessa Madden[*], **Jenifer E. Allsworth**, **Katherine J. Hladky**, **Gina M. Secura**, and **Jeffrey F. Peipert**
[1]Washington University in Saint Louis School of Medicine, Department of Obstetrics and Gynecology, Saint Louis, MO 63110

Abstract

Background—Many obstacles to intrauterine contraception use exist, including provider and patient misinformation, high upfront cost, and clinician practice patterns. The aim of our study was to investigate knowledge and attitudes about intrauterine contraception among obstetricians and gynecologists in the area of Saint Louis.

Study Design—We mailed a self-administered, anonymous survey to 250 clinicians who provide obstetric and gynecologic care in Saint Louis City and County which included questions about demographics, training, family planning visits, and intrauterine contraceptive knowledge and use.

Results—The overall survey response rate among eligible clinicians was 73.7%. Clinicians who had recently finished training or saw higher numbers of contraceptive patients per week were more likely to insert intrauterine contraception than clinicians who completed training prior to 1989 or saw fewer contraceptive patients. Several misconceptions among clinicians were identified, including an association between intrauterine contraceptives and an elevated risk of pelvic inflammatory disease.

Conclusions—Physician misconceptions about the risks of intrauterine contraception continue to occur. Improved clinician education is greatly needed to facilitate the use of these highly effective, long-acting, reversible methods of contraception.

Keywords

Intrauterine contraception; intrauterine device; clinician knowledge; obstacles; survey

1. Introduction

Unintended pregnancy continues to be a substantial public health problem in the United States; over 3 million unintended pregnancies occur annually with poor and minority women disproportionately affected. Inconsistent and incorrect use of contraception is associated with unintended pregnancy [1]. Intrauterine contraception (IUC) is safe, highly effective with failure rates of less than 1%, [2] and does not require regular compliance from the user. Two types of IUC are available in the United States, the levonorgestrel intrauterine system (Mirena®, Bayer

*Corresponding Author: Tessa Madden, MD, MPH Washington University in Saint Louis School of Medicine, Department of Obstetrics and Gynecology, 4533 Clayton Ave, Box 8219, Saint Louis, MO 63110, Tel: 314 747 6495, Fax: 314 747 4019, maddent@wustl.edu.

HealthCare Pharmaceuticals, Wayne, NJ) and the copper T380A (Paragard®, Barr Pharmaceuticals, Montvale, NJ). These methods are associated with minimal side effects [3, 4], and have been shown to be safe when inserted immediately post-abortion [5,6]. Despite the many advantages of this "forgettable" contraception, only 1.3% of women aged 15 to 44 years currently use IUC [7]. In fact, use in the United States is substantially lower than many European and Asian countries with reported rates as high as 27% in Norway [8] and 30% in China [9]. Factors that contribute to the low uptake of IUC in the United States include knowledge and attitudes among clinicians and women, practice patterns among providers, and high initial cost.

A 2002 survey of Fellows of the American College of Obstetricians and Gynecologists regarding knowledge, attitudes, and practices about IUC found that while 95% of respondents agreed IUC was safe, the majority stated that nulliparous patients, and patients with a history of sexually transmitted infections (STIs) and pelvic inflammatory disease (PID) were not candidates for IUC [10]. Another study of over 800 California family planning providers found that fewer than 50% of respondents considered nulliparous women, teenagers, women who were immediate postpartum or postabortion, or women with a history of PID within the past 5 years to be candidates for IUC [11].

Increased use of highly effective methods of contraception such as IUC has been shown to decrease the rate of unintended pregnancies [12]. However, to increase uptake, physicians must be willing to provide these most effective methods of contraception to a wide range of patients. The purpose of this cross-sectional survey was to describe local clinician attitudes, knowledge and practice patterns about IUC; factors greatly influencing access to these contraceptive methods.

2. Materials and methods

We created a written, self-administered questionnaire to assess knowledge and attitudes about IUC among practicing providers of obstetrics and gynecology in the Saint Louis area. The questionnaire included demographics items such as age, race, and ethnicity, as well as questions about graduate medical training, contraceptive patients seen, and willingness to insert IUC. The survey was pretested among three clinician researchers.

Our goal was to obtain 100 completed surveys which would represent approximately 30% of practicing obstetrician-gynecologist clinicians in the area. Prior response rates for surveys mailed to physicians have been reported to be 34 to 51% [13,14]; we estimated a 40% response rate, requiring [250] mailed surveys We compiled a list of providers of obstetrics and gynecology in the Saint Louis area using publically available data sources such as faculty listings, the yellow pages of the telephone book, and Internet listing of obstetrician-gynecologist offices. We then performed a simple random sample using computer-generated numbers and mailed 250 questionnaires to the randomly selected clinicians between April and June of 2008. Surveys were mailed a second time to non-responders. All mailings contained a $20 gift card as an incentive.

All respondents were practicing in Saint Louis City or County, English-speaking, and willing to complete the survey. Written consent was waived, and completion and return of the survey implied consent to participate. This study was approved by the Washington University in Saint Louis Human Research Protections Office.

2.1. Statistical methods

Clinician characteristics were compared using the χ^2 test statistic. Unfortunately, we had no information regarding the characteristics of the clinicians who did not respond to the survey.

Low IUC rate was defined as having both inserted IUC and referred for IUC insertion zero to 10 times in the past year. We created this variable to capture clinicians who were both inserting none or few IUCs and referring none or few patients to another provider. Fisher exact tests were used for contingency tables with small cell size ($N \leq 5$). Since the outcomes of interest were common (> 10%), odd ratios are computed using Poisson regression as logistic regression would overestimate the odd ratios [15,16]. Univariable and multivariable odd ratios were estimated for the outcomes of interest. Statistically significant covariates in the univariable model were included in the final model. All statistical analyses were conducted using SAS version 9.1 (SAS Institute Inc., Cary, NC).

3. Results

3.1. Clinician characteristics

A total of 137 (54.8%) surveys were competed and returned. Fig. 1 shows the outcome of the 250 mailed surveys. Our final response rate among eligible clinicians with a valid mailing address was 73.7%. Table 1 shows the characteristics of the clinician respondents. The mean age of respondents was 49 years and they were mostly physicians (98.5%), predominantly white, and non-Hispanic.

3.2. IUC training and use

Thirty-six percent of clinicians were not trained in IUC insertion during their residency or advanced practice nurse core clinical training. Clinicians who trained at a Catholic institution were less likely to have received training in insertion during residency than clinicians who trained at secular institutions (OR 0.49 95% CI 0.36-0.68). Table 2 shows factors associated with low IUC rate; clinicians who finished training after 1999 (OR 0.13, 95% CI 0.02-0.85) or who saw a greater number of contraceptive patients per week (OR 0.36, 95% CI 0.19-0.69) were less likely to have low rates of IUC insertion and referral than clinicians who finished training before 1989 or saw fewer contraceptive patients.

Eighteen percent of respondents reported that they “always” discussed IUC with their patients, 76% reported that they discussed IUC most or some of the time. The majority of providers (67.9%) reported they had counseled more than 50 patients about IUC in the past year, and almost 66% reported that they had inserted greater than 10 IUCs in the past year. Less than 2% reported not counseling patients about intrauterine contraception within the past year and 12.4% reported not inserting IUC. Most respondents (79.6%) reported that they did not refer patients for IUC insertion; while 17.6% reported that they had referred 1 or more patients for IUC insertion. Only 2.9% of providers had neither inserted nor referred for IUC insertion in the past year. Forty percent of clinicians reported “always” and 52% reported “sometimes” testing for gonorrhea and chlamydia prior to insertion of IUC.

3.3. Clinician knowledge

Most respondents “strongly agreed” or “agreed” that IUC was safe (98.5%); however, 29% of clinicians reported that IUC causes an increased risk of PID other than at the time of insertion. The majority of clinicians correctly stated that antibiotics should not be given prophylactically at the time of insertion (78.1%), and that IUC does not cause abortion (86.1%). Seventy-eight percent of clinicians routinely recommended a follow-up visit after insertion. Table 3 shows the typical side effects emphasized by the clinician for each IUC type.

When asked about patient characteristics and the appropriateness of IUC, 62.0% agreed IUC was appropriate for a nulliparous patient; 30.7% for a teenaged patient, 45.3% for a patient with a STI in the past 2 years, 36.5% for a patient with PID in the past 5 years, and 36.5% for a patient in a non-monogamous relationship. The vast majority of clinicians (97.8%) reported

that they would offer IUC to a 35-year-old patient, who was married, monogamous and had 3 children; 67.1% reported they would offer intrauterine contraception to a 30 year old who was unmarried, had 2 children, and had a boyfriend; 49.6% said they would offer IUC to a 17-year-old who was unmarried, monogamous and had 1 child. Only 19% would be willing to offer IUC to an unmarried 17-year-old who had never been pregnant.

4. Discussion

This survey was designed to collect information about providers' attitudes and knowledge about IUC. Our results suggest that physicians who completed training after 1999 are more likely to insert IUC than physicians who completed training prior to 1989. The overwhelming majority of clinicians (98%) believed IUC is safe, however, almost one third reported incorrectly that IUC is associated with an increased risk of PID other than the increased peri-insertional risk. Table 3 also suggests some providers may not appropriately emphasize the most common side effects encountered with IUC; 75.9% reported counseling patients about an increase in spotting and breakthrough bleeding with both levonorgestrel IUC (LNG-IUC) and copper IUC (copper-IUC); this side effect can be seen with both types of IUC; however, is more commonly associated with LNG-IUC and is typically limited to the first 3 to 6 months of use. Eleven percent reported counseling patients about cessation of menstruation with both LNG-IUC and copper-IUC; however, copper-IUC should not cause amenorrhea. Thirty-three percent reported that both LNG-IUC and copper-IUC can be associated with painful menstruation; however, the use of LNG-IUC has been shown to decrease the incidence of menorrhagia and dysmenorrheal [17].

Most of the clinicians surveyed were willing to offer IUC to a 35-year-old multiparous patient who is married. However, we find it worrisome that the number of clinicians who will offer IUC declines dramatically with patients of decreasing age, parity, and non married status. For example, only 19% would offer IUC to a 17 year old who is not married and has never been pregnant. This suggests the misconceptions about associations between IUC, PID, and infertility continue to persist. The World Health Organization states only current cervicitis and recent PID within 3 months are contraindications to IUC. They do not consider age less than 20 years or nulliparity to be contraindications [18]. A recent editorial identified limited education and outdated beliefs as one of the main provider-based barriers to contraception among teens and young adults and called for the increased education and training of health-care providers [19]. Since young women are at high risk for unintended pregnancy, it is imperative that we increase this population's access to the most effective methods of contraception.

Our study has similarities to the study by Stanwood et al. [10] However, there are several important differences: 1) The prior study was administered before the introduction of LNG-IUC in the United States; in comparison, our survey obtains knowledge and attitudes about both types of IUC available, 2) In 2005, the copper-IUC FDA labeling was liberalized to not restrict insertion in nulliparous women, and in the immediate postabortion and postpartum time period. In addition, having more than one sexual partner was removed as a contraindication. Since we surveyed physicians after the new labeling was introduced, our survey adds new information about clinician knowledge about and attitudes towards IUC.

This study is limited by its relatively small sample size and has limited power to detect statistically significant differences. Additionally, because our findings are from a single geographical area, they may not be generalizable to clinicians in other geographical regions. Strengths of our study include a random sample of practicing obstetrician-gynecologists rather than a convenience sample, and a high response rate, which strengthens the generalizability of our findings.

Despite the introduction of LNG-IUC and liberalization of the current package labeling of copper-IUC, our survey has identified several potential barriers that remain to increasing the use of IUC: 1) inadequate training of clinicians (more than one-third of physicians were not trained in residency); 2) misperceptions regarding appropriate candidates for IUC; and 3) inaccurate knowledge of IUC and possible side effects. These barriers may influence contraceptive counseling and subsequent method choice. Targeted education and training programs to clinicians should dispel these pervasive myths and inaccuracies and encourage the use of these highly effective and safe contraceptive methods.

Acknowledgments

This research was supported by a grant from an anonymous foundation. Individual investigators (JP and JA) were supported, in part, by a Mid-career Investigator Award in Women's Health Research (K24 HD01298) and by the Clinical and Translational Science Awards (UL1RR024992) and Grant Number KL2RR024994 from the National Center for Research Resources (NCRR), a component of the National Institutes of Health (NIH) and NIH Roadmap for Medical Research. Its contents are solely the responsibility of the authors and do not necessarily represent the official view of NCRR or NIH. Information on NCRR is available at http://www.ncrr.nih.gov/. Information on Re-engineering the Clinical Research Enterprise can be obtained from http://nihroadmap.nih.gov/clinicalresearch/overview-translational.asp.

We would like to thank Cynthia Harper for sharing her previously developed survey questions and Younathan Abdia for his assistance with statistical analysis.

References

1. Finer LB, Henshaw SK. Disparities in rates of unintended pregnancy in the United States, 1994 and 2001. Perspect Sex Reprod Health 2006;38:90–96. [PubMed: 16772190]
2. Trussell, J. Contraceptive Efficacy. In: Hatcher, RATJ.; Nelson, AL.; Cates, W.; Stewart, FH.; Kowal, D., editors. Contraceptive Technology. New York (NY): Ardent Media; 2007. p. 747-760.
3. Suhonen S, Haukkamaa M, Jakobsson T, Rauramo I. Clinical performance of a levonorgestrel-releasing intrauterine system and oral contraceptives in young nulliparous women: a comparative study. Contraception 2004;69:407–412. [PubMed: 15105064]
4. Baldaszti E, Wimmer B, Loschke K. Acceptability of the long-term contraceptive levonorgestrel-releasing intrauterine system (Mirena): a 3-year follow-up study. Contraception 2003;67:87–91. [PubMed: 12586318]
5. Ortayli N, Bulut A, Sahin T, Sivin I. Immediate postabortal contraception with the levonorgestrel intrauterine device, Norplant, and traditional methods. Contraception 2001;63:309–314. [PubMed: 11672552]
6. Pakarinen P, Toivonen J, Luukkainen T. Randomized comparison of levonorgestrel- and copper-releasing intrauterine systems immediately after abortion, with 5 years' follow-up. Contraception 2003;68:31–34. [PubMed: 12878284]
7. Chandra A, Martinez GM, Mosher WD, Abma JC, Jones J. Fertility, family planning, and reproductive health of U.S. women: data from the 2002 National Survey of Family Growth. Vital Health Stat 2005;23:1–60.
8. Sonfield A. Popularity Disparity: Attitudes about the IUD in Europe and the United States. Guttmacher Policy Review 2007;10:19–24.
9. Wang D. Contraceptive failure in China. Contraception 2002;66:173–178. [PubMed: 12384206]
10. Stanwood NL, Garrett JM, Konrad TR. Obstetrician-gynecologists and the intrauterine device: a survey of attitudes and practice. Obstet Gynecol 2002;99:275–280. [PubMed: 11814509]
11. Harper CC, Blum M, de Bocanegra HT, et al. Challenges in translating evidence to practice: the provision of intrauterine contraception. Obstet Gynecol 2008;111:1359–1369. [PubMed: 18515520]
12. Goodman S, Hendlish SK, Benedict C, Reeves MF, Pera-Floyd M, Foster-Rosales A. Increasing intrauterine contraception use by reducing barriers to post-abortal and interval insertion. Contraception 2008;78:136–142. [PubMed: 18672115]

13. Hogben M, Wimberly YH, Moore S. Estimating dissemination of Centers for Disease Control and Prevention Sexually Transmitted Disease Treatment Guidelines from a survey of physicians. Int J STD AIDS 2007;18:318–320. [PubMed: 17524191]

14. McCormick D, Woolhandler S, Bose-Kolanu A, Germann A, Bor DH, Himmelstein DU. U.S. Physicians' Views on Financing Options to Expand Health Insurance Coverage: A National Survey. J Gen Intern Med 2009;29

15. Zhang J, Yu KF. What's the relative risk? A method of correcting the odds ratio in cohort studies of common outcomes. JAMA 1998;280:1690–1691. [PubMed: 9832001]

16. McNutt LA, Wu C, Xue X, Hafner JP. Estimating the relative risk in cohort studies and clinical trials of common outcomes. Am J Epidemiol 2003;157:940–943. [PubMed: 12746247]

17. Varma R, Sinha D, Gupta JK. Non-contraceptive uses of levonorgestrel-releasing hormone system (LNG-IUS)--a systematic enquiry and overview. Eur J Obstet Gynecol Reprod Biol 2006;125:9–28. [PubMed: 16325993] WHO. Medical eligibility criteria for contraceptive use. Third edition2008 [Accessed 10/31/08]. http://www.who.int/reproductive-health/publications/mec/iuds.htmlBrown SS, Burdette L, Rodriguez P. Looking inward: provider-based barriers to contraception among teens and young adults. Contraception 2008;78:355–357. [PubMed: 18929730]

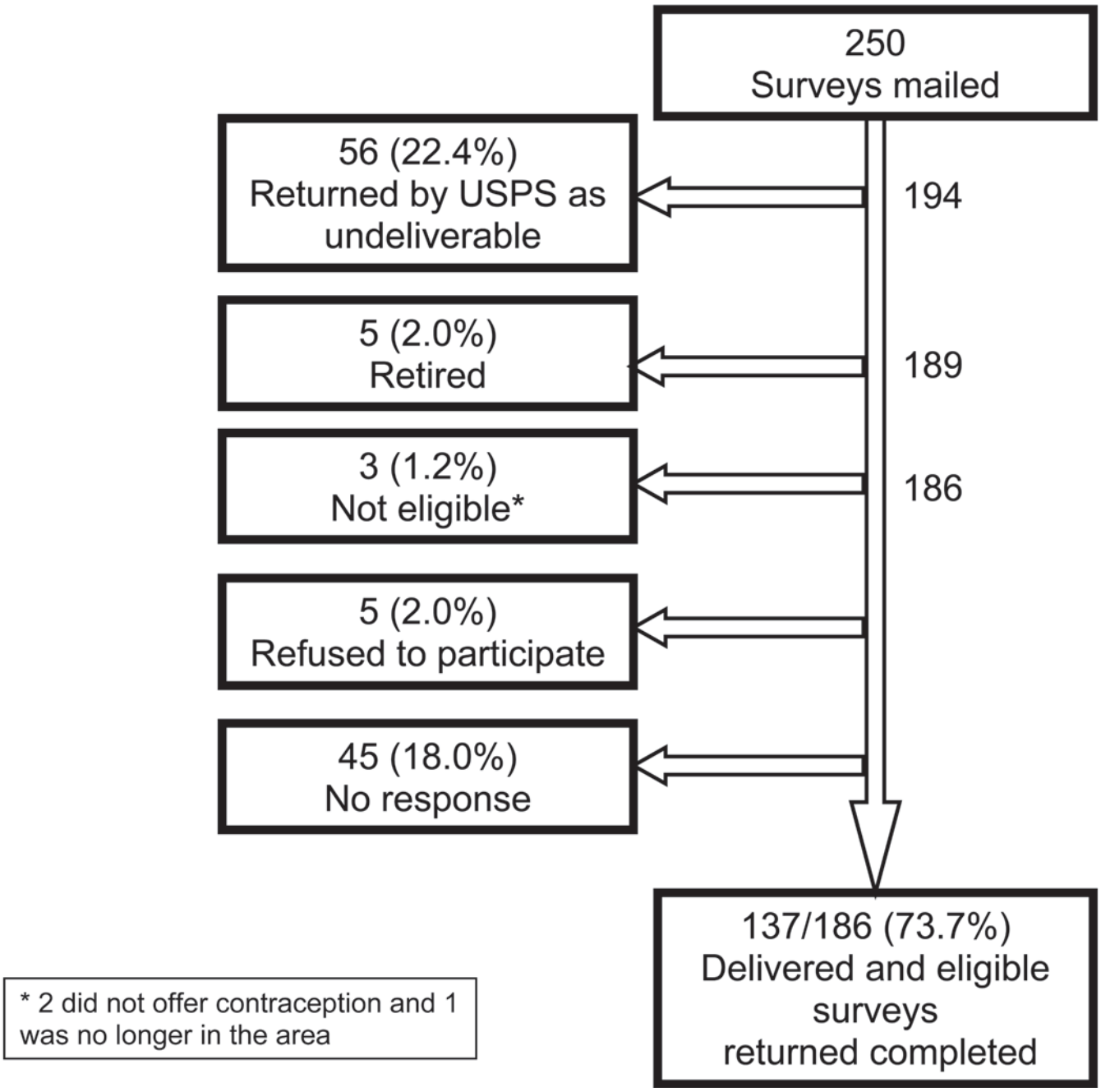


Figure 1.
Schematic of mailed and returned questionnaires.

Table 1

Characteristics of the 137 Clinician Respondents

Characteristic	N (%)
Job Title	
Physician	135 (98.8)
Advanced Practice Nurse	2 (1.5)
Race	
White	117 (85.4)
Black	6 (4.4)
Other	14 (10.2)
Ethnicity	
Hispanic	6 (4.4)
Year Completed Residency	
Before 1989	59 (43.7)
1989 – 1999	55 (40.7)
After 1999	21 (15.6)
Catholic Institution for Residency	
Yes	61 (55.5)
No	76 (44.5)
IUC Insertion Included During Residency	
Yes	88 (64.2)
No	49 (35.8)
Avg. Number of Contraceptive Patients/Week	
0 – 25	45 (35.43)
26 – 50	63(49.61)
51 +	19(14.96)
Not Ascertained	10 (7.30)

Table 2

Univariable and Multivariable Models of Provider Characteristics Associated with Low IUC Rate

Characteristic	Univariable OR (95%CI)	Multivariable OR (95% CI)*
Year residency completed		
Before 1989	Reference	Reference
1989 – 1999	0.66 (0.3 –1.17)	0.62 (0.36–1.07)
After 1999	0.13 (0.02–0.92)	0.13 (0.02–0.85)
Catholic institution		
Yes	0.68 (0.37–1.26)	1.23 (0.71–2.13)
No	Reference	Reference
Trained to insert IUC in residency		
Yes	1.53 (0.78–3.01)	1.16 (0.62–2.15)
No	Reference	Reference
Average Contraceptive Patients Seen per Week		
0 – 25	Reference	Reference
26 – 50	0.36 (0.19–0.69)	0.34 (0.19–0.65)
51 +	0.24 (0.06–0.93)	0.20 (0.05–0.77)

*Adjusted for year residency completed, training at a Catholic institution, training in IUC insertion during residency, and average number of contraceptives seen per week

IUC – Intrauterine contraception

Table 3

Clinician Report of Side Effects Commonly Associated with Levonorgestrel IUC and Copper IUC

Side Effect	LNG-IUC Only	% Copper-IUC Only	Both	Neither
Spotting or breakthrough bleeding	15.3	4.4	75.9	8.7
Heavy or prolonged menstruation	0.7	63.5	24.8	8.8
No menstruation	83.2	0	11.0	3.7
Painful menstruation	0.7	46.0	33.6	17.5

LNG-IUC – levonorgestrel intrauterine contraception; Copper-IUC – copper intrauterine contraception

ELSEVIER

Contraception 74 (2006) 389–393

Contraception

Original research article

Conformity with current guidelines on oral contraceptive prescribing for breastfeeding women: a New Mexico survey

Eve Espey[a,*], Tony Ogburn[a], Larry Leeman[b], Shilpa Reddy[c], Carisa Lee[a], Clifford Qualls[d]

[a]*Department of Obstetrics and Gynecology, University of New Mexico, Albuquerque, NM 87131, USA*
[b]*Department of Family and Community Medicine, University of New Mexico, Albuquerque, NM 87131, USA*
[c]*University of New Mexico, School of Medicine, Albuquerque, NM 87131, USA*
[d]*Clinical Research Center, University of New Mexico, Albuquerque, NM 87131, USA*

Received 18 April 2006; revised 13 June 2006; accepted 20 June 2006

Abstract

Background: National and international contraceptive guidelines reflect expert opinion that recommends against the use of estrogen-containing hormonal contraception in the early postpartum period. This study was undertaken to estimate providers' practices in prescribing hormonal contraception to breastfeeding women.

Methods: A 19-item survey was mailed to 397 obstetrician gynecologists, midwives and family physicians in the state of New Mexico. The survey included items covering attitudes about the impact of hormonal contraception on breastfeeding and prescribing practices. One hundred ninety-nine (50%) providers completed the survey.

Results: The majority (70%) of providers prescribe progestin-only contraceptive methods to breastfeeding women within the first 6 weeks. Despite these recommendations, a sizable minority of providers prescribe combined pills in the early postpartum period: 27% of providers have prescribed combined pills and 13% of providers, mostly those in a university setting, routinely recommend them within the first 6 weeks postpartum.

Conclusion: Most providers follow expert recommendations regarding the initiation of hormonal contraception for breastfeeding women.

Keywords: Hormonal contraceptives; Breastfeeding; Prescribing

1. Introduction

A long-standing convention cautions breastfeeding women against using combined oral contraceptives (OCs). A number of organizations involved in women's health care support this guideline, including the American College of Obstetricians and Gynecologists (ACOG), the World Health Organization (WHO) and the International Planned Parenthood Federation (IPPF). The ACOG's practice bulletin on the use of contraception in women with coexisting medical conditions states that, "Combination OCs are not recommended as the first choice for breastfeeding mothers because of the negative effect of contraceptive doses of estrogen on lactation. The estrogenic component of combination OCs can reduce the volume of milk production and the caloric and mineral content of breast milk in lactating women" [1]. In contrast, progestin-only OCs are considered first line for lactating women.

The WHO, in its 2004 medical eligibility criteria for contraceptives, states that combined OCs are contraindicated in the first 6 weeks of lactation, citing a "theoretical concern that the neonate may be at risk due to exposure to steroid hormones during the first 6 weeks postpartum." The WHO states that the use of combined OCs in breastfeeding women from 6 weeks to 6 months postpartum is a practice for which harm probably outweighs the benefits, as the "use of combined oral contraceptives during breastfeeding diminishes the quantity of breast milk, decreases the duration of lactation and may thereby adversely affect the growth of the infant" [2]. The IPPF's opinion is similar to that of the WHO, but extends the caution even further, viewing combined OC use prior to 6 months postpartum in breastfeeding women as contraindicated, citing diminished quantity of breast milk and decreased duration of lactation [3].

These recommendations are based on scanty and flawed research. A recent Cochrane collaboration review concludes that evidence is insufficient to reach conclusions about the impact of hormonal contraception on

* Corresponding author. Tel.: +1 505 272 6309; fax: +1 505 272 6385.
E-mail address: eespey@salud.unm.edu (E. Espey).

0010-7824/$ – see front matter © 2006 Elsevier Inc. All rights reserved.
doi:10.1016/j.contraception.2006.06.007

Table 1
Demographic characteristics of the three groups of survey respondents (OB-GYNs, CNMs and FPs)

	OB-GYN ($n=63$)	CNM ($n=65$)	FP ($n=71$)	p
Gender [n (%)]				
Male	30	0	41	<.001[a]
Female	33 (52)	65 (100)	28 (41)	
Deliveries per year [n (%)]				
1–50	9 (14)	11 (17)	54 (79)	<.001[a]
>50	55 (86)	52 (83)	14 (21)	
Type of practice [n (%)]				
Solo–group	42 (65)	38 (59)	33 (50)	<.001[a]
Health maintenance organization	9 (9)	8 (13)	0 (0)	
University	15 (23)	11 (17)	16 (24)	
Other	2 (3)	7 (11)	17 (26)	
Age in years [mean (range)]	47 (33, >60)	48 (28, >60)	43 (28, 58)	.006[b]
Years in practice [mean (range)]	15 (9–20)	11 (6–18)	10 (5–17)	.01[b]

[a] Fisher's Exact Test was used for gender, deliveries per year and type of practice.
[b] Kruskal–Wallis test was used for mean age and years in practice.

breastfeeding [4]. As many women worldwide breastfeed and use OCs, these recommendations have a major public health impact. Overall, the combined pill has advantages over the progestin-only pill: it has fewer side effects (such as irregular bleeding), better efficacy and higher continuation rates [5]. If, however, the combined pill truly diminishes the quantity of breast milk, then a progestin-only pill might be preferable, taking into consideration the needs of a particular patient. As a result of the need to balance the importance of breastfeeding with the need for optimal contraception, some providers offer combination pills to breastfeeding mothers. The extent of this practice is unknown.

The purpose of this study was to estimate what types of OCs are prescribed for postpartum breastfeeding women by New Mexico obstetrician gynecologists (OB-GYNs), family physicians (FPs) and certified nurse midwives (CNMs), and to estimate differences between provider types in prescribing practices.

2. Materials and methods

A 19-item survey was designed by the authors to assess prescribing practices for postpartum OCs. The survey was mailed to all FPs who performed deliveries and to all OB-GYNs and CNMs in the state of New Mexico in January 2005. Contact information was obtained through the American Medical Association physician list, the CNM state midwifery society list and a University of New Mexico Family Practice maternity care database. Reminder postcards were sent at 2 weeks, and telephone follow-up was used for nonresponders. All survey data remained confidential, available only to the investigators.

The survey was pretested and modified to clarify questions based on responses from 20 OB-GYN residents. Survey questions included demographic characteristics such as age, years in practice, type of medical practice and number of deliveries per year. Several questions explored whether providers prescribe hormonal contraception to breastfeeding women, the timing of initiation of hormonal contraception relative to delivery and whether providers routinely prescribe progestin-only methods, combined methods or both. "True–false" knowledge and opinion questions about their understanding of the impact of hormonal contraception on milk quantity, milk quality and infant growth were posed.

Two breastfeeding scenarios were presented as part of the survey to determine whether providers altered their recommendations about hormonal contraception based on patient factors that could influence the success of breastfeeding. In the favorable scenario, the patient successfully breastfed her other children and is breastfeeding without problems in the hospital. In the unfavorable scenario, the patient is primiparous and is having difficulty breastfeeding in the hospital. Responses were categorized into four categories:

"Progestin-only methods to begin after 6 weeks" (the most cautious)
"Progestin-only methods to begin within 6 weeks"
"Combined pills to begin after 6 weeks"
"Combined pills to begin within 6 weeks" (the least cautious).

Additional questions included opinions about the merits of progestin-only versus combined OCs and whether providers who recommended progestin-only pills for breastfeeding women switched their patients to combined pills when they had stopped breastfeeding.

The study was approved by the Human Research Review Committee of the University of New Mexico. All data were analyzed using SAS package. Because data for age and years in practice were skewed, Kruskal–Wallis tests were used for comparisons. Fisher's Exact Test for two-way frequency tables was used for overall analysis, and post hoc testing was used to determine differences between providers. For providers' opinion questions, estimated proportions were reported with 95% confidence intervals (95% CIs). The Generalized McNemar test was used to compare differences in providers' prescribing responses to two

Table 2
Providers' opinions on the impact of combined OC pills on breastfeeding

Survey item	OB-GYN	CNM	FP	p
	n yes/total *n* (%) [95% CI]			
Combined OCs decrease milk quantity	48/61 (79) [66–88]	55/58 (95) [86–99]	45/55 (82) [69–81]	.02[a]
Combined OCs decrease milk quality	6/43 (14) [5–28]	11/34 (32) [17–51]	5/49 (10) [3–22]	.03[a]
Breastfeeding infants show appropriate growth	40/41 (98) [87–100]	26/29 (90) [73–98]	43/45 (96) [85–96]	.38
Combined OCs decrease breastfeeding duration	20/42 (48) [32–64]	20/33 (61) [42–77]	23/50 (46) [32–61]	.41
Combined OCs are superior to progestin-only pills	50/61 (82) [70–91]	45/59 (76) [63–86]	41/64 (64) [51–76]	.07

The proportions reported reflect those persons answering either yes or no. Each estimated proportion is followed by its 95% CI. Those answering "did not know" were excluded from the analysis.

[a] CNMs were more likely than FPs or OB-GYNs to answer "yes" in post hoc testing.

different breastfeeding scenarios — one more favorable and one less favorable.

3. Results

The survey was mailed to 397 providers in total: 160 OB-GYNs, 130 CNMs and 107 FPs. We received a total of 205 surveys; 199 answered affirmatively to the first question, "Do you perform deliveries?" The six who did not perform deliveries were excluded from the analysis. The overall response rate of usable surveys was 51% (63 from OB-GYNs, 65 from CNMs and 71 from FPs). The demographic characteristics of the three groups of providers appear in Table 1. The midwives were all female, creating a difference in gender from the other two groups. OB-GYNs and CNMs were older and have performed more deliveries than FPs. Eighty percent of all practitioners responding to the questionnaire were either in solo–group practice or in university practice. OB-GYNs have been in practice longer than the CNMs and FPs surveyed.

Providers differed in some beliefs about the impact of combined OC pills on breastfeeding (Table 2). A majority of providers, but significantly more CNMs, believed that combined OCs diminished milk supply, and about half of all providers believed that breastfeeding duration was decreased in combined OC users. The minority of providers, but more CNMs, believed that combined OCs affected milk quality. Most providers believed that infants of breastfeeding mothers using combined OCs grow appropriately and that combined pills are superior to progestin-only pills.

Only four providers, two OB-GYNs and two CNMs, reported that they never recommend hormonal contraception within 6 weeks or after 6 weeks postpartum. Of the remaining providers, prescribing practices changed as a function of time postpartum (Table 3). Within the first 6 weeks postpartum, 70% of all providers exclusively prescribed progestin-only methods of hormonal contraception to breastfeeding women, and the majority reported that they routinely discouraged combination pills because they may decrease milk production. Thirty percent of providers reported they had prescribed both progestin and combined methods. After 6 weeks postpartum, the percentage of providers who had ever prescribed both rose to 56%.

Although 30% had ever prescribed combined pills within the first 6 weeks, 13% routinely recommended them in that time frame. The majority of those who routinely recommended combined pills within the first 6 weeks were university providers, and the major reasons they cited for the recommendation of combination pills over progestin-only pills were greater effectiveness and better compliance. Ninety-six percent of OB-GYNs and CNMs versus 79% of FPs responded that they switched women from progestin-only to combined pills when they stopped breastfeeding (Fisher's Exact Test, $p=.003$).

Providers who prescribe hormonal contraception to breastfeeding women are more likely to prescribe in a cautious manner when faced with a less favorable breastfeeding scenario than when faced with a more favorable scenario (Generalized McNemar test, $p=.01$). Of 171 respondents, 27 prescribed more cautiously in an unfavorable scenario versus 7 who prescribed less cautiously and 134 who remained neutral.

4. Discussion

The major finding of this survey is that most providers prescribe progestin-only methods for hormonal contraception in breastfeeding women. A minority prescribe combined pills prior to 6 weeks postpartum, and more providers prescribe combined pills after 6 weeks postpartum.

Table 3
Types and timing of methods prescribed by providers who indicated that they "ever" prescribed hormonal contraception to breastfeeding women

Timing	Type prescribed	OB-GYN	CNM	FP	p	Post hoc test
Within 6 weeks postpartum	Progestin methods only	34 (62%)	53 (83%)	37 (65%)	.02	OB-GYNs and FPs more likely than
	Combined and progestin methods	21 (38%)	11 (17%)	20 (35%)		CNMs to prescribe combined pills
	n	55	64	57		
After 6 weeks postpartum	Progestin methods only	27 (48%)	35 (56%)	16 (27%)	.003	FPs more likely than CNMs and
	Combined and progestin methods	29 (52%)	28 (44%)	44 (73%)		OB-GYNs to prescribe combined pills
	n	56	63	60		

OB-GYNs were more likely than midwives or FPs to prescribe combined pills. University providers were more likely than nonuniversity providers to prescribe combined pills. The majority of all providers consider combined pills to be superior to progestin-only pills.

The fact that providers used more caution in prescribing combined pills to women in an unfavorable breastfeeding scenario indicates that providers do take into account the potential negative impact of hormonal contraceptives on breastfeeding and factor individual patients' circumstances into their recommendations for the type and the timing of hormonal contraceptives.

A limitation of our study is the 49% nonresponse rate. The main demographic data available in our database for nonresponders were on type of practice: university versus nonuniversity. We did find a difference in response rates between university (88%) and nonuniversity providers (45%; $p<.001$). Since university providers were more likely to routinely recommend combined pills within 6 weeks postpartum, we probably overestimated the total percentage of providers who routinely recommend combined pills within 6 weeks postpartum.

One other report in the literature examines prescribing practices. A 1981 survey of 754 doctors in 65 countries found that 45% had ever prescribed a combination pill to breastfeeding women [6]. The investigators found that women's preference, previous breastfeeding history and the use of feeding supplements influenced their decision to prescribe an estrogen-containing OC.

Recommendations from maternal–child health authorities, such as the ACOG and the WHO, against the use of combined OCs in breastfeeding women are unambiguous. The rationale for the ACOG's conservative guidance is that 6 weeks of breastfeeding may give women time to overcome initial difficulties in establishing milk supply and that estrogen-containing pills would be less likely to diminish milk production [7]. Although concerns have been raised about the passage of hormones in breast milk, estrogen-containing contraceptives are generally considered compatible with breastfeeding [8]. An additional rationale for waiting at least 2–3 weeks before beginning combined OCs is the hypercoagulable postpartum state. Although hypercoagulability and increased risk of thrombotic events have been documented during the puerperium [9], no studies have shown a higher incidence in women who breastfeed and use OCs.

Despite strong recommendations against the initiation of combined hormonal contraceptives prior to 6 weeks, a sizable minority of providers prescribe combined pills in the early postpartum period. Ironically, university providers — those who have a particular responsibility to educate the next generation of providers — were more likely not to follow expert guidelines.

One explanation for not following the guidelines may be the near-universality of providers' opinions about the superior contraceptive efficacy of combined pills over progestin-only pills. In weighing the two important public health issues of promoting breastfeeding and supporting the prevention of unintended pregnancy, some providers may believe that the superior nature of combined pills as a contraceptive method outweighs the potential negative impact on breastfeeding. Additionally, patients seen in our university setting are often poor and may be uninsured or may have lost their insurance shortly after delivery. Access to return appointments for switching from one pill type to another poses a greater difficulty for this population.

Another answer may lie in the scanty and imprecise evidence linking the use of combined pills to a decline in milk volume and a reduction in the duration of breastfeeding. Although several reports in the literature address hormonal contraception and lactation, most studies are nonrandomized or simply reflect expert opinion [10–12]. Results are conflicting regarding the impact of estrogen-containing contraceptives on milk volume and, more importantly, breastfeeding duration and infant growth. A Cochrane collaborative review cites only three randomized controlled trials of sufficient quality to be included in their review of the impact of estrogen-containing OCs on breastfeeding [4]. All three trials suffered from major methodologic flaws such as a large loss to follow-up, which may invalidate the main conclusions. Additionally, the reports failed to describe adequately a number of elements of the study design, including randomization allocation, allocation concealment, blinding of treatments and/or use of intention-to-treat analysis. The Cochrane review concludes that existing data are insufficient to make recommendations about lactation and the use of hormonal contraception.

The study most often cited as the reason for expert recommendations against prescribing combined OCs in breastfeeding women is a WHO trial in which 343 women were randomized to low-dose combined OCs or to progestin-only pills, both initiated at 6 weeks postpartum [13]. The major outcomes of the study were mean milk volume in a given feeding, measured using a complicated methodology, and infant growth. At 12 and 24 weeks postpartum, mean milk volume was found to be significantly lower in the combined OC group than in the progestin-only pill group (51 and 41 ml, respectively, in the combined pill group versus 72 and 65 ml, respectively, in the progestin-only pill group). No differences in infant growth were noted between the two groups, and the investigators concluded that their method of measuring milk output "may have little relationship to the amount actually ingested by the baby during that or any 24-hour period." Despite these limitations, the authors concluded that combination pills should not be used in breastfeeding women.

In a detailed review of the evidence, recommendations and controversies surrounding estrogen-containing contraceptives for breastfeeding women, Erwin [14] eloquently describes the importance of choosing the correct contraceptive. He affirms the superior efficacy and side-effect profile of estrogen-containing pills over progestin-only pills and alludes to the concern that women may discontinue

breastfeeding in order to initiate combined pills. He acknowledges the risk of rapid repeat pregnancy in a woman who foregoes her preferred contraceptive in order to breastfeed. He concludes, in agreement with experts, that combined pills should not be used in the first 2 months postpartum, although "no consistent findings show deleterious effects of combined pill use on infant growth and development when pill use has begun after lactation is established." The Cochrane review concludes by calling for appropriately designed randomized controlled trials examining the impact of hormonal contraception on breastfeeding. This study was undertaken to delineate practice patterns of clinicians in New Mexico and not to promote changes in prescribing practices. Until well-designed randomized trials have been completed, providers may reasonably continue to prescribe according to their preference or to counsel patients about nonhormonal contraceptive methods such as the intrauterine device, an ideal method of contraception for breastfeeding women. A well-designed study is urgently needed to determine the impact of hormonal contraception on breastfeeding and infant growth to help guide clinicians' practice.

References

[1] Kaunitz A. ACOG practice bulletin no. 18. Clinical management guidelines for obstetrician-gynecologists. The use of hormonal contraception in women with coexisting medical conditions 2000. www.acog.org/publications/educational_bulletins/pb018.cfm.

[2] WHO medical eligibility criteria. 3rd ed. 2004. Available at: http://www.who.int/reproductive-health/publications/MEC_3/mec.pdf [Retrieved on 5/15/2005].

[3] International Planned Parenthood Federation: directory of hormonal contraceptives medical and service delivery guidelines. 3rd ed. 2004. Available at: http://content.ippf.org/output/ORG/files/5950.pdf, [Retrieved on 5/15/2005].

[4] Truitt ST, Fraser AB, Grimes DA, Gallo MF, Schulz KF. Combined hormonal vs. non-hormonal versus progestin-only contraception in lactation. Cochrane Database Syst Rev 2003;(2):1–14 [art. no. CD003988, DOI: 10.1002/14651858.CD003988].

[5] Hatcher RA, Rinehart W, Blackburn R, Geller JS, Shelton JD. Essentials of contractive technology. Baltimore (MD): Population Information Program, Johns Hopkins Bloomberg School of Public Health; 1997.

[6] Strauss LT, Speckhard M, Rochat RW, Senanayake P. Oral contraception during lactation — a global survey of physician practice. Int J Gynaecol Obstet 1981;169–75.

[7] Queenan J. ACOG educational bulletin no. 258. Breastfeeding: maternal and infant aspects 2000. http://www.acog.org/publications/educational_bulletins/btb258.cfm.

[8] American Academy of Pediatrics. The transfer of drugs and other chemicals into human milk. Pediatrics 2001;108(3):776–89.

[9] James AH, Tapson VF, Goldhaber SZ. Thrombosis during pregnancy and the postpartum period. Am J Obstet Gynecol 2005;193:216–9.

[10] Nilsson S, Mellbin T, Hofvander Y, Sundelin C, Valentin J, Nygren KG. Long-term follow-up of children breast-fed by mothers using oral contraceptives. Contraception 1986;34:443–57.

[11] Halderman LD, Nelson AL. Impact of early postpartum administration of progestin-only hormonal contraceptives compared with nonhormonal contraceptives on short-term breast-feeding patterns. Am J Obstet Gynecol 2002;186:1250–8.

[12] Visness CM, Rivera R. Progestin-only pill use and pill switching during breastfeeding. Contraception 1995;51:279–81.

[13] Tankeyoon M, Dusitsin N, Chalapati S, Koetsawang S, Saibiang S, Sas M. Effects of hormonal contraceptives on milk volume and infant growth. Contraception 1984;30:505–22.

[14] Erwin PC. To use or not use combined hormonal oral contraceptives during lactation. Fam Plann Perspect 1994;26:26–31.

Obstetrician-Gynecologists and the Intrauterine Device: A Survey of Attitudes and Practice

Nancy L. Stanwood, MD, MPH, Joanne M. Garrett, PhD, and Thomas R. Konrad, PhD

OBJECTIVE: To assess obstetrician-gynecologists' clinical use of the intrauterine device (IUD), their attitudes toward the IUD and how they select IUD candidates, and to test the hypotheses that limited residency training in IUDs, fear of litigation, and a belief that IUDs cause pelvic inflammatory disease decrease IUD use.

METHODS: We performed a national mailed survey of 811 practicing obstetrician-gynecologists obtained from systematic sampling of ACOG membership listings to assess use of and attitudes toward the IUD.

RESULTS: The survey response rate was 50%. Most respondents agreed that the copper IUD is safe (95%) and effective (98%). However, 20% of respondents had not inserted an IUD in the past year, and of those who had, most (79%) reported inserting 10 or fewer. Fear of litigation and a belief that IUDs cause pelvic inflammatory disease were associated with lower IUD use; the number of IUDs inserted during residency was not. In selecting IUD candidates, respondents were most restrictive about patient monogamy. Having less conservative criteria for selecting IUD candidates was associated with greater IUD use. Respondents with liberal criteria inserted a mean of nine IUDs in the past year, whereas those with conservative criteria inserted four.

CONCLUSIONS: Because most obstetrician-gynecologists are inserting few IUDs, educational programs should target these physicians to expand their IUD use. Such programs should highlight modern IUD safety and the rarity of litigation. The number of IUDs inserted in residency may be less important than the development of less restrictive, more evidence-based criteria for selecting IUD candidates. (Obstet Gynecol 2002;99:275–80. © 2002 by the American College of Obstetricians and Gynecologists.)

The intrauterine device (IUD) provides safe[1] and effective[2] contraception, and 12% of married women of reproductive age worldwide use it.[1] In the United States, however, only 0.8% of women using contraception use the IUD.[3] Before the debacle involving the Dalkon shield, 9.5% of married, white US women using contraception used the IUD.[4] After the Dalkon Shield, manufacturers withdrew most devices from the US market, and IUD use decreased.[5] Physicians and the public developed a persistent fear that all IUDs cause pelvic inflammatory disease (PID) despite evidence that PID after modern copper IUD insertion occurs rarely, at a rate of 1.6 per 1000 woman-years of use.[6] Reanalysis of the early studies linking IUDs and PID question their methodology and generalizability.[7,8] The relative underuse of the IUD in the United States reflects these public and professional concerns. The reluctance of US physicians to recommend IUDs except in narrowly selected patients[9] contributes to this underuse. Many factors contribute to this reluctance, including a lack of training in use of IUDs during residency, a fear of litigation, and a belief that the IUD creates a high risk for PID.[10–12]

Little is known about how obstetrician-gynecologists use the IUD in clinical practice, what their attitudes toward the IUD are, or how they select IUD candidates in their practices. In 1989, Kooiker and Scutchfield[11] surveyed obstetrician-gynecologists and family and general internal medicine physicians in San Diego County, CA, shortly after the 1988 release of the Paragard T380A Intrauterine Copper Contraceptive device (Ortho Pharmaceutical Corporation, Raritan, NJ) in the United States. They asked the physicians whether they recommended this new IUD to patients and whether they planned to use it in their practices. In their sample, 40% were not recommending this IUD to any patients. Respondents with a low knowledge score about this IUD, limited experience with IUD insertion, and non–obstetrician-gynecologist specialty had a more negative attitude toward this IUD and a lower willingness to recommend it. The top two reasons given for not recommending this IUD were fear of legal liability and a belief that the IUD was not medically safe.

No surveys of IUD use by US physicians have been

From the Department of Obstetrics and Gynecology, the Robert Wood Johnson Clinical Scholars Program, the Division of General Medicine, and the Department of Social Medicine, Cecil G. Sheps Center for Health Services Research, University of North Carolina at Chapel Hill, Chapel Hill, North Carolina.

This study was supported by the Robert Wood Johnson Clinical Scholars Program.

PII S0029-7844(01)01726-4

published in the peer-reviewed literature since the study by Kooiker and Scutchfield. Recent advances in IUD technology, exemplified by the entry of a new type of IUD into the US market—the Mirena levonorgestrel-releasing intrauterine system (Berlex Laboratories, Montville, NJ)—make our study particularly timely. Understanding how physicians feel about and use the IUD in practice is important in designing targeted educational programs for the current generation of physicians. We aimed to assess obstetrician-gynecologists' current use of the IUD in practice, their attitudes toward the IUD, and the factors they consider when selecting patients as IUD candidates. Further, we aimed to test the hypotheses that limited IUD training in residency, fear of litigation, and a belief in a strong causal link between IUDs and PID are associated with lower IUD use in practice.

MATERIALS AND METHODS

We designed a self-administered written survey to ascertain knowledge, attitudes, and practices among obstetrician-gynecologists with regard to the IUD, as well as demographic, training, and practice information. We constructed the sampling frame from the geographic listing of the membership directory of the ACOG, including all members in active practice (Fellow or Junior Fellow in Practice) who had a mailing address in the 50 United States or the District of Columbia and were not listed in the military. Starting at a random number, we selected a systematic sample of every 30th name, yielding a list of 811 names. We mailed the questionnaire to each of these obstetrician-gynecologists with a cover letter signed by the primary investigator and a postpaid return envelope. We conducted two mailings between July and September of 2000.

We tested for nonresponse bias by comparing respondents and nonrespondents on county-level demographic information linked by ZIP code to data in the Area Resource File (http://www.arfsys.com). Specifically, we looked at urban or rural practice location, county per capita income, several physician workforce variables, and variables reflecting the demographic structure of the communities served by the physicians (eg, total births and the percentage of women in the population). We had no demographic information on nonresponders from our original sampling frame, the ACOG Membership Directory. To address this issue, we compared a random sample of 100 responders and 100 nonresponders. We obtained information on sex and year of graduation from medical school in nonresponders by using the American Medical Association's Web-based physician directory (http://www.ama-assn.org). We added four to the year of medical school graduation to estimate the year of residency graduation. In analyses comparing responders with nonresponders, we used a two-sample Student t test for normally distributed continuous variables, the Wilcoxon rank-sum test for non-normal variables, and the Pearson χ^2 test for categorical variables.

We performed descriptive analyses of the main outcome of interest—the reported number of IUDs inserted in the past year—and demographic and attitudinal variables. We tested for associations between the reported number of IUDs inserted in the past year and demographic and attitudinal variables. Because the main outcome had a non-normal distribution, we performed both parametric and nonparametric tests; data are reported as means when results were similar. We used a two-sample Student t test and Spearman correlation for continuous independent variables and one-way analysis of variance and the Kruskal–Wallis test for nominal independent variables. We also created a scale of conservative-to-liberal IUD candidate selection by summing responses to five variables, measured on a Likert scale, that queried respondents' attitude toward parity, history of sexually transmitted disease (STD) and PID, marital status, and monogamy. We analyzed this scale in three categories: most conservative, moderate, and most liberal. This scale had an acceptable Cronbach α value of .70. We performed all analyses using Stata 6.0 (Stata Corporation, College Station, TX).

RESULTS

Of the 811 ACOG fellows to whom we mailed our survey, 400 returned complete responses, 17 had their mailing returned as undeliverable, and 26 returned invalid or incomplete responses, for a final response rate of 50%. This response rate is only slightly lower than the 52% response typical of large national physician surveys[13] and is higher than that reported in previous random surveys of general ACOG members, which averaged 40%.[14–22] Respondents and nonrespondents did not differ in county-level demographic information on income; rural or urban mix; or physician workforce, including obstetrician-gynecologist workforce. Respondents completed residency training more recently than nonrespondents did (mean year of graduation 1984 versus 1981, $P = .04$) and were more likely to be female (37% versus 26%, $P = .07$). The difference in sex distribution was not significant after adjustment for year of graduation, and the 3-year difference in the mean year of residency graduation was small.

Among respondents, 37% were female, the mean age was 47 years, and the mean year of residency completion was 1984 (Table 1). Seventy percent were in private practice and 14% were in academic departments, and

Table 1. Respondent Characteristics

Characteristic	Percentage or mean (range)
Female (%)	37
Male (%)	63
Age (y)	47 (31–73)
Year graduated from residency	1984 (1957–98)
Generalists (%)	83
Practice type (%)	
Private practice	70
Academic	14
Multispecialty	8
HMO	2
Other	6
Patients seen per week	88 (5–300)

HMO = health maintenance organization.

83% were generalists. They reported seeing an average of 88 patients in the office during a normal week. These characteristics are similar to those observed in recent surveys of ACOG members.[14–16,18,19,23]

Most respondents (80%) reported inserting IUDs in the last year, although 79% reported inserting 10 or fewer. The mean number of IUDs inserted in the past year was seven. Almost one third (32%) of respondents inserted 1 to 4 IUDs, 31% inserted 5 to 10 IUDs, and 17% inserted more than 10 IUDs. Respondents reported that they discussed a new or different contraceptive method with an average of 15% of their patients seen in the office. When asked to estimate the proportion of their patients using contraception whom they consider to be IUD candidates, half of respondents said 15% or less. During residency, most respondents reported inserting some IUDs; 13% inserted none, 38% inserted 1 to 20, 22% inserted 21 to 50, and 27% inserted more than 50.

Respondents had a remarkably positive attitude toward the IUD in general as a contraceptive method. Ninety-eight percent agreed that the Copper T380A IUD is effective, and 95% agreed that it is safe. Most (79%) agreed that they had enough time to counsel their patients on contraceptive options, and 64% believed that their patients were receptive to learning about the IUD. However, 20% percent agreed that the IUD was an abortifacient, and 16% agreed that it would lead to lawsuits against them.

Respondents restricted IUD candidates most tightly on the basis of monogamy and PID history. When presented with hypothetical characteristics of patients, 84% agreed that a woman in a nonmonogamous relationship should not have an IUD, and 81% agreed that a woman with a history of PID should not have an IUD. About two thirds of respondents did not recommend IUDs to nulliparous women or those with a history of STDs. Respondents were least restrictive about marital status; 31% agreed that a woman who is not married should not have an IUD. We then asked them how strongly different characteristics affect their selection of IUD candidates. Most (81%) were strongly affected by monogamy status. About two thirds (68%) responded that parity status had a strong effect on their decision, and a minority felt that marital status and the patient's education level affected their decision (40% and 30%, respectively).

We asked, "By what percent do you believe that a Copper T380A IUD increases the risk of PID over 10 years?" Twenty percent responded "zero," indicating that this minority does not believe in a causal relationship between IUDs and PID. Nearly 40% responded that the increased risk is 1% to 9%, 17% that the risk is increased 10%, and 12% that the increase in risk is greater than 10%. Thus, nearly one third of physicians responded that IUDs increase the risk of PID by 10% or more, indicating a strong belief in a long-term causal relationship.

We tested for associations between the reported number of IUDs inserted in the last year and demographic characteristics of respondents. Sex and geographic region were not associated with the reported number of IUDs inserted. Age and year of graduation of residency were highly correlated, and both were associated with IUD insertion. Younger and more recent graduates inserted more IUDs. Respondents aged 31 to 45 inserted a median of 5 IUDs, those aged 46 to 55 inserted a median of 4, and those aged 56 to 73 inserted a median of 3 ($P = .04$). Each practice type had similar reported median numbers of IUDs inserted, ranging from 2 to 5, except for one: Physicians in health maintenance organizations reported inserting a median of 10 IUDs, but only 8 respondents were in this group.

To test our main hypotheses about underuse of the IUD in the United States, we tested for associations between the reported number of IUDs inserted in the past year and residency training in IUDs, fear of litigation, and a belief in a causal link between IUDs and PID (Table 2). We found no association between the number of IUDs inserted during residency and the reported number inserted in the last year. We found a significant association between fear of litigation and reported number of IUDs inserted in the last year. Sixteen percent of respondents agreed that using the IUD in practice puts them at risk for litigation. These respondents reported inserting a mean of 4 IUDs, whereas those who disagreed inserted 10 ($P < .001$).

We found a significant overall association ($P = .004$) between a belief in a causal link between IUDs and PID

Table 2. Factors Associated With Reported Intrauterine Device Use in Clinical Practice

Factor	Respondents (%)	Mean IUDs inserted last year (*n*)	*P**
Number of IUDs inserted during residency			
None	13	7	.49
1–20	38	6	
21–50	22	8	
51–100	13	6	
>100	14	8	
IUD use leads to litigation			
Agreed	16	4	<.001
Neutral	23	6	
Disagreed	61	10	
Percentage increased risk of PID due to IUDs			
0%	20	9	.004
1–5%	36	7	
>5%	32	7	
Unsure/missing	12	3	
IUD candidate selection criteria			
Conservative	35	4	<.001
Moderate	27	7	
Liberal	38	9	

IUD = intrauterine device; PID = pelvic inflammatory disease.
* Overall significance from one-way analysis of variance.

and reported IUD use in practice when we asked the question, "By what percent do you believe that a Copper T380A IUD increases the risk of PID over 10 years of use?" Physicians who responded "zero" had a higher mean number of IUDs inserted in the past year than did those who responded ">1%" (nine versus seven, two-sample Student *t* test $P = .008$).

We developed a score of conservative-to-liberal IUD candidate selection behavior using five questions on patient characteristics (nulliparity, STD and PID history, marital status, and monogamy). On the basis of score, we categorized respondents as conservative, moderate, or liberal. Reported use of the IUD in practice was associated with this score. The conservative group reported inserting fewer IUDs in the past year than the liberal group (four versus nine, $P < .001$).

DISCUSSION

Most obstetrician-gynecologists insert IUDs in clinical practice, but they report inserting few per year. Although attitudes toward the safety and effectiveness of the IUD are very positive, most respondents believe that a long-term causal relationship exists between the modern copper IUD and PID. Twenty-nine percent of respondents considered this increased risk to be 10% or greater.

The evidence argues against such a conclusion. A transient sixfold increased risk exists for 21 days after insertion, after which the rate of PID decreases to 0.059 per 100 woman-years of use with the Copper T380A device.[6] The evidence does not demonstrate a long-term risk of PID definitively attributable to the IUD.[6] Instead, sexual behavior and resultant exposure to chlamydia and gonorrhea produce the largest attributable risk for PID[24] (Shelton JD. Risk of clinical pelvic inflammatory disease attributable to an intrauterine device [Research Letter]. Lancet 2001;357:443). No study has answered the question of whether the risk of progression to PID is greater or lesser in a woman with cervicitis and a modern copper IUD than in a woman with cervicitis but no IUD.[8] Nor does evidence indicate that having an IUD in place will worsen a case of PID. In contrast to current evidence that the IUD does not cause PID in the long term, manufacturer recommendations (Paragard® T380A Copper Contraceptive. Prescribing information. Raritan, NJ: Ortho Pharmaceutical Corporation, 1997) list any lifetime history of PID as a contraindication to use of a copper IUD. Similarly, the ACOG Technical Bulletin on IUDs[9] describes the ideal IUD candidate as having no history of PID.

Our survey supports the hypothesis that a fear of litigation limits use of the IUD in practice among obstetrician-gynecologists. Fortunately, a majority of respondents believed that IUDs do not lead to lawsuits, an opinion supported by reviews of physician litigation experience.[12] Our results do not support the hypothesis that the number of IUDs inserted in residency is associ-

ated with current reported use of the IUD in clinical practice. Rather, the association of a respondent's conservative-to-liberal score in patient selection with IUD use may indicate that the teaching of candidate selection drives physicians' future use of the IUD.

Our survey has several limitations. Because it is cross-sectional, we can find associations but not cause-and-effect relationships. Thus, respondents may have developed more liberal criteria for IUD candidate selection after inserting many IUDs and becoming comfortable with the low rate of complications rather than by developing more liberal criteria that then cause them to insert higher numbers. We also relied on respondents' recall of the number of IUDs inserted in the past year and during residency. Our results may not be generalizable to all obstetrician-gynecologists in the United States. Because respondents graduated slightly more recently from residency than did nonrespondents, our survey may overestimate the use of IUDs by practicing obstetrician-gynecologists. Finally, our survey addresses neither the decision to use an IUD from the woman's perspective nor issues of insurance coverage for contraceptives.

Our findings nevertheless have important implications for physician training and education. Because most obstetrician-gynecologists are inserting few IUDs, educational programs should target these physicians to expand their IUD use. Such programs should highlight the evidence for IUD safety and the rarity of litigation. The number of IUDs inserted in residency may be less important than the development of less conservative, more evidence-based criteria for selecting IUD candidates.

The IUD is a safe and effective method of contraception. Its broader use in the United States is limited, not because obstetrician-gynecologists believe that the IUD is ineffective, or that they lack skill or knowledge in its use, but because of a persistent belief that IUDs cause PID, which results in lawsuits. The evidence does not support these fears about modern devices, and such tight restrictions fail to weigh the competing risk of unintended pregnancy from use of less effective methods. Failure to weigh this risk has profound public health implications in a country in which 49% of pregnancies are unintended and 53% of unintended pregnancies happen after contraceptive failure or misuse.[25]

Many women could safely use the IUD but are not offered this contraceptive method because physicians' selection of IUD candidates is unduly restrictive. Educating physicians about the safety of IUDs may expand their use of IUDs. More women would then be offered a method of convenient, safe, and highly effective long-term contraception.

REFERENCES

1. Rinehart W. IUDs–An update. Popul Rep B 1995:1–35.
2. Trussell J, Vaughan B. Contraceptive failure, method-related discontinuation and resumption of use: Results from the 1995 National Survey of Family Growth. Fam Plann Perspect 1999;31:64–72, 93.
3. Piccinino LJ, Mosher WD. Trends in contraceptive use in the United States: 1982-1995. Fam Plann Perspect 1998; 30:4–10, 46.
4. Westoff CF, Jones EF. Contraception and sterilization in the United States, 1965-1975. Fam Plann Perspect 1977;9: 153–7.
5. Forrest JD. The end of IUD marketing in the United States: What does it mean for American women? Fam Plann Perspect 1986;18:52–7.
6. Farley TM, Rosenberg MJ, Rowe PJ, Chen JH, Meirik O. Intrauterine devices and pelvic inflammatory disease: An international perspective. Lancet 1992;339:785–8.
7. Kessel E. Pelvic inflammatory disease with intrauterine device use: A reassessment. Fertil Steril 1989;51:1–11.
8. Grimes DA. Intrauterine device and upper-genital-tract infection. Lancet 2000;356:1013–9.
9. American College of Obstetricians and Gynecologists. The intrauterine device. ACOG technical bulletin no. 164. Washington, DC: American College of Obstetricians and Gynecologists, 1992.
10. Westhoff C. The IUD in evolution. Obstet Gynecol Surv 1996;51:S20–4.
11. Kooiker CH, Scutchfield FD. Barriers to prescribing the Copper T 380A intrauterine device by physicians. West J Med 1990;153:279–82.
12. Westhoff C, Marks F, Rosenfield A, Roig A, Nicholas A. Clinical and legal factors influencing IUD use in the United States. In: Bardin C, Mishell D, eds. Proceedings from the Fourth International Conference on IUDs. Boston: Butterworth-Heinemann, 1994:100–8.
13. Cummings SM, Savitz LA, Konrad T. Reported response rates to mailed physician questionnaires. Health Serv Res 2001;36:1347–55.
14. Erickson K, Schmidt L, Santesso DL, Schulkin J, Gregory K, Hobel C. Obstetrician-gynecologists' knowledge and training about antenatal corticosteroids. Obstet Gynecol 2001;97:140–6.
15. Power ML, Holzman GB, Schulkin J. Knowledge and clinical practice regarding folic acid among obstetrician-gynecologists. Obstet Gynecol 2000;95:895–8.
16. Diekman ST, Floyd RL, Decoufle P, Schulkin J, Ebrahim SH, Sokol RJ. A survey of obstetrician-gynecologists on their patients' alcohol use during pregnancy. Obstet Gynecol 2000;95:756–63.
17. Wilkins-Haug L, Hill LD, Power ML, Holzman GB, Schulkin J. Gynecologists' training, knowledge, and experiences in genetics: A survey. Obstet Gynecol 2000;95: 421–4.

18. Power ML, Holzman GB, Schulkin J. Knowledge and clinical practice regarding calcium nutrition among obstetrician-gynecologists. Obstet Gynecol 1999;94:421–6.
19. Wilkins-Haug L, Hill L, Schmidt L, Holzman GB, Schulkin J. Genetics in obstetricians' offices: A survey study. Obstet Gynecol 1999;93:642–7.
20. Horan DL, Chapin J, Klein L, Schmidt LA, Schulkin J. Domestic violence screening practices of obstetrician-gynecologists. Obstet Gynecol 1998;92:785–9.
21. Scroggs JA, Griffin LP, Bayerl M, Schulkin J. Obstetrician-gynecologists as primary care physicians: The perspectives of health maintenance organization medical directors and obstetrician-gynecologists. Obstet Gynecol 1997;90: 291–5.
22. Schmidt LA, Greenberg BD, Holzman GB, Schulkin J. Treatment of depression by obstetrician-gynecologists: A survey study. Obstet Gynecol 1997;90:296–300.
23. American College of Obstetricians and Gynecologists. Profile of Ob-gyn Practice, 1991-1998. Washington, DC: American College of Obstetricians and Gynecologists, 2000.
24. Lee NC, Rubin GL, Borucki R. The intrauterine device and pelvic inflammatory disease revisited: New results from the Women's Health Study. Obstet Gynecol 1988; 72:1–6.
25. Henshaw SK. Unintended pregnancy in the United States. Fam Plann Perspect 1998;30:24–9.

Address reprint requests to: Nancy L. Stanwood, MD, MPH, Department of Obstetrics and Gynecology, University of Rochester Medical Center, Box 668, Room 2-4446, 601 Elmwood Avenue, Rochester, NY 14642-8668; E-mail: nancy_stanwood@urmc.rochester.edu.

Received May 31, 2001. Received in revised form October 22, 2001. Accepted October 25, 2001.

Occupational Employment Statistics

Occupational Employment and Wages, May 2017

29-1062 Family and General Practitioners

Physicians who diagnose, treat, and help prevent diseases and injuries that commonly occur in the general population. May refer patients to specialists when needed for further diagnosis or treatment.

National estimates for this occupation
Industry profile for this occupation
Geographic profile for this occupation

National estimates for this occupation: Top

Employment estimate and mean wage estimates for this occupation:

Employment (1)	Employment RSE (3)	Mean hourly wage	Mean annual wage (2)	Wage RSE (3)
126,440	2.0 %	$100.27	$208,560	1.0 %

Percentile wage estimates for this occupation:

Percentile	10%	25%	50% (Median)	75%	90%
Hourly Wage	$35.21	$66.39	$95.55	(5)	(5)
Annual Wage (2)	$73,240	$138,100	$198,740	(5)	(5)

Industry profile for this occupation: Top

Industries with the highest published employment and wages for this occupation are provided. For a list of all industries with employment in this occupation, see the Create Customized Tables function.

Industries with the highest levels of employment in this occupation:

Industry	Employment (1)	Percent of industry employment	Hourly mean wage	Annual mean wage (2)
Offices of Physicians	83,320	3.27	$102.73	$213,670
General Medical and Surgical Hospitals	22,620	0.41	$97.66	$203,140
Outpatient Care Centers	9,440	1.07	$102.84	$213,900
Colleges, Universities, and Professional Schools	3,820	0.13	$57.35	$119,280
Local Government, excluding schools and hospitals (OES Designation)	1,510	0.03	$98.21	$204,280

Industries with the highest concentration of employment in this occupation:

Industry	Employment (1)	Percent of industry employment	Hourly mean wage	Annual mean wage (2)
Offices of Physicians	83,320	3.27	$102.73	$213,670
Outpatient Care Centers	9,440	1.07	$102.84	$213,900
General Medical and Surgical Hospitals	22,620	0.41	$97.66	$203,140
Psychiatric and Substance Abuse Hospitals	560	0.24	$95.97	$199,620
Colleges, Universities, and Professional Schools	3,820	0.13	$57.35	$119,280

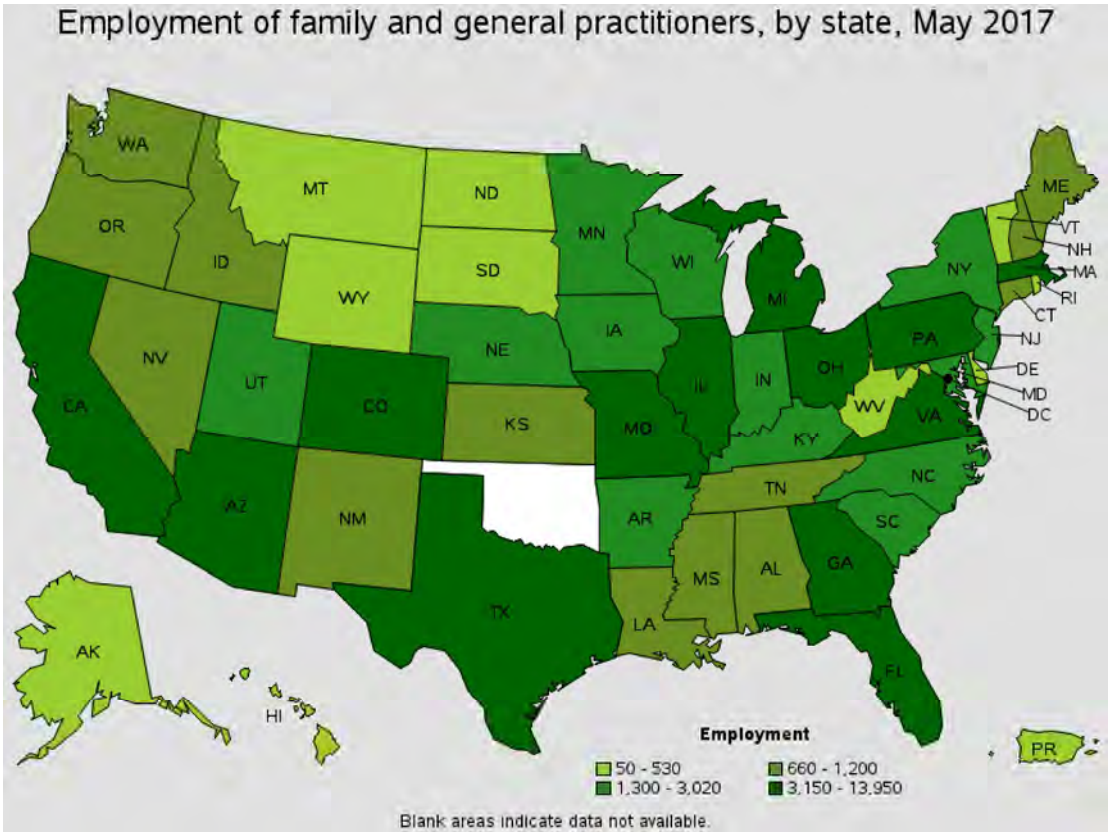
Top paying industries for this occupation:

Industry	Employment (1)	Percent of industry employment	Hourly mean wage	Annual mean wage (2)
Specialty (except Psychiatric and Substance Abuse) Hospitals	190	0.07	$112.63	$234,270
Medical and Diagnostic Laboratories	90	0.03	$112.48	$233,950

Residential Intellectual and Developmental Disability, Mental Health, and Substance Abuse Facilities	50	0.01	$112.11	$233,200
Management of Companies and Enterprises	150	0.01	$111.78	$232,510
Management, Scientific, and Technical Consulting Services	90	0.01	$106.36	$221,220

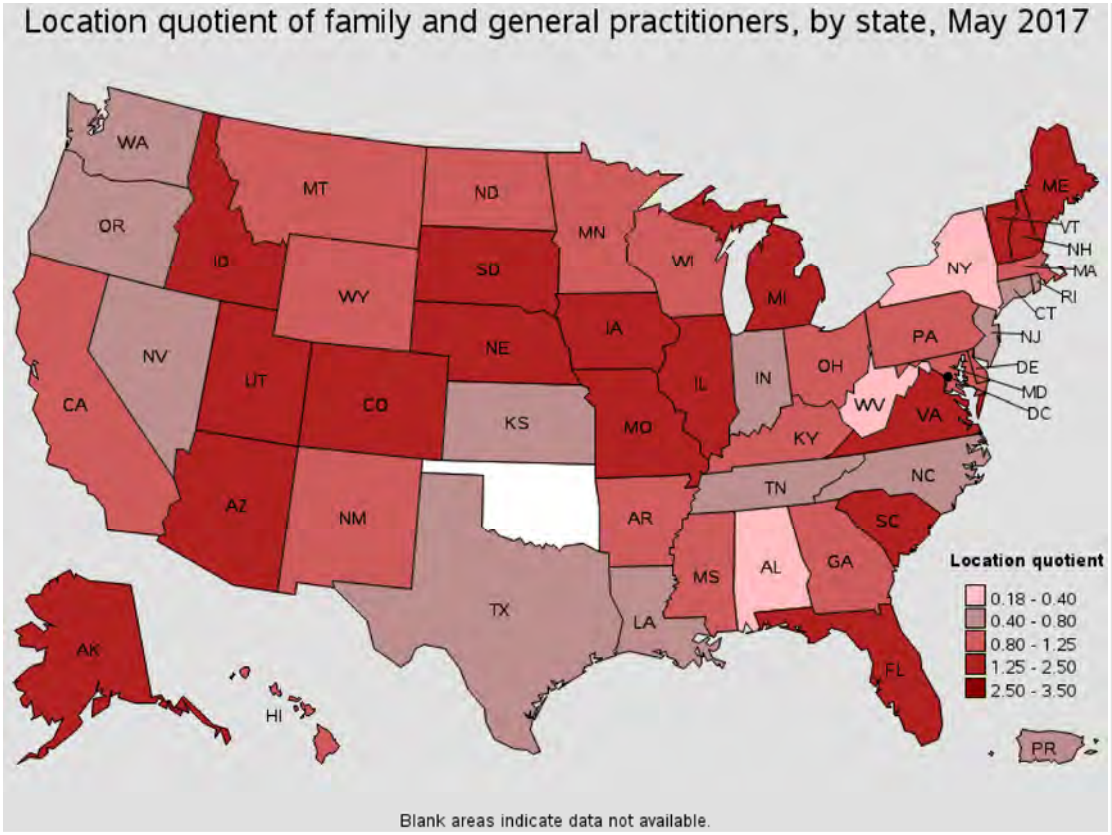
Geographic profile for this occupation: Top

States and areas with the highest published employment, location quotients, and wages for this occupation are provided. For a list of all areas with employment in this occupation, see the Create Customized Tables function.



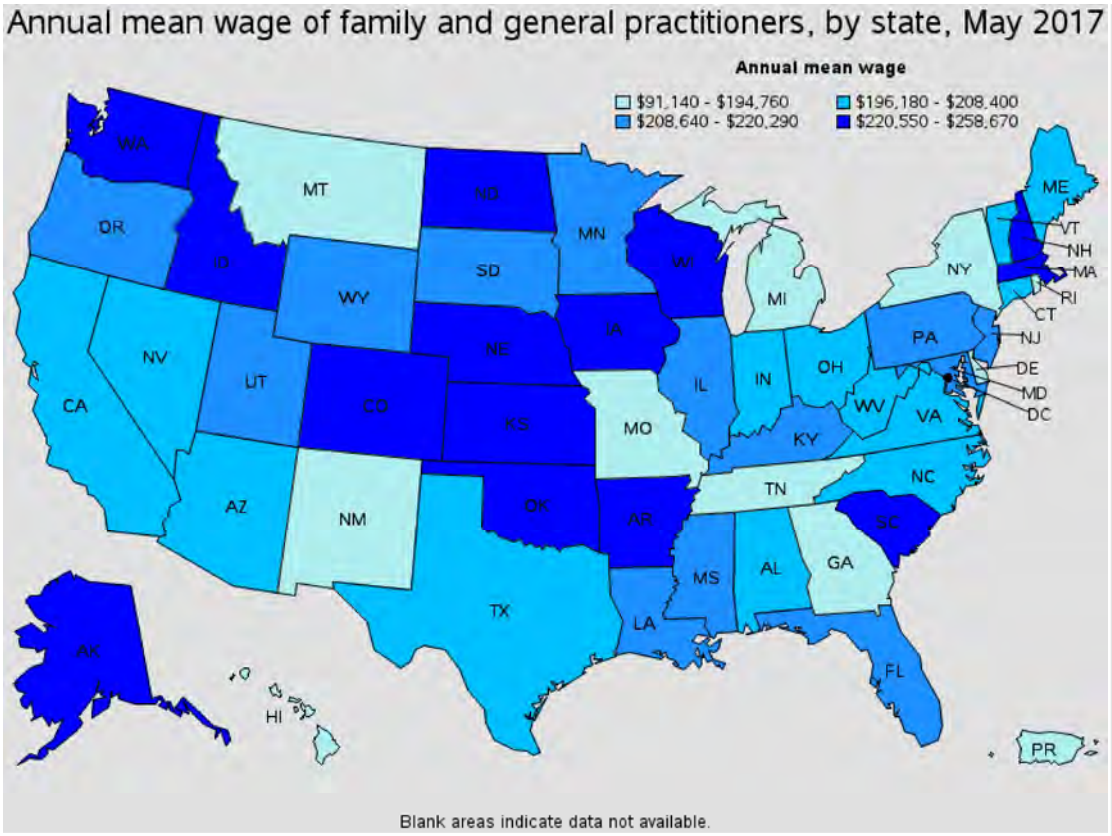
States with the highest employment level in this occupation:

State	Employment (1)	Employment per thousand jobs	Location quotient (9)	Hourly mean wage	Annual mean wage (2)
California	13,950	0.84	0.94	$94.32	$196,180
Florida	12,710	1.51	1.70	$103.51	$215,300
Illinois	9,160	1.54	1.74	$105.91	$220,290
Texas	7,910	0.67	0.75	$98.39	$204,660
Missouri	5,280	1.89	2.14	$83.14	$172,940



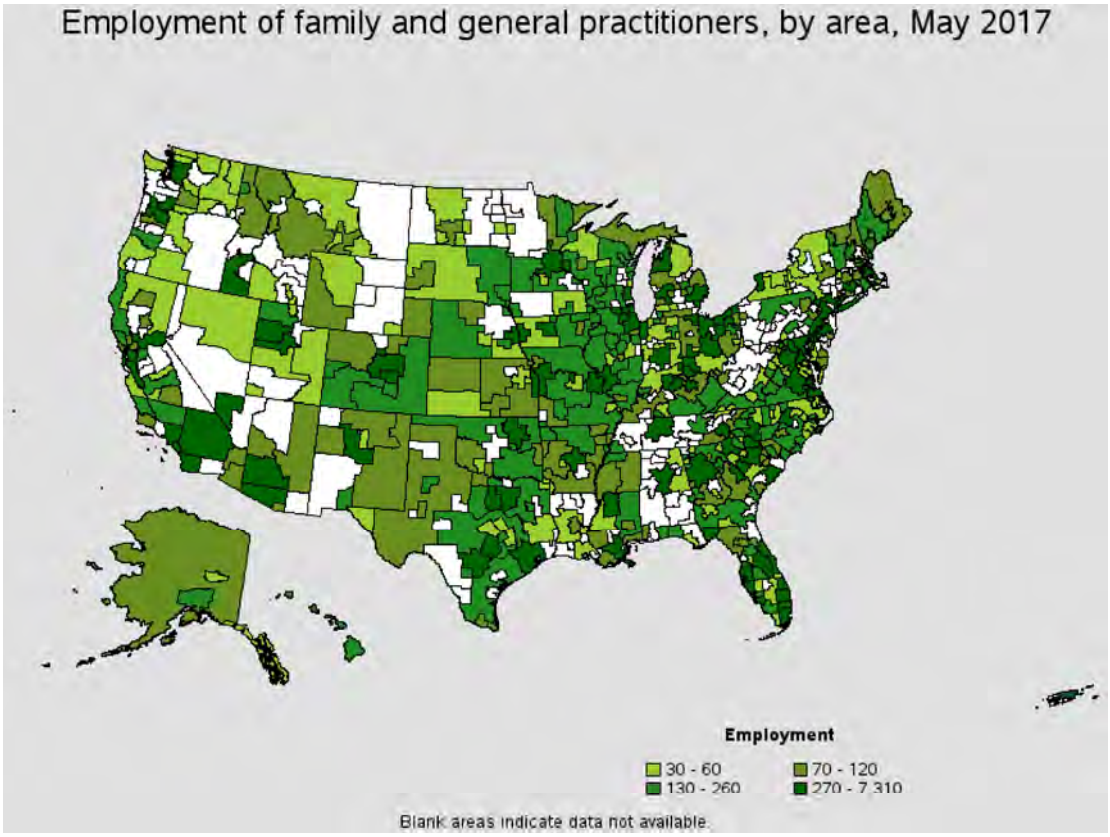
States with the highest concentration of jobs and location quotients in this occupation:

State	Employment (1)	Employment per thousand jobs	Location quotient (9)	Hourly mean wage	Annual mean wage (2)
Missouri	5,280	1.89	2.14	$83.14	$172,940
Nebraska	1,680	1.73	1.95	$109.15	$227,020
Illinois	9,160	1.54	1.74	$105.91	$220,290
Idaho	1,060	1.54	1.73	$106.03	$220,550
Florida	12,710	1.51	1.70	$103.51	$215,300



Top paying States for this occupation:

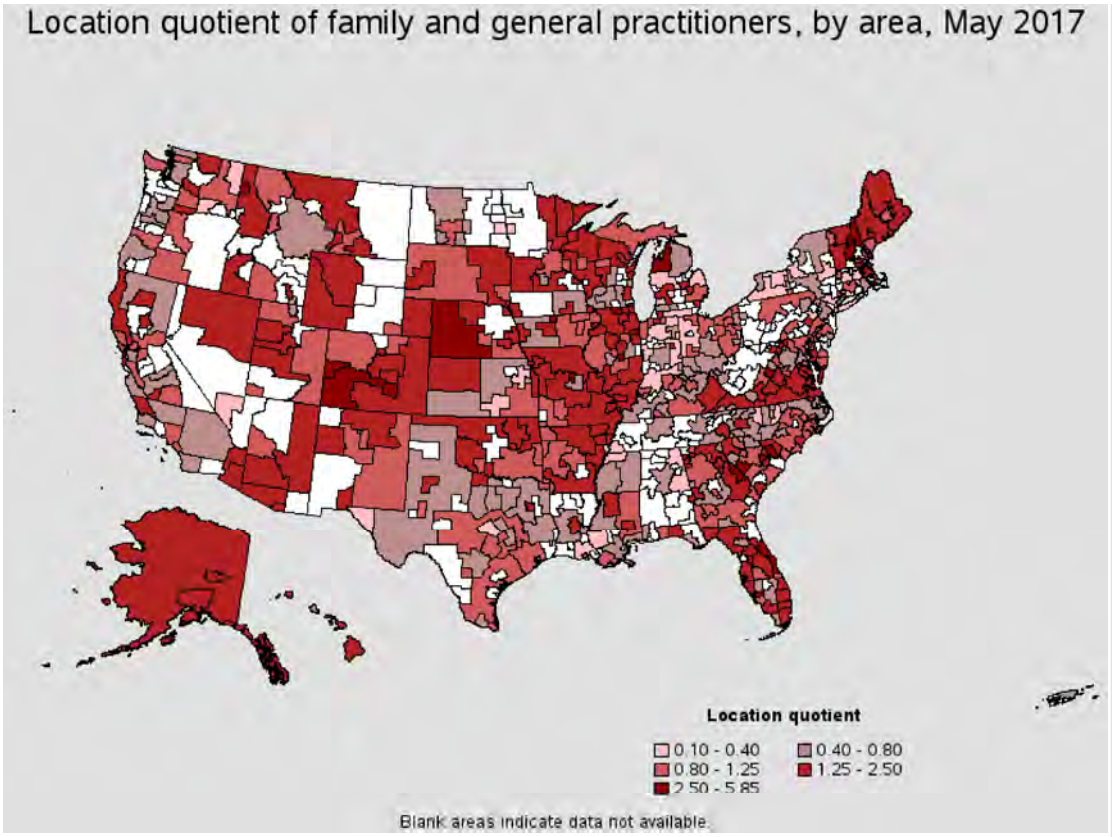
State	Employment (1)	Employment per thousand jobs	Location quotient (9)	Hourly mean wage	Annual mean wage (2)
New Hampshire	910	1.41	1.58	$124.36	$258,670
Wisconsin	2,420	0.86	0.96	$116.51	$242,340
Iowa	1,840	1.20	1.35	$116.23	$241,760
South Carolina	2,670	1.33	1.50	$112.70	$234,420
Washington	1,200	0.38	0.42	$110.73	$230,330



<

Metropolitan areas with the highest employment level in this occupation:

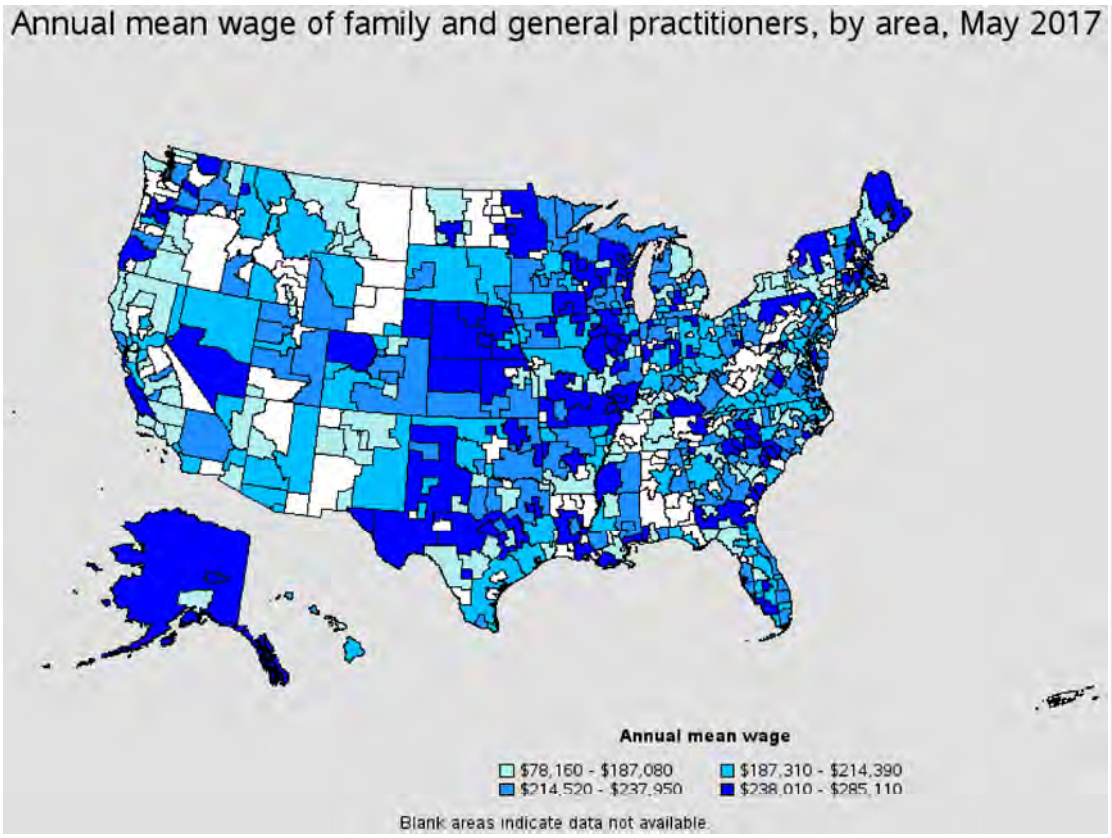
Metropolitan area	Employment (1)	Employment per thousand jobs	Location quotient (9)	Hourly mean wage	Annual mean wage (2)
Chicago-Naperville-Arlington Heights, IL Metropolitan Division	6,200	1.69	1.91	$101.36	$210,820
Los Angeles-Long Beach-Glendale, CA Metropolitan Division	4,440	1.00	1.13	$86.25	$179,400
Houston-The Woodlands-Sugar Land, TX	2,760	0.94	1.06	$102.55	$213,300
St. Louis, MO-IL	2,570	1.89	2.13	$68.11	$141,670
Boston-Cambridge-Newton, MA NECTA Division	2,500	1.36	1.53	$104.78	$217,940
Phoenix-Mesa-Scottsdale, AZ	2,330	1.18	1.33	$96.32	$200,340
Atlanta-Sandy Springs-Roswell, GA	1,880	0.72	0.81	$97.17	$202,100
Tampa-St. Petersburg-Clearwater, FL	1,850	1.44	1.63	$108.25	$225,160
Washington-Arlington-Alexandria, DC-VA-MD-WV Metropolitan Division	1,750	0.69	0.78	$82.65	$171,900
Minneapolis-St. Paul-Bloomington, MN-WI	1,740	0.90	1.01	$103.07	$214,390



<

Metropolitan areas with the highest concentration of jobs and location quotients in this occupation:

Metropolitan area	Employment (1)	Employment per thousand jobs	Location quotient (9)	Hourly mean wage	Annual mean wage (2)
Columbia, MO	480	5.19	5.85	$115.06	$239,320
Punta Gorda, FL	170	3.60	4.06	$93.56	$194,600
Coeur d'Alene, ID	190	3.20	3.60	$128.54	$267,370
Staunton-Waynesboro, VA	150	3.17	3.57	$114.01	$237,130
New Bedford, MA	200	3.05	3.44	$130.36	$271,140
Sumter, SC	110	3.01	3.39	$124.85	$259,690
Augusta-Richmond County, GA-SC	630	2.87	3.23	$60.19	$125,190
Florence, SC	240	2.85	3.21	$118.12	$245,680
Ames, IA	130	2.85	3.22	$132.48	$275,550
Palm Bay-Melbourne-Titusville, FL	560	2.72	3.06	$108.50	$225,690



<

Top paying metropolitan areas for this occupation:

Metropolitan area	Employment (1)	Employment per thousand jobs	Location quotient (9)	Hourly mean wage	Annual mean wage (2)
The Villages, FL	30	1.28	1.45	(5)	(5)
St. Joseph, MO-KS	100	1.90	2.15	(5)	(5)
Enid, OK	(8)	(8)	(8)	$137.07	$285,110
Longview, TX	(8)	(8)	(8)	$134.69	$280,150
Hilton Head Island-Bluffton-Beaufort, SC	120	1.67	1.88	$133.82	$278,350
Sebring, FL	(8)	(8)	(8)	$133.66	$278,020
Appleton, WI	(8)	(8)	(8)	$133.59	$277,870
Kankakee, IL	110	2.55	2.87	$133.24	$277,140
Portsmouth, NH-ME	130	1.40	1.58	$133.02	$276,680
Knoxville, TN	140	0.37	0.42	$132.54	$275,680

Nonmetropolitan areas with the highest employment in this occupation:

Nonmetropolitan area	Employment (1)	Employment per thousand jobs	Location quotient (9)	Hourly mean wage	Annual mean wage (2)
North Northeastern Ohio non-metropolitan area (non-contiguous)	330	0.99	1.12	$103.05	$214,350
Northwest Lower Peninsula of Michigan nonmetropolitan area	270	2.23	2.51	$107.10	$222,770
Southeast Missouri nonmetropolitan area	260	1.60	1.80	$115.81	$240,880
Central Missouri nonmetropolitan area	260	1.61	1.82	$115.81	$240,880
Central Nebraska nonmetropolitan area	250	2.35	2.65	$127.51	$265,220

Nonmetropolitan areas with the highest concentration of jobs and location quotients in this occupation:

Nonmetropolitan area	Employment (1)	Employment per thousand jobs	Location quotient (9)	Hourly mean wage	Annual mean wage (2)
West Central New Hampshire nonmetropolitan area	200	3.06	3.45	$127.98	$266,210
Northern New Hampshire nonmetropolitan area	90	2.54	2.87	$132.12	$274,810
Southwest Colorado nonmetropolitan area	240	2.50	2.82	$93.24	$193,940
Central Nebraska nonmetropolitan area	250	2.35	2.65	$127.51	$265,220
Northwest Lower Peninsula of Michigan nonmetropolitan area	270	2.23	2.51	$107.10	$222,770

Top paying nonmetropolitan areas for this occupation:

Nonmetropolitan area	Employment (1)	Employment per thousand jobs	Location quotient (9)	Hourly mean wage	Annual mean wage (2)
West Texas Region of Texas nonmetropolitan area	80	0.39	0.44	$134.51	$279,780
South Georgia nonmetropolitan area	150	0.83	0.93	$133.62	$277,930
Balance of Alaska nonmetropolitan area	120	1.66	1.87	$133.23	$277,110
Northeast Nebraska nonmetropolitan area	(8)	(8)	(8)	$132.66	$275,930
Northern New Hampshire nonmetropolitan area	90	2.54	2.87	$132.12	$274,810

About May 2017 National, State, Metropolitan, and Nonmetropolitan Area Occupational Employment and Wage Estimates

These estimates are calculated with data collected from employers in all industry sectors, all metropolitan and nonmetropolitan areas, and all states and the District of Columbia. The top employment and wage figures are provided above. The complete list is available in the downloadable XLS files.

The percentile wage estimate is the value of a wage below which a certain percent of workers fall. The median wage is the 50th percentile wage estimate--50 percent of workers earn less than the median and 50 percent of workers earn more than the median. More about percentile wages.

(1) Estimates for detailed occupations do not sum to the totals because the totals include occupations not shown separately. Estimates do not include self-employed workers.

(2) Annual wages have been calculated by multiplying the hourly mean wage by a "year-round, full-time" hours figure of 2,080 hours; for those occupations where there is not an hourly wage published, the annual wage has been directly calculated from the reported survey data.

(3) The relative standard error (RSE) is a measure of the reliability of a survey statistic. The smaller the relative standard error, the more precise the estimate.

(5) This wage is equal to or greater than $100.00 per hour or $208,000 per year.

(8) Estimate not released.

(9) The location quotient is the ratio of the area concentration of occupational employment to the national average concentration. A location quotient greater than one indicates the occupation has a higher share of employment than average, and a location quotient less than one indicates the occupation is less prevalent in the area than average.

Other OES estimates and related information:

May 2017 National Occupational Employment and Wage Estimates

May 2017 State Occupational Employment and Wage Estimates

May 2017 Metropolitan and Nonmetropolitan Area Occupational Employment and Wage Estimates

May 2017 National Industry-Specific Occupational Employment and Wage Estimates

May 2017 Occupation Profiles

Technical Notes

Last Modified Date: March 30, 2018

U.S. Bureau of Labor Statistics | Division of Occupational Employment Statistics, PSB Suite 2135, 2 Massachusetts Avenue, NE Washington, DC 20212-0001
www.bls.gov/OES | Telephone: 1-202-691-6569 | Contact OES

Occupational Employment Statistics

BROWSE OES
OES HOME
OES OVERVIEW
OES NEWS RELEASES
OES DATA
OES CHARTS
OES MAPS
OES PUBLICATIONS
OES DATABASES
OES FAQS
CONTACT OES

SEARCH OES
Go

OES TOPICS
RESPONDENTS
DOCUMENTATION
SPECIAL NOTICES
RELATED LINKS

Occupational Employment and Wages, May 2017

29-1141 Registered Nurses

Assess patient health problems and needs, develop and implement nursing care plans, and maintain medical records. Administer nursing care to ill, injured, convalescent, or disabled patients. May advise patients on health maintenance and disease prevention or provide case management. Licensing or registration required. Includes Clinical Nurse Specialists. Excludes "Nurse Anesthetists" (29-1151), "Nurse Midwives" (29-1161), and "Nurse Practitioners" (29-1171).

National estimates for this occupation
Industry profile for this occupation
Geographic profile for this occupation

National estimates for this occupation: Top

Employment estimate and mean wage estimates for this occupation:

Employment (1)	Employment RSE (3)	Mean hourly wage	Mean annual wage (2)	Wage RSE (3)
2,906,840	0.5 %	$35.36	$73,550	0.2 %

Percentile wage estimates for this occupation:

Percentile	10%	25%	50% (Median)	75%	90%
Hourly Wage	$23.41	$27.57	$33.65	$41.33	$50.05
Annual Wage (2)	$48,690	$57,340	$70,000	$85,960	$104,100

Industry profile for this occupation: Top

Industries with the highest published employment and wages for this occupation are provided. For a list of all industries with employment in this occupation, see the Create Customized Tables function.

Industries with the highest levels of employment in this occupation:

Industry	Employment (1)	Percent of industry employment	Hourly mean wage	Annual mean wage (2)
General Medical and Surgical Hospitals	1,685,820	30.64	$36.45	$75,820
Offices of Physicians	196,040	7.69	$32.16	$66,890
Home Health Care Services	179,310	12.84	$33.77	$70,230
Nursing Care Facilities (Skilled Nursing Facilities)	155,450	9.59	$31.59	$65,710
Outpatient Care Centers	132,070	15.00	$36.39	$75,680

Industries with the highest concentration of employment in this occupation:

Industry	Employment (1)	Percent of industry employment	Hourly mean wage	Annual mean wage (2)
General Medical and Surgical Hospitals	1,685,820	30.64	$36.45	$75,820
Specialty (except Psychiatric and Substance Abuse) Hospitals	62,310	23.98	$37.16	$77,290
Psychiatric and Substance Abuse Hospitals	39,100	16.33	$34.27	$71,290
Outpatient Care Centers	132,070	15.00	$36.39	$75,680
Home Health Care Services	179,310	12.84	$33.77	$70,230

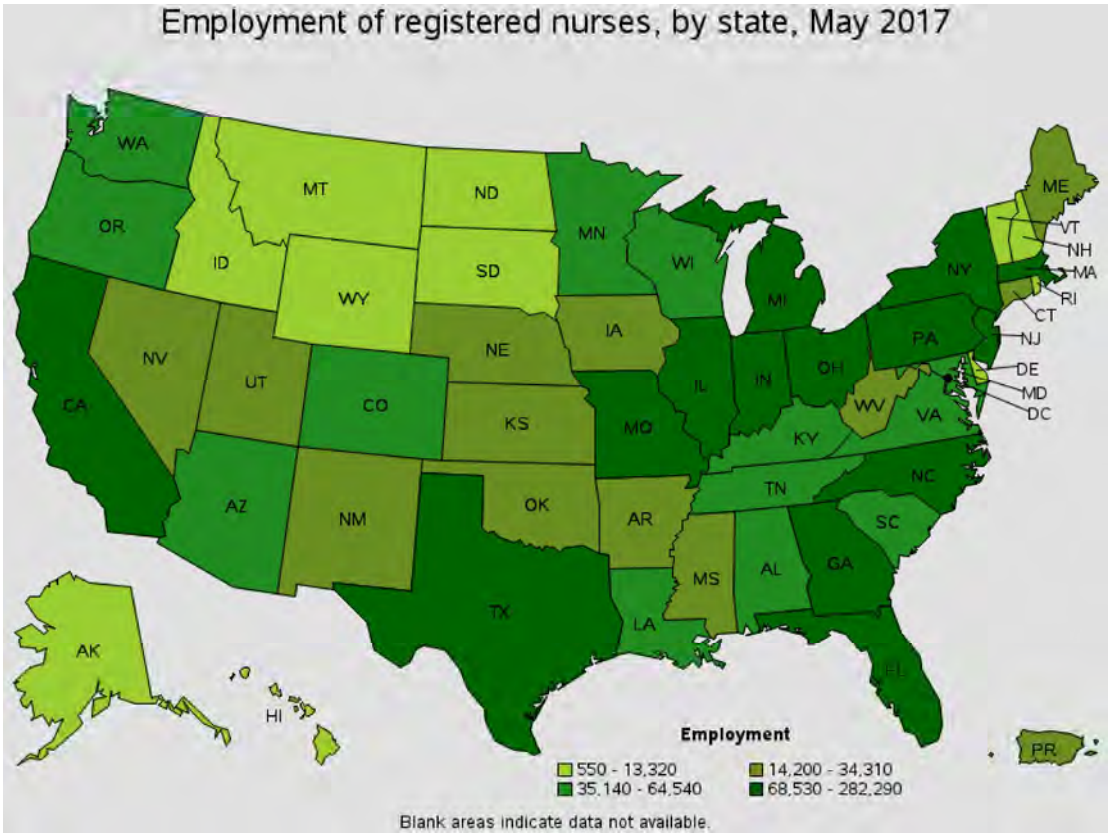
Top paying industries for this occupation:

Industry	Employment (1)	Percent of industry employment	Hourly mean wage	Annual mean wage (2)

Pharmaceutical and Medicine Manufacturing	210	0.07	$43.51	$90,510
Accounting, Tax Preparation, Bookkeeping, and Payroll Services	940	0.10	$42.81	$89,050
Federal Executive Branch (OES Designation)	79,220	3.91	$41.84	$87,030
Business Schools and Computer and Management Training	70	0.10	$41.82	$86,990
Wholesale Electronic Markets and Agents and Brokers	240	0.03	$39.99	$83,190

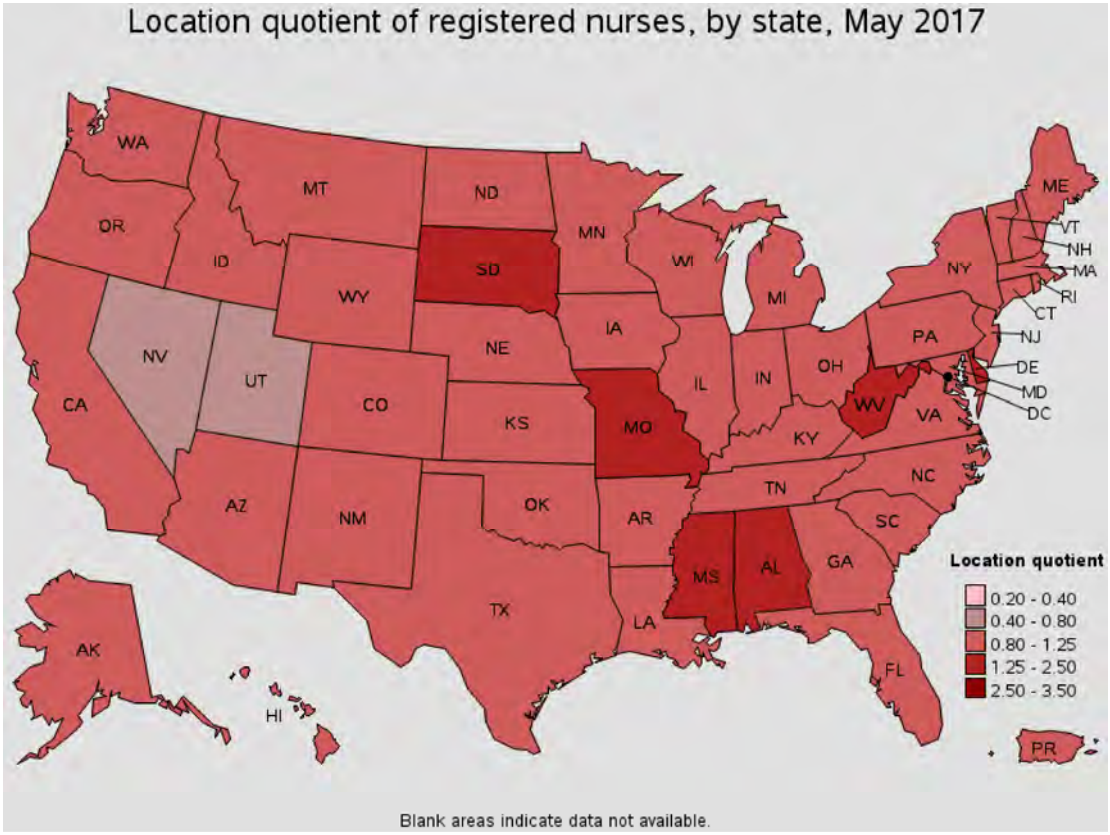
Geographic profile for this occupation: Top

States and areas with the highest published employment, location quotients, and wages for this occupation are provided. For a list of all areas with employment in this occupation, see the Create Customized Tables function.



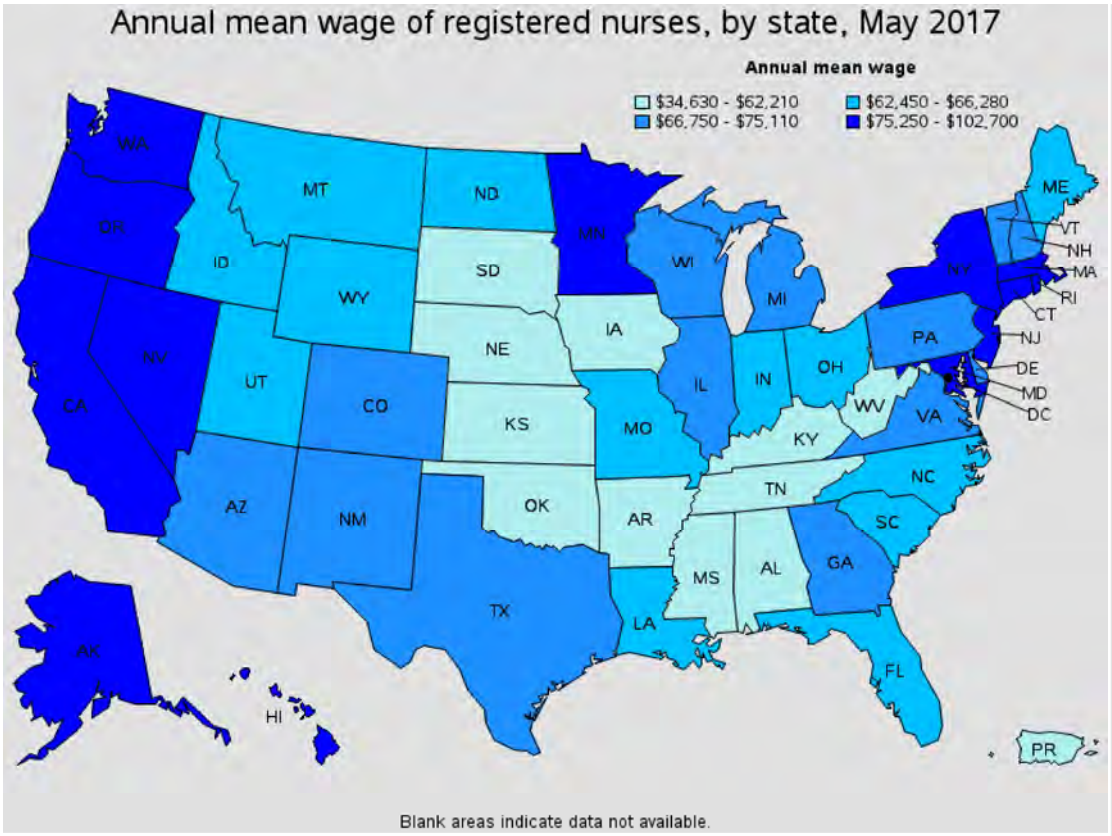
States with the highest employment level in this occupation:

State	Employment (1)	Employment per thousand jobs	Location quotient (9)	Hourly mean wage	Annual mean wage (2)
California	282,290	16.91	0.83	$49.37	$102,700
Texas	212,230	17.85	0.88	$34.65	$72,070
New York	180,170	19.57	0.96	$40.12	$83,450
Florida	178,330	21.18	1.04	$31.20	$64,890
Pennsylvania	143,130	24.76	1.21	$33.57	$69,820



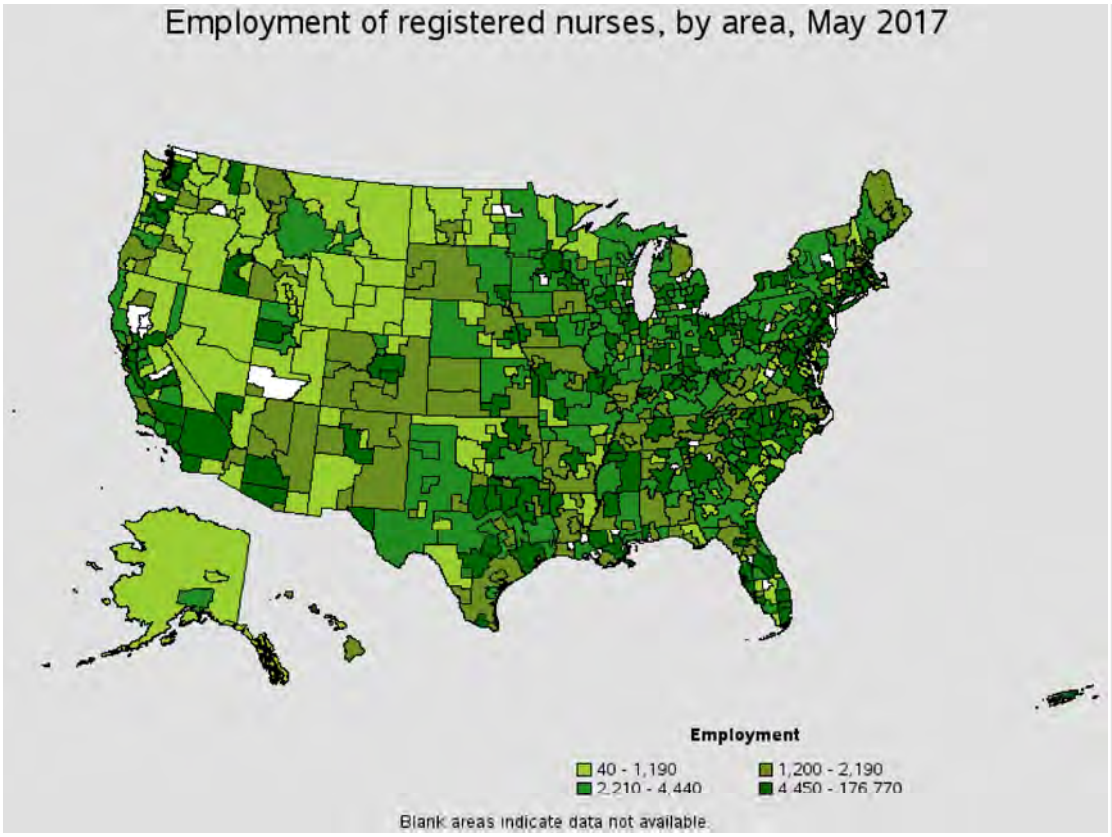
States with the highest concentration of jobs and location quotients in this occupation:

State	Employment (1)	Employment per thousand jobs	Location quotient (9)	Hourly mean wage	Annual mean wage (2)
South Dakota	12,530	29.87	1.47	$27.41	$57,010
West Virginia	20,410	29.65	1.45	$29.03	$60,380
Delaware	11,620	26.25	1.29	$35.18	$73,180
Missouri	72,090	25.85	1.27	$30.43	$63,300
Mississippi	28,760	25.69	1.26	$27.74	$57,700



Top paying States for this occupation:

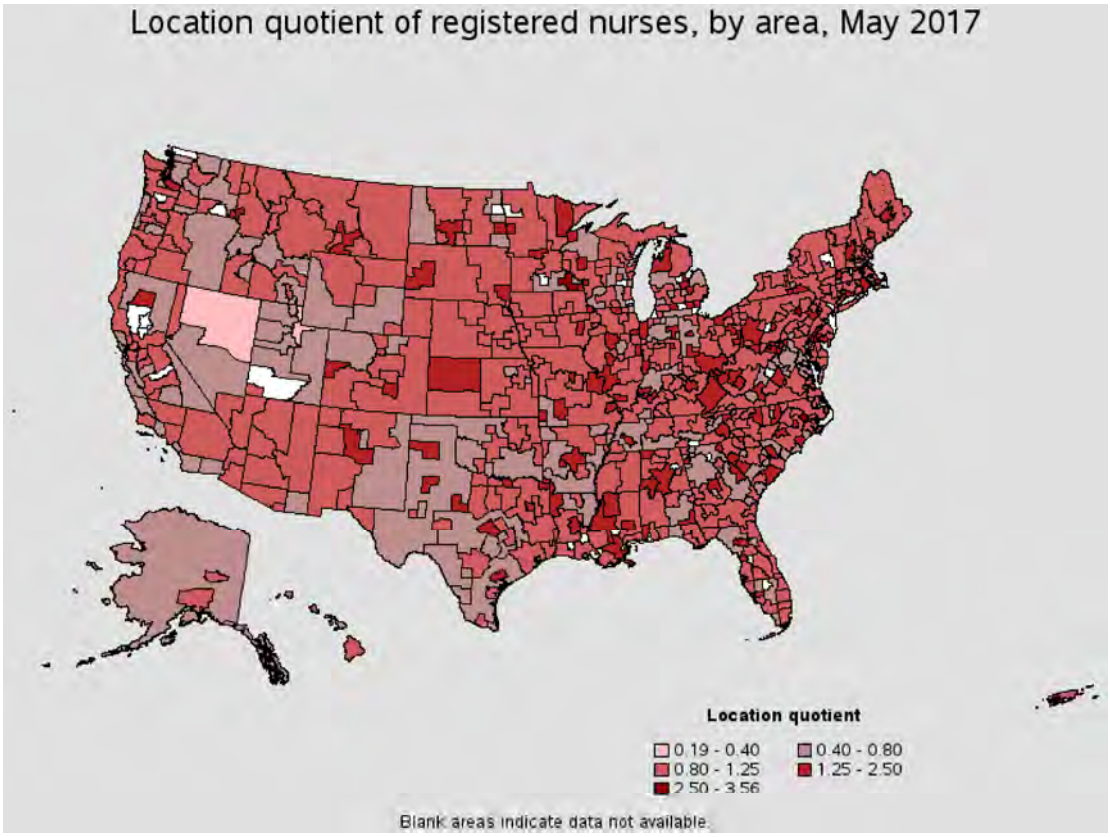
State	Employment (1)	Employment per thousand jobs	Location quotient (9)	Hourly mean wage	Annual mean wage (2)
California	282,290	16.91	0.83	$49.37	$102,700
Hawaii	10,800	17.07	0.84	$46.63	$96,990
District of Columbia	11,000	15.54	0.76	$43.32	$90,110
Massachusetts	82,870	23.49	1.15	$42.95	$89,330
Oregon	35,140	19.19	0.94	$42.68	$88,770



<

Metropolitan areas with the highest employment level in this occupation:

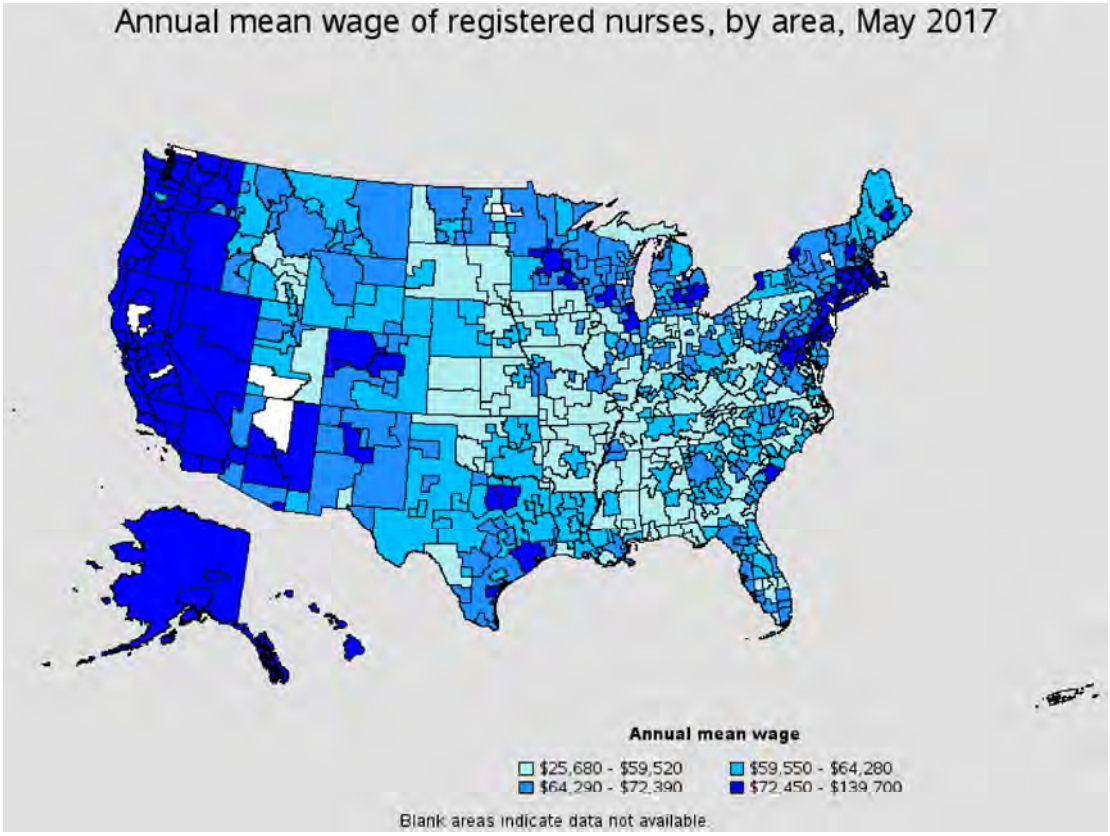
Metropolitan area	Employment (1)	Employment per thousand jobs	Location quotient (9)	Hourly mean wage	Annual mean wage (2)
New York-Jersey City-White Plains, NY-NJ Metropolitan Division	122,780	18.34	0.90	$43.67	$90,840
Los Angeles-Long Beach-Glendale, CA Metropolitan Division	79,420	17.92	0.88	$45.99	$95,650
Chicago-Naperville-Arlington Heights, IL Metropolitan Division	75,320	20.57	1.01	$36.84	$76,640
Houston-The Woodlands-Sugar Land, TX	51,610	17.62	0.86	$38.01	$79,060
Boston-Cambridge-Newton, MA NECTA Division	43,150	23.45	1.15	$46.70	$97,130
Dallas-Plano-Irving, TX Metropolitan Division	41,140	16.51	0.81	$35.81	$74,480
Atlanta-Sandy Springs-Roswell, GA	40,400	15.42	0.76	$33.92	$70,540
Minneapolis-St. Paul-Bloomington, MN-WI	39,290	20.34	1.00	$39.19	$81,510
Phoenix-Mesa-Scottsdale, AZ	38,670	19.53	0.96	$36.61	$76,140
St. Louis, MO-IL	36,670	27.03	1.33	$31.69	$65,910



<

Metropolitan areas with the highest concentration of jobs and location quotients in this occupation:

Metropolitan area	Employment (1)	Employment per thousand jobs	Location quotient (9)	Hourly mean wage	Annual mean wage (2)
Bloomsburg-Berwick, PA	3,090	72.52	3.56	$28.26	$58,780
Rochester, MN	7,640	66.54	3.26	$37.46	$77,920
Greenville, NC	4,150	55.65	2.73	$29.36	$61,060
Morgantown, WV	3,050	47.29	2.32	$31.12	$64,730
Gainesville, FL	5,470	41.51	2.04	$32.42	$67,430
Sherman-Denison, TX	1,860	41.34	2.03	$30.83	$64,130
Sioux Falls, SD	6,050	39.93	1.96	$27.77	$57,750
Durham-Chapel Hill, NC	11,700	39.18	1.92	$32.39	$67,360
Rome, GA	1,440	38.92	1.91	$30.65	$63,740
Huntington-Ashland, WV-KY-OH	5,060	38.70	1.90	$29.47	$61,290



<

Top paying metropolitan areas for this occupation:

Metropolitan area	Employment (1)	Employment per thousand jobs	Location quotient (9)	Hourly mean wage	Annual mean wage (2)
San Francisco-Redwood City-South San Francisco, CA Metropolitan Division	14,170	12.69	0.62	$67.16	$139,700
Salinas, CA	2,470	14.36	0.70	$62.47	$129,940
San Jose-Sunnyvale-Santa Clara, CA	15,990	14.69	0.72	$62.09	$129,140
Santa Cruz-Watsonville, CA	1,520	15.65	0.77	$59.84	$124,470
Vallejo-Fairfield, CA	3,170	23.32	1.14	$57.61	$119,830
Oakland-Hayward-Berkeley, CA Metropolitan Division	19,550	17.18	0.84	$56.09	$116,660
Sacramento--Roseville--Arden-Arcade, CA	18,240	18.99	0.93	$55.85	$116,170
Napa, CA	1,430	19.66	0.96	$54.68	$113,740
Santa Rosa, CA	3,170	15.65	0.77	$53.62	$111,530
Stockton-Lodi, CA	4,270	17.59	0.86	$52.03	$108,230

Nonmetropolitan areas with the highest employment in this occupation:

Nonmetropolitan area	Employment (1)	Employment per thousand jobs	Location quotient (9)	Hourly mean wage	Annual mean wage (2)
Piedmont North Carolina nonmetropolitan area	6,200	24.16	1.18	$28.88	$60,060
North Northeastern Ohio non-metropolitan area (non-contiguous)	5,420	16.33	0.80	$28.45	$59,190
Northeast Mississippi nonmetropolitan area	5,340	23.08	1.13	$26.03	$54,150
Southeast Coastal North Carolina nonmetropolitan area	4,620	18.65	0.91	$28.28	$58,810
	4,450	16.53	0.81	$29.87	$62,130

North Texas Region of Texas nonmetropolitan area					

Nonmetropolitan areas with the highest concentration of jobs and location quotients in this occupation:

Nonmetropolitan area	Employment (1)	Employment per thousand jobs	Location quotient (9)	Hourly mean wage	Annual mean wage (2)
West Central New Hampshire nonmetropolitan area	2,440	37.79	1.85	$35.73	$74,310
Upper Savannah South Carolina nonmetropolitan area	2,900	37.15	1.82	$29.32	$60,980
East Kentucky nonmetropolitan area	3,300	32.94	1.62	$27.01	$56,190
Northwest Kansas nonmetropolitan area	1,560	26.20	1.28	$26.84	$55,820
Northwest Lower Peninsula of Michigan nonmetropolitan area	3,190	26.14	1.28	$30.95	$64,380

Top paying nonmetropolitan areas for this occupation:

Nonmetropolitan area	Employment (1)	Employment per thousand jobs	Location quotient (9)	Hourly mean wage	Annual mean wage (2)
Mother Lode Region of California nonmetropolitan area	730	17.19	0.84	$48.89	$101,700
Eastern Sierra Region of California nonmetropolitan area	190	13.54	0.66	$45.04	$93,690
Hawaii / Kauai nonmetropolitan area	1,610	16.35	0.80	$44.78	$93,130
North Central Massachusetts nonmetropolitan area	(8)	(8)	(8)	$44.12	$91,770
Balance of Alaska nonmetropolitan area	760	10.74	0.53	$43.98	$91,480

About May 2017 National, State, Metropolitan, and Nonmetropolitan Area Occupational Employment and Wage Estimates

These estimates are calculated with data collected from employers in all industry sectors, all metropolitan and nonmetropolitan areas, and all states and the District of Columbia. The top employment and wage figures are provided above. The complete list is available in the downloadable XLS files.

The percentile wage estimate is the value of a wage below which a certain percent of workers fall. The median wage is the 50th percentile wage estimate--50 percent of workers earn less than the median and 50 percent of workers earn more than the median. More about percentile wages.

(1) Estimates for detailed occupations do not sum to the totals because the totals include occupations not shown separately. Estimates do not include self-employed workers.

(2) Annual wages have been calculated by multiplying the hourly mean wage by a "year-round, full-time" hours figure of 2,080 hours; for those occupations where there is not an hourly wage published, the annual wage has been directly calculated from the reported survey data.

(3) The relative standard error (RSE) is a measure of the reliability of a survey statistic. The smaller the relative standard error, the more precise the estimate.

(8) Estimate not released.

(9) The location quotient is the ratio of the area concentration of occupational employment to the national average concentration. A location quotient greater than one indicates the occupation has a higher share of employment than average, and a location quotient less than one indicates the occupation is less prevalent in the area than average.

Other OES estimates and related information:

May 2017 National Occupational Employment and Wage Estimates

May 2017 State Occupational Employment and Wage Estimates

May 2017 Metropolitan and Nonmetropolitan Area Occupational Employment and Wage Estimates

May 2017 National Industry-Specific Occupational Employment and Wage Estimates

May 2017 Occupation Profiles

Technical Notes

Last Modified Date: March 30, 2018

RECOMMEND THIS PAGE USING: Facebook Twitter LinkedIn

TOOLS
Areas at a Glance
Industries at a Glance
Economic Releases
Databases & Tables
Maps

CALCULATORS
Inflation
Injury And Illness

HELP
Help & Tutorials
FAQs
Glossary
About BLS
Contact Us

INFO
What's New
Careers @ BLS
Find It! DOL
Join our Mailing Lists
Linking & Copyright Info

RESOURCES
Inspector General (OIG)
Budget and Performance
No Fear Act
USA.gov
Benefits.gov
Disability.gov

Freedom of Information Act | **Privacy & Security Statement** | **Disclaimers** | **Customer Survey** | **Important Web Site Notices**

U.S. Bureau of Labor Statistics | Division of Occupational Employment Statistics, PSB Suite 2135, 2 Massachusetts Avenue, NE Washington, DC 20212-0001
www.bls.gov/OES | Telephone: 1-202-691-6569 | Contact OES

Occupational Employment Statistics

Occupational Employment and Wages, May 2017

11-9111 Medical and Health Services Managers

Plan, direct, or coordinate medical and health services in hospitals, clinics, managed care organizations, public health agencies, or similar organizations.

National estimates for this occupation
Industry profile for this occupation
Geographic profile for this occupation

National estimates for this occupation: Top

Employment estimate and mean wage estimates for this occupation:

Employment (1)	Employment RSE (3)	Mean hourly wage	Mean annual wage (2)	Wage RSE (3)
346,980	0.5 %	$53.69	$111,680	0.4 %

Percentile wage estimates for this occupation:

Percentile	10%	25%	50% (Median)	75%	90%
Hourly Wage	$28.05	$36.22	$47.29	$62.00	$84.68
Annual Wage (2)	$58,350	$75,340	$98,350	$128,960	$176,130

Industry profile for this occupation: Top

Industries with the highest published employment and wages for this occupation are provided. For a list of all industries with employment in this occupation, see the Create Customized Tables function.

Industries with the highest levels of employment in this occupation:

Industry	Employment (1)	Percent of industry employment	Hourly mean wage	Annual mean wage (2)
General Medical and Surgical Hospitals	116,940	2.13	$57.95	$120,540
Offices of Physicians	40,490	1.59	$50.87	$105,810
Outpatient Care Centers	24,880	2.83	$49.74	$103,460
Nursing Care Facilities (Skilled Nursing Facilities)	22,670	1.40	$44.59	$92,750
Home Health Care Services	20,020	1.43	$47.70	$99,220

Industries with the highest concentration of employment in this occupation:

Industry	Employment (1)	Percent of industry employment	Hourly mean wage	Annual mean wage (2)
Outpatient Care Centers	24,880	2.83	$49.74	$103,460
General Medical and Surgical Hospitals	116,940	2.13	$57.95	$120,540
Specialty (except Psychiatric and Substance Abuse) Hospitals	5,080	1.96	$60.45	$125,730
Medical and Diagnostic Laboratories	5,090	1.92	$58.45	$121,570
Other Ambulatory Health Care Services	5,260	1.76	$46.71	$97,160

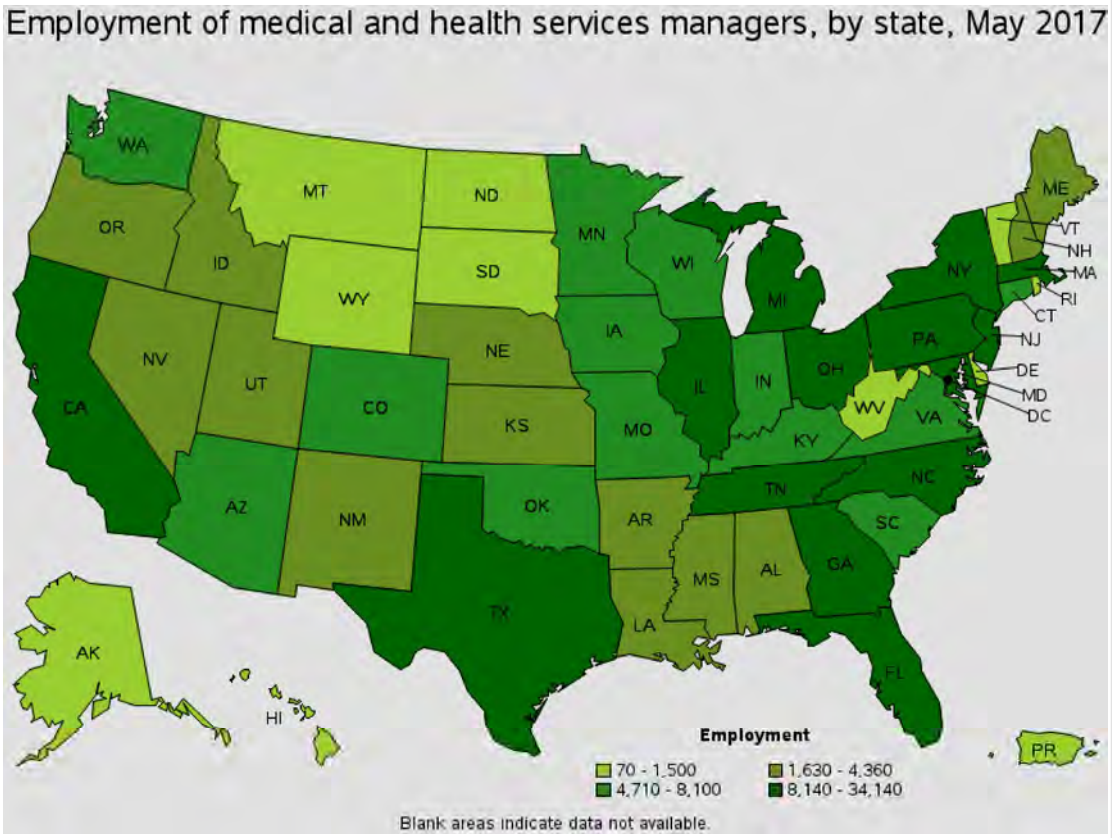
Top paying industries for this occupation:

Industry	Employment (1)	Percent of industry employment	Hourly mean wage	Annual mean wage (2)
Pharmaceutical and Medicine Manufacturing	570	0.20	$106.42	$221,360
Scientific Research and Development Services	2,720	0.42	$83.18	$173,020

Navigational, Measuring, Electromedical, and Control Instruments Manufacturing	60	0.02	$75.59	$157,220
Computer Systems Design and Related Services	120	0.01	$74.22	$154,380
Business, Professional, Labor, Political, and Similar Organizations	320	0.07	$73.71	$153,320

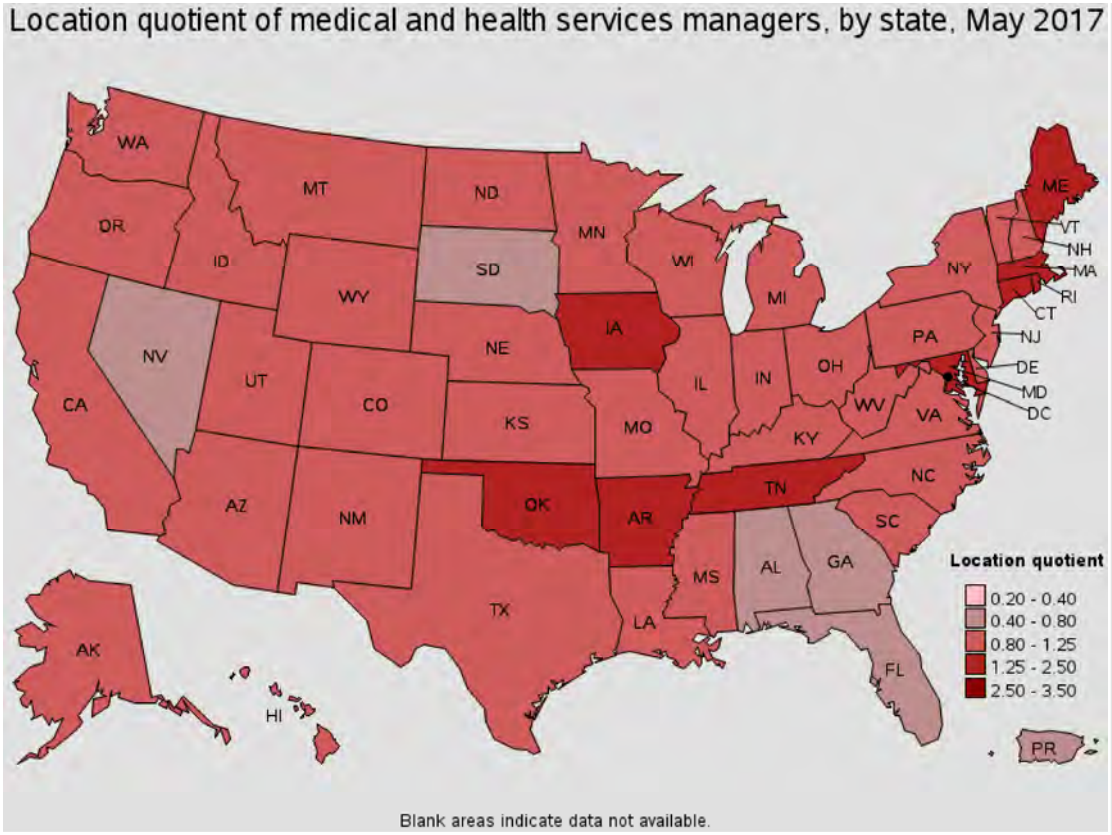
Geographic profile for this occupation: Top

States and areas with the highest published employment, location quotients, and wages for this occupation are provided. For a list of all areas with employment in this occupation, see the Create Customized Tables function.



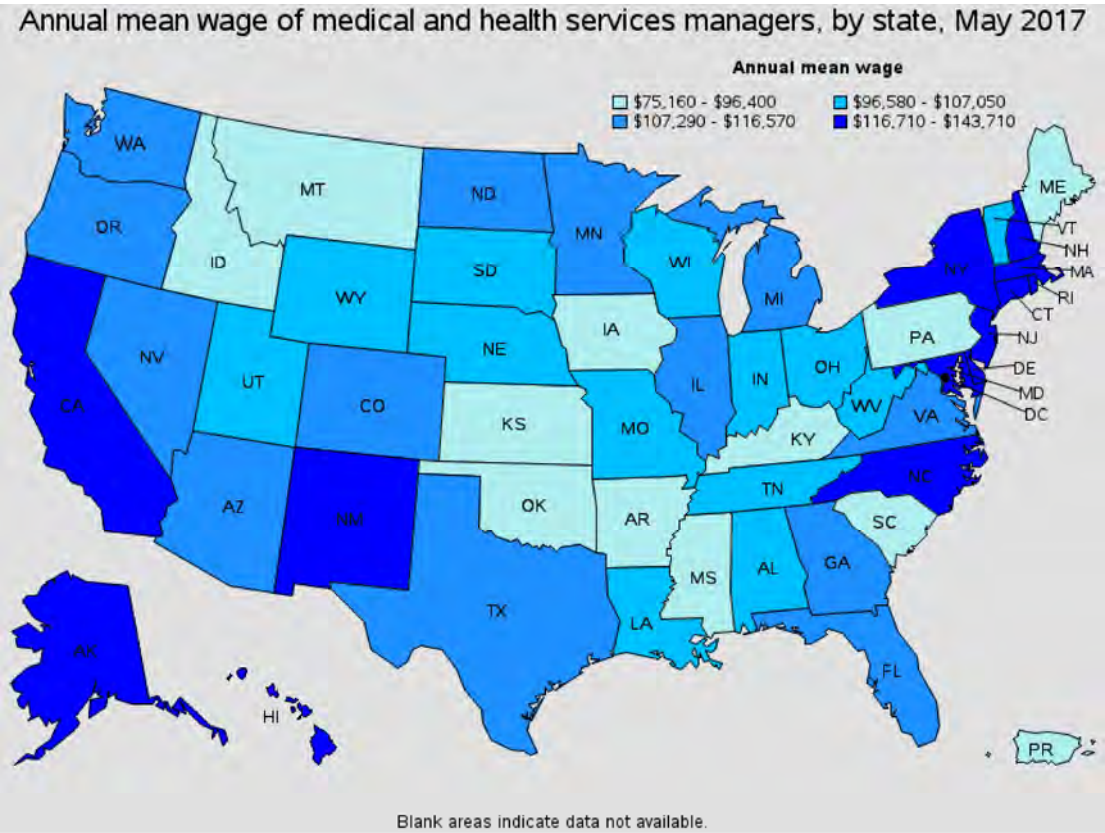
States with the highest employment level in this occupation:

State	Employment (1)	Employment per thousand jobs	Location quotient (9)	Hourly mean wage	Annual mean wage (2)
California	34,140	2.05	0.84	$58.90	$122,500
New York	25,850	2.81	1.15	$65.75	$136,770
Texas	23,740	2.00	0.82	$51.58	$107,290
Ohio	15,330	2.85	1.17	$48.21	$100,290
Pennsylvania	14,540	2.52	1.03	$46.35	$96,400



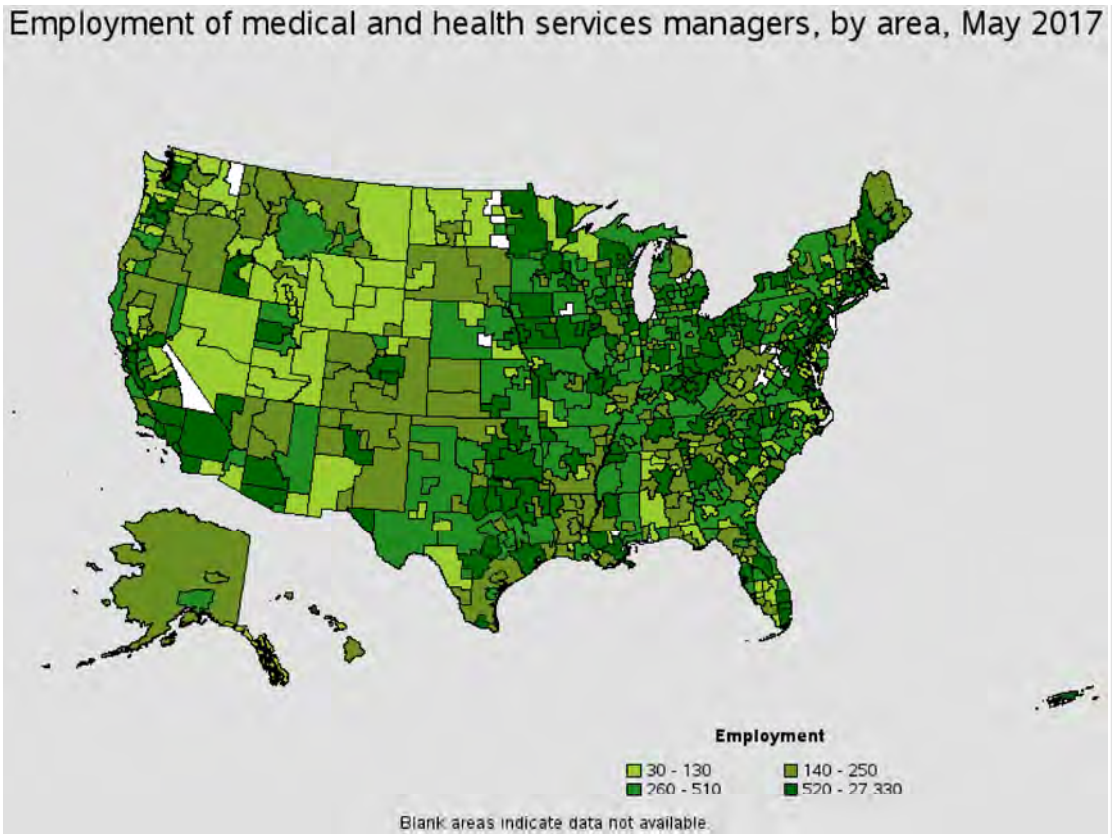
States with the highest concentration of jobs and location quotients in this occupation:

State	Employment (1)	Employment per thousand jobs	Location quotient (9)	Hourly mean wage	Annual mean wage (2)
Iowa	6,160	4.01	1.65	$41.69	$86,710
Oklahoma	6,210	3.95	1.62	$42.79	$88,990
Massachusetts	13,770	3.90	1.60	$61.89	$128,730
Maryland	10,210	3.83	1.57	$57.15	$118,860
Arkansas	4,360	3.63	1.49	$39.87	$82,930



Top paying States for this occupation:

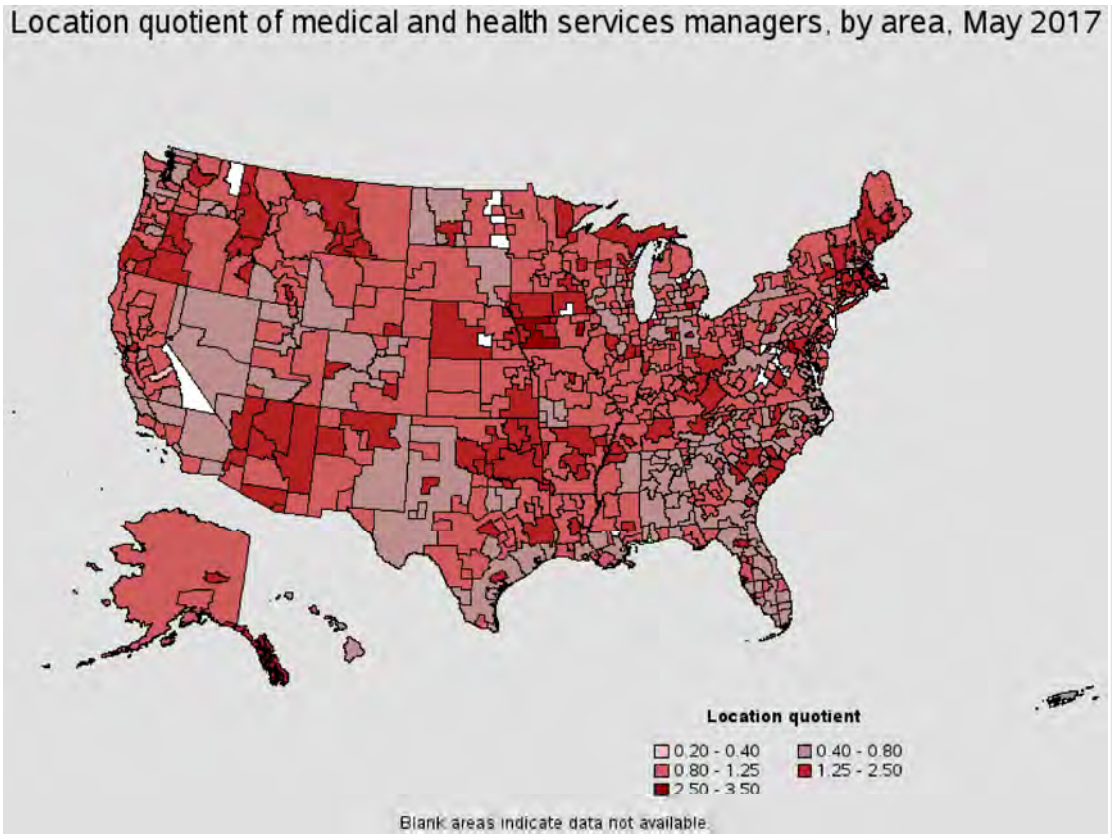
State	Employment (1)	Employment per thousand jobs	Location quotient (9)	Hourly mean wage	Annual mean wage (2)
District of Columbia	1,670	2.36	0.97	$69.09	$143,710
New York	25,850	2.81	1.15	$65.75	$136,770
Connecticut	5,440	3.29	1.35	$63.75	$132,600
Delaware	1,010	2.29	0.94	$62.06	$129,070
Massachusetts	13,770	3.90	1.60	$61.89	$128,730



<

Metropolitan areas with the highest employment level in this occupation:

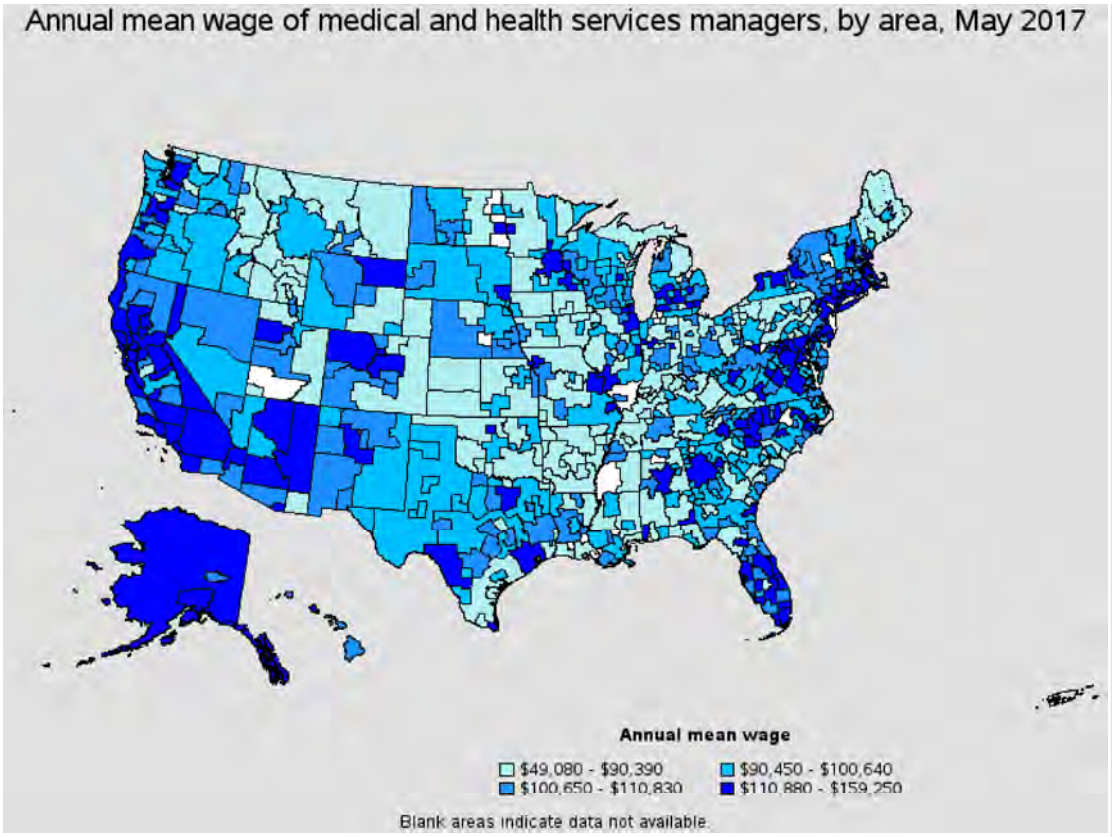
Metropolitan area	Employment (1)	Employment per thousand jobs	Location quotient (9)	Hourly mean wage	Annual mean wage (2)
New York-Jersey City-White Plains, NY-NJ Metropolitan Division	19,870	2.97	1.22	$66.34	$137,980
Los Angeles-Long Beach-Glendale, CA Metropolitan Division	9,200	2.08	0.85	$54.59	$113,540
Chicago-Naperville-Arlington Heights, IL Metropolitan Division	7,930	2.16	0.89	$56.99	$118,540
Boston-Cambridge-Newton, MA NECTA Division	7,130	3.87	1.59	$71.60	$148,930
Houston-The Woodlands-Sugar Land, TX	5,620	1.92	0.79	$57.01	$118,590
Phoenix-Mesa-Scottsdale, AZ	5,610	2.83	1.16	$55.41	$115,260
Minneapolis-St. Paul-Bloomington, MN-WI	5,140	2.66	1.09	$55.77	$116,000
Washington-Arlington-Alexandria, DC-VA-MD-WV Metropolitan Division	4,920	1.95	0.80	$62.44	$129,880
Dallas-Plano-Irving, TX Metropolitan Division	4,630	1.86	0.76	$54.02	$112,370
Atlanta-Sandy Springs-Roswell, GA	4,400	1.68	0.69	$55.65	$115,740



<

Metropolitan areas with the highest concentration of jobs and location quotients in this occupation:

Metropolitan area	Employment (1)	Employment per thousand jobs	Location quotient (9)	Hourly mean wage	Annual mean wage (2)
Peabody-Salem-Beverly, MA NECTA Division	700	7.37	3.03	$52.43	$109,060
Silver Spring-Frederick-Rockville, MD Metropolitan Division	4,010	6.86	2.82	$58.53	$121,730
Iowa City, IA	620	6.76	2.78	$48.14	$100,130
Ann Arbor, MI	1,340	6.24	2.56	$53.31	$110,880
Pittsfield, MA	220	5.54	2.27	$56.19	$116,870
Hot Springs, AR	180	5.22	2.14	$38.11	$79,260
Rochester, MN	590	5.13	2.11	$55.39	$115,210
Lawrence-Methuen Town-Salem, MA-NH NECTA Division	420	5.11	2.10	$53.87	$112,060
Ames, IA	210	4.76	1.95	$43.98	$91,490
Jackson, TN	310	4.75	1.95	$40.69	$84,630



<

Top paying metropolitan areas for this occupation:

Metropolitan area	Employment (1)	Employment per thousand jobs	Location quotient (9)	Hourly mean wage	Annual mean wage (2)
San Francisco-Redwood City-South San Francisco, CA Metropolitan Division	2,170	1.94	0.80	$76.56	$159,250
Vallejo-Fairfield, CA	520	3.83	1.57	$73.89	$153,680
Nassau County-Suffolk County, NY Metropolitan Division	4,120	3.16	1.30	$72.91	$151,660
Boston-Cambridge-Newton, MA NECTA Division	7,130	3.87	1.59	$71.60	$148,930
Bridgeport-Stamford-Norwalk, CT	1,220	2.93	1.20	$70.30	$146,220
Athens-Clarke County, GA	180	2.15	0.88	$69.37	$144,280
Madera, CA	40	0.89	0.37	$69.20	$143,940
San Jose-Sunnyvale-Santa Clara, CA	2,110	1.94	0.80	$68.96	$143,440
Santa Cruz-Watsonville, CA	340	3.50	1.44	$66.45	$138,220
New York-Jersey City-White Plains, NY-NJ Metropolitan Division	19,870	2.97	1.22	$66.34	$137,980

Nonmetropolitan areas with the highest employment in this occupation:

Nonmetropolitan area	Employment (1)	Employment per thousand jobs	Location quotient (9)	Hourly mean wage	Annual mean wage (2)
North Northeastern Ohio non-metropolitan area (non-contiguous)	730	2.21	0.91	$42.83	$89,090
North Texas Region of Texas nonmetropolitan area	710	2.63	1.08	$45.24	$94,100
Southern Ohio non-metropolitan area	690	4.41	1.81	$44.53	$92,620
Southeast Iowa nonmetropolitan area	690	3.02	1.24	$38.33	$79,730

Southwest Maine nonmetropolitan area	630	3.30	1.35	$42.76	$88,940

Nonmetropolitan areas with the highest concentration of jobs and location quotients in this occupation:

Nonmetropolitan area	Employment (1)	Employment per thousand jobs	Location quotient (9)	Hourly mean wage	Annual mean wage (2)
Southwest Iowa nonmetropolitan area	520	6.16	2.53	$40.97	$85,210
West Central New Hampshire nonmetropolitan area	380	5.94	2.44	$65.50	$136,240
Southwest Oklahoma nonmetropolitan area	290	4.70	1.93	$32.15	$66,880
Northeast Iowa nonmetropolitan area	470	4.68	1.92	$36.92	$76,800
Northwest Massachusetts nonmetropolitan area	120	4.46	1.83	$45.46	$94,570

Top paying nonmetropolitan areas for this occupation:

Nonmetropolitan area	Employment (1)	Employment per thousand jobs	Location quotient (9)	Hourly mean wage	Annual mean wage (2)
Eastern Sierra Region of California nonmetropolitan area	(8)	(8)	(8)	$65.97	$137,230
West Central New Hampshire nonmetropolitan area	380	5.94	2.44	$65.50	$136,240
Arizona nonmetropolitan area	360	4.00	1.64	$64.02	$133,170
Mother Lode Region of California nonmetropolitan area	90	2.14	0.88	$61.80	$128,530
Balance of Alaska nonmetropolitan area	180	2.56	1.05	$60.36	$125,540

About May 2017 National, State, Metropolitan, and Nonmetropolitan Area Occupational Employment and Wage Estimates

These estimates are calculated with data collected from employers in all industry sectors, all metropolitan and nonmetropolitan areas, and all states and the District of Columbia. The top employment and wage figures are provided above. The complete list is available in the downloadable XLS files.

The percentile wage estimate is the value of a wage below which a certain percent of workers fall. The median wage is the 50th percentile wage estimate--50 percent of workers earn less than the median and 50 percent of workers earn more than the median. More about percentile wages.

(1) Estimates for detailed occupations do not sum to the totals because the totals include occupations not shown separately. Estimates do not include self-employed workers.

(2) Annual wages have been calculated by multiplying the hourly mean wage by a "year-round, full-time" hours figure of 2,080 hours; for those occupations where there is not an hourly wage published, the annual wage has been directly calculated from the reported survey data.

(3) The relative standard error (RSE) is a measure of the reliability of a survey statistic. The smaller the relative standard error, the more precise the estimate.

(8) Estimate not released.

(9) The location quotient is the ratio of the area concentration of occupational employment to the national average concentration. A location quotient greater than one indicates the occupation has a higher share of employment than average, and a location quotient less than one indicates the occupation is less prevalent in the area than average.

Other OES estimates and related information:

May 2017 National Occupational Employment and Wage Estimates

May 2017 State Occupational Employment and Wage Estimates

May 2017 Metropolitan and Nonmetropolitan Area Occupational Employment and Wage Estimates

May 2017 National Industry-Specific Occupational Employment and Wage Estimates

May 2017 Occupation Profiles

Technical Notes

Last Modified Date: March 30, 2018

RECOMMEND THIS PAGE USING: Facebook Twitter LinkedIn

TOOLS
Areas at a Glance
Industries at a Glance
Economic Releases
Databases & Tables
Maps

CALCULATORS
Inflation
Injury And Illness

HELP
Help & Tutorials
FAQs
Glossary
About BLS
Contact Us

INFO
What's New
Careers @ BLS
Find It! DOL
Join our Mailing Lists
Linking & Copyright Info

RESOURCES
Inspector General (OIG)
Budget and Performance
No Fear Act
USA.gov
Benefits.gov
Disability.gov

Freedom of Information Act | **Privacy & Security Statement** | **Disclaimers** | **Customer Survey** | **Important Web Site Notices**

U.S. Bureau of Labor Statistics | Division of Occupational Employment Statistics, PSB Suite 2135, 2 Massachusetts Avenue, NE Washington, DC 20212-0001
www.bls.gov/OES | Telephone: 1-202-691-6569 | Contact OES