Attachment 3.2a
Anniston Community Health Survey: Follow up Study and Dioxin Analyses
List of Chemicals

Table 1. Polychlorinated biphenyls (PCB)[1] (ng/g lipid) measured in the study.

IUPAC No.	Test Name	NHANES Reference Range[2] (Units)
	Polychlorinated biphenyls (PCBs)	***(ng/g lipid)***
PCB 28	2,4,4′-trichlorobiphenyl	4.98 - 11.1
PCB 44	2,2',3,5-tetrachlorobiphenyl	2.00 - 5.44
PCB 49	2,2',4,5'-tetrachlorobipheny	1.33 - 3.36
PCB 52	2,2',5,5'-tetrachlorobiphenyl	2.70 - 7.15
PCB 66	2,3',4,4'-tetrachlorobiphenyl	1.40 - 4.20
PCB 74	2,4,4',5-tetrachlorobiphenyl	5.00 – 24.1
PCB 87	3,4,4',5-tetrachlorobiphenyl	<LOD – 13.1
PCB 99	2,2',3,4,5'-pentachlorobiphenyl	0.90 - 2.60
PCB 101	2,2',4,4',5-pentachlorobiphenyl	4.08 - 18.6
PCB 105	2,2',4,5,5'-pentachlorobiphenyl†	1.67 - 5.51
PCB 110	2,3,3',4,4'-pentachlorobiphenyl	1.15 - 6.82
PCB 118	2,3,3',4',6-pentachlorobiphenyl†	1.20 - 4.18
PCB 128	3,3',4,4',5-pentachlorobiphenyl	16.0 – 74.8
PCB 138-158	2,2',3,3',4,4'-hexachlorobiphenyl	<LOD - 0.62
PCB 146	2,2',3,4,4',5'and 2,3,3',4,4',6-hexachlorobiphenyl	17.6 – 77.4
PCB 149	2,2',3,4',5,5'-hexachlorobiphenyl	2.60 - 12.7
PCB 151	2,2',3,4',5',6-hexachlorobiphenyl	0.60 - 1.89
PCB 153	2,2',3,5,5',6-hexachlorobiphenyl	<LOD – 1.02
PCB 156	2,2',4,4',5,5'-hexachlorobiphenyl†	24.2 - 101
PCB 157	2,3,3',4,4',5-hexachlorobiphenyl †	4.10 – 16.8
PCB 167	2,3,3',4,4',5'-hexachlorobiphenyl †	0.98 – 3.97
PCB 170	3,3',4,4',5,5'-hexachlorobiphenyl	<LOD – 43.2
PCB 172	2,2',3,3',4,4',5-heptachlorobiphenyl	7.83 – 29.5
PCB 177	2,2',3,3',4,5,5'-heptachlorobiphenyl	1.08 - 4.38
PCB 178	2,2',3,3',4',5,6-heptachlorobiphenyl	1.50 - 7.80
PCB 180	2,2',3,3',5,5',6-heptachlorobiphenyl	1.46 - 6.50
PCB 183	2,2',3,4,4',5,5'-heptachlorobiphenyl	21.5 – 88.0
PCB 187	2,2',3,4,4',5',6-heptachlorobiphenyl	1.88 – 8.40
PCB 189	2,2',3,4',5,5',6-heptachlorobiphenyl†	5.71 – 25.9
PCB 194	2,3,3',4,4',5,5'-heptachlorobiphenyl	<LOD – 1.50
PCB 195	2,2',3,3',4,4',5,5'-octachlorobiphenyl	4.95 - 20.1
PCB 196-203	2,2',3,3',4,4',5,6-octachlorobiphenyl	1.10 - 4.68
PCB 199	2,2',3,3',4,4',5,6'and 2,2',3,4,4',5,5',6-octachlorobiphenyl	4.07 - 15.9
PCB 206	2,2',3,3',4,5,5',6'-octachlorobiphenyl	4.60 – 20.6
PCB 209	2,2',3,3',4,4',5,5',6'-nonachlorobiphenyl	2.80 – 14.2
PCB 28	2,2',3,3',4,4',5,5',6,6'-decachlorobiphenyl	1.40 – 12.3

[1] Dioxin like coplanar PCB congeners results (PCBs 81, 126, and 169) are shown in Table 2.
[2] CDC. 2009. 2003-2004 NHANES 50th to 95th percentiles among adults 20+ years old from the Fourth National Report on Human Exposure to Environmental Chemicals (http://www.cdc.gov/exposurereport/pdf/FourthReport.pdf).
<LOD – Below the limit of detection. †Mono-*ortho* substituted PCB congeners.

Table 2. Dioxins (PCDDs), dibenzofurans (PCDFs), coplanar PCBs, chlorinated pesticides and polybrominated diphenyl ethers (PBDEs) measured in the study.

Serial or IUPAC No.	Test Name	NHANES Reference Range[1] (Units)
	Polychlorinated dibenzo-p-dioxins (PCDDs)	***(pg/g lipid)***
1	2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD)	<LOD – 5.30
2	1,2,3,7,8-pentachlorodibenzo-*p*-dioxin (PeCDD)	<LOD – 11.3
3	1,2,3,4,7,8-hexachlorodibenzo-*p*-dioxin (HxCDD)	< LOD – < LOD
4	1,2,3,6,7,8-hexachlorodibenzo-*p*-dioxin (HxCDD)	23.8 – 70.8
5	1,2,3,7,8,9-hexachlorodibenzo-*p*-dioxin (HxCDD)	< LOD – < LOD
6	1,2,3,4,6,7,8-heptachlorodibenzo-*p*-dioxin (HpCDD)	27.3 – 95.0
7	1,2,3,4,6,7,8,9-octachlorodibenzo-*p*-dioxin (OCDD)	223 – 794
	Polychlorinated dibenzo-p-furans (PCDFs)	***(pg/g lipid)***
1	2,3,7,8-tetrachlorodibenzofuran (TCDF)	<LOD - <LOD
2	1,2,3,7,8-pentachlorodibenzofuran (PeCDF)	<LOD - <LOD
3	2,3,4,7,8-pentachlorodibenzofuran (PeCDF)	<LOD – 13.0
4	1,2,3,4,7,8-hexachlorodibenzofuran (HxCDF)	<LOD – 9.50
5	1,2,3,6,7,8-hexachlorodibenzofuran (HxCDF)	<LOD – 9.00
6	2,3,4,6,7,8-hexchlorodibenzofuran (HxCDF)	<LOD - <LOD
7	1,2,3,7,8,9-hexachlorodibenzofuran (HxCDF)	<LOD - <LOD
8	1,2,3,4,6,7,8-heptachlorodibenzofuran (HpCDF)	<LOD – 18.0
9	1,2,3,4,7,8,9-heptachlorodibenzofuran (HpCDF)	<LOD - <LOD
10	1,2,3,4,6,7,8,9-octachlorodibenzofuran (OCDF)	<LOD - <LOD
	Coplanar Polychlorinated Biphenyls (co-PCBs)	***(pg/g lipid)***
PCB 81	2,4,4',5-tetrachlorobiphenyl	0.006 – 0.17
PCB 126	2,3',4,4',5-pentachlorobiphenyl †	5.56 – 34.3
PCB 169	2,3',4,4',5,5'-hexachlorobiphenyl †	0.86 – 4.30
	Chlorinated Pesticides	***(ng/g lipid)***
1	Hexachlorobenzene (HCB)	15.1 – 29.0
2	*β*-Hexachlorocyclohexane (HCH)	<LOD – 62.2
3	*p,p'*-Dichlorodiphenyltrichloroethane (DDT)	<LOD – 20.7
4	*p,p'*-Dichlorodiphenyldichloroethene (DDE)	233 – 1,990
5	Oxychlordane	11.4 – 39.2
6	*trans*-Nonachlor	17.3 – 74.7
7	Heptachlor epoxide	<LOD – 20.6
8	Mirex	<LOD – 15.4
9	Dieldrin	<LOD – 19.5
	Polybrominated diphenyl ethers (PBDEs)	***(ng/g lipid)***
BDE 17	2,2',4-tribromodiphenyl ether	<LOD - <LOD
BDE 28	2,4,4'-tribromodiphenyl ether	1.10 – 8.20
BDE 47	2,2',4,4'-tetrabromodiphenyl ether	18.0 – 163
BDE 66	2,3',4,4'-tetrabromodiphenyl ether	<LOD – 1.30
BDE 85	2,2',3,4,4'-pentabromodiphenyl ether	<LOD – 4.10
BDE 99	2,2',4,4',5-pentabromodiphenyl ether	<LOD – 41.6
BDE 100	2,2',4,4',6-pentabromodiphenyl ether	3.30 – 36.6
BDE 153	2,2',4,4',5,5'-hexabromodiphenyl ether	4.40 – 73.3
BDE 154	2,2',4,4',5,6'-hexabromodiphenyl ether	<LOD – 4.20
BDE 183	2,2',3,4,4',5',6-heptabromodiphenyl ether	<LOD - <LOD

[1] CDC. 2009. 2003-2004 NHANES 50th to 95th percentiles among adults 20+ years old from the Fourth National Report on Human Exposure to Environmental Chemicals (http://www.cdc.gov/exposurereport/pdf/FourthReport.pdf).
<LOD – Below the limit of detection.

Polychlorinated Biphenyls (PCBs)

Polychlorinated biphenyls (PCBs) are a class of chlorinated aromatic hydrocarbon chemicals that once were used as heat-exchanger, transformer, and hydraulic fluids, and as additives to paints, oils, joint caulking, and floor tiles. Peak production occurred in the early 1970s, and production was banned in the United States after 1979. More than 1.5 billion pounds of PCBs were manufactured in the United States prior to 1977. The continued concern about these chemicals is because of their persistence in the environment and accumulation in wildlife and the animal food chain.

How People Are Exposed to PCBs: Food is the main source of exposure for the general population. PCBs enter the food chain by a variety of routes, including migration into food from external sources, contamination of animal feeds, and accumulation in the fatty tissues of animals. PCBs are found at higher concentrations in fatty foods (e.g., dairy products and fish). The transfer of PCBs from mother to infant via breast milk is another important source of exposure. The lesser-chlorinated PCBs are more volatile and indoor inhalational exposure from buildings containing caulking made with these PCBs prior to 1979 can increase background serum levels (Johansson et al., 2003; Kohler et al., 2005). Other sources of exposure in the general population include the release of these chemicals from PCB-containing waste sites and from fires involving transformers and capacitors. Additionally, the heat from fires can result in the production of polychlorinated dibenzofurans from PCBs. In certain occupational settings, workers can be exposed to PCBs such as when repairing or manufacturing transformers, capacitors, and hydraulic systems, and when remediating hazardous-waste sites. Both U.S. FDA and OSHA have developed criteria on the allowable levels of these chemicals in foods and the workplace. The U.S. EPA has also set criteria for allowable levels in water and waste materials. The international Stockholm Convention on Persistent Organic Pollutants of 2001 establishes the most stringent guidelines to date regarding elimination, restriction and unintentional production of PCBs and selected organochlorine chemicals (Porta and Zumeta, 2002).

Exposure to these chemicals nearly always occurs as mixtures rather than from individual PCBs. The different types of PCB chemicals are known as congeners, which are compounds that are distinguished by the number of chlorine atoms and their location on the biphenyl structure. PCB congeners can be divided into the coplanar, the mono-ortho-substituted PCBs, and other non-dioxin-like PCBs. The significance of this designation is that the coplanar and some of the mono-ortho-substituted PCBs have dioxin-like toxicologic effects. Structural nomenclature is available at: http://www.epa.gov/oswer/riskassessment/pdf/1340-erasc-003.pdf . The non-dioxin-like PCBs and their metabolites do not interact substantially with the aryl hydrocarbon receptor (AhR) and may act through different pathways than the dioxin-like chemicals, so their effects are not represented in the use of toxic equivalency factors (TEFs) (Carpenter, 2006).

How Can PCBs Affect Human Health: Human health effects that have been reported after investigations of occupational and accidental exposures to high levels of PCBs include elevations of serum hepatic enzymes, dermal changes, inconsistent associations with serum lipid levels, and some types of cancer (e.g., liver, biliary) (ATSDR, 2000; Carpenter, 2006; Charles et al., 2001; Negri et al., 2003). Animal studies have demonstrated varied effects of PCBs including neurotoxicity, immune suppression, altered thyroid and reproductive function, and liver cancer (Carpenter, 2006; U.S. EPA, 2008). Effects of PCBs in humans are difficult to study due to coexposures to the dioxin-like chemicals and other organochlorine chemicals. (Also see the section titled: "Dioxin-Like Chemicals: Polychlorinated Dibenzo-p-dioxins, Polychlorinated Dibenzofurans, and the Coplanar and Mono-ortho-substituted Polychlorinated Biphenyls").

Transplacental transfer of PCBs after maternal environmental exposure has been reported to be associated with altered psychomotor development in children and lower birth weight and size in newborns (Hertz-Picciotto et al., 2005; Jacobson and Jacobson, 1996; Koopman-Essenboom et al., 1996; Longnecker et al., 2003; Lundqvist et al., 2006; Sagiv et al., 2007; Sala et al., 2001), although other studies have either not confirmed these findings or found that such effects do not persist into toddler and school aged children (Gladen and Rogan, 1991; Gray et al., 2005; Hertz-Picciotto et al., 2005; Koopman-Essenboom et al., 1996; Wolff et al., 2007). Many animal studies demonstrate that high dose PCB impairs neurodevelopment or their hydroxylated metabolites may interfere with thyroid hormone-dependent neurodevelopment (Kimura-Kuroda et al., 2007; Nguon et al., 2005; Purkey et al., 2004; Roegge et al., 2006).

The non-dioxin-like PCBs weakly interact with estrogen and thyroid receptors and with transport proteins, and the hydroxylated metabolites of PCBs may be more potent mediators of these actions (Azulmozhiraja et al., 2005; DeCastro et al., 2006; Langer et al., 2005; Purkey et al., 2004; Kitamura et al., 2005; You et al., 2006). Variations in thyroid hormone levels have been associated with PCB exposures in human populations (Langer et al., 2007a; Meeker et al., 2007; Otake et al., 2007; Wang et al., 2005). Though only limited investigation of estrogenic or reproductive effects has occurred in women, inconsistent associations of PCB levels with altered spermatogenesis and reproductive hormone levels have been reported in environmentally exposed men (Giwercman et al., 2006; Rignell-Hydbom et al., 2005; Toft et al., 2006).

PCBs are not considered directly genotoxic. They are classified as probable human carcinogens by IARC and are classified by NTP as reasonably anticipated to be carcinogens. Early studies associated workplace PCB exposures with increased deaths from cancer of the liver, gallbladder, biliary tract, gastrointestinal tract, brain and malignant melanoma (Knerr and Schrenk, 2006). Follow up studies of these earlier investigations have shown no increase in deaths or cancers, with the exception of liver cancer (Kimbrough et al., 2003; Prince et al., 2006; Ross, 2004), though the contributions of dioxin-like chemicals or other organochlorines were unclear. Recent studies have associated PCB exposures with other cancers (De Roos et al., 2005; Engel et al., 2007; Prince et al., 2006). Information about external exposure (i.e., environmental levels) and health effects is available from ATSDR at: http://www.atsdr.cdc.gov/toxprofiles/ and from the U.S. EPA at: http://www.epa.gov/iris.

Levels of PCBs in the U.S. Population: Measurement of serum PCBs generally reflect cumulative past exposure. Levels of non-dioxin-like PCBs in NHANES 2003–2004 are observed to be roughly similar to the previous two NHANES survey periods. Many PCBs can remain in the body for years after exposure, though some of the PCBs with fewer chlorine atoms have short residence times. The levels of individual PCB congeners in the body may vary by exposure source and by differences in pharmacokinetics, i.e., those with longer half-lives accumulate to higher levels. Adult age-related accumulations in the non-dioxin-like PCBs have been observed in many studies (Apostoli et al., 2005; Park et al., 2007; Patterson et al., 1994). Breastfeeding is a major source of PCBs, with serum levels increasing after birth in breastfed infants and then decreasing in early adolescence due to dilution as body mass increases (Barr et al., 2006). Fish consumption from the Great Lakes region contributed a twofold to tenfold increase in the mean concentrations of non-dioxin-like PCBs over referent populations (Patterson et al., 1994; Turyk et al., 2006). Arctic native Alaskans who consumed locally-caught fish, meat, and eggs had mean serum levels of total PCB that were nearly three times higher than the adult NHANES 1999–2000 subsample (Carpenter et al., 2005; CDC, 2005; Needham et al., 2005). Much higher levels due to contaminated fish intake have also been noted also in eastern Europe (Langer et al., 2007b).

The concentrations of the di-ortho-substituted PCBs are usually higher than the mono-ortho-substituted PCBs, which in turn are higher than the coplanar PCBs (CDC, 2005; Glynn et al., 2000; Longnecker et al., 2000; Patterson et al., 1994). The most frequently detected di-ortho-chlorine-substituted PCBs in population studies are 138, 153, and 180 (CDC, 2005; Heudorf et al., 2002; Patterson et al., 1994, 2009; Turyk et al., 2006). These three congeners contributed a substantial portion of the total PCB concentration observed in pooled specimens representative of a New Zealand population (Bates et al., 2004); in a small population of Swedish men (Glynn et al., 2000), and in blood bank specimens from Canada (Longnecker et al., 2000). In the U.S representative subsample from NHANES 1999–2000, non-dioxin-like PCBs 138, 153, and 180 accounted for 65% of the measured total sum of PCBs (Needham et al., 2005) and for 78% of the total in a referent population of 311 Italian residents in 2001–2003 (Apostoli et al., 2005). Non-dioxin-like PCBs with five, six, and seven chlorines attached comprised about 80% of the total PCBs in human serum, or alternatively, PCBs 138, 153, 180, 187 and 118 composed 57% of the total PCB concentration in a small sample of South Korean residents and incineration workers (Park et al., 2007). In the sera of Yucheng victims analyzed 15 years after the rice oil contamination event (See the section "Dioxin-Like Chemicals" for further discussion.), 73% of the total PCB concentration was contributed by PCBs 99, 138, 153, 156, 170, 179, and 180 (Hsu et al., 2005).

As has been shown for other organochlorines, median serum lipid-adjusted levels of PCB 153 declined by 38% from 1991 to 2001 in a small sample of Swedish men (Hagmar et al., 2006). In four biannual surveys covering the years 1996–2003, about 400 German fourth grade children were sampled each period and demonstrated a decrease of more than one-half in mean whole blood levels of PCBs 138, 153, and 180 (Link et al., 2005). Lipid adjusted levels of the non-dioxin-like PCBs seen in the U.S representative subsamples from NHANES 2001–2002 are generally lower than levels in selected populations during the 1980s to 1990s (CDC, 2005; Glynn et al., 2000; Longnecker et al., 2000; Patterson et al., 1994).

In a convenience sample of 624 urban Germans aged 0-65 years conducted during 1998 (Heudorf et al., 2002), 95th percentile levels for PCBs 138, 153, and 180 were similar or up to two-fold higher than 95th percentile levels in the U.S. NHANES 1999–2000 subsample (CDC, 2005). In contrast, a representative pooled sampling of New Zealand residents in 1996–1997 demonstrated slightly lower levels than for NHANES 1999–2000 (Bates et al., 2004). In two separate Italian studies of a regional reference population and a convenience sample in 2001–2003, median serum levels of PCBs 138, 153, and 180, as well as the sum of measurable PCBs, were about fivefold higher than NHANES 1999–2000 (Apostoli et al., 2005; CDC, 2005; Needham et al., 2005; Turci et al., 2006). Mean levels of PCBs 153 and 180 in 753 adult native Americans were approximately similar to the 95th percentile for the overall adult NHANES 2001–2002 population (CDC, 2005; DeCaprio et al., 2005). In some other countries, comparable population levels are ten or more times higher than those reported for NHANES subsamples from 1999–2000 and 2001–2002 (CDC, 2005; Jursa et al.,2006; Petrik et al., 2006). In the sera of Yucheng victims analyzed at 15 years following the rice oil contamination event, mean serum lipid adjusted levels of PCBs 99, 153, 170, and 180 were several to eightfold higher than the 95th percentiles of NHANES 1999–2000 (Hsu et al., 2005).

Finding a measurable amount of one or more PCBs in serum does not mean that the levels of the PCBs cause an adverse health effect. Biomonitoring studies of serum PCBs can provide physicians and public health officials with reference values so that they can determine whether or not people have been exposed to higher levels of PCBs than levels found in the general population. Biomonitoring data can also help scientists plan and conduct research on exposure and health effects.

For More Information and References*:

Agency for Toxic Substances and Disease Registry (ATSDR).
Toxicological profile for polychlorinated biphenyls, http://www.atsdr.cdc.gov/toxprofiles/tp17.html. 03/17/05

Center for Disease Control and Prevention*
National Biomonitoring Program, http://www.cdc.gov/biomonitoring/NDL-PCBs_BiomonitoringSummary.html*

Dioxins, Furans and Dioxin-Like Polychlorinated Biphenyls

Dioxins, furans, and dioxin-like polychlorinated biphenyls (PCBs) are the abbreviated names for a family of chemicals that have similar toxicity and shared chemical characteristics. The dioxins and furans are not manufactured or produced intentionally but are created when other chemicals or products are made. These chemicals may be created during burning of forests or household trash; chlorine bleaching of pulp and paper; or manufacturing or processing of certain types of chemicals, such as pesticides. Until banned in 1979, PCBs were manufactured as insulator fluids in heat-exchangers and transformers, as hydraulic fluids, and as additives to paints, oils, and caulks. All of these chemicals remain in the environment even though they are no longer manufactured. They enter the food chain and build up in larger animals.

How People Are Exposed to Dioxins, Furans, and Dioxin-Like PCBs: People can be exposed to these chemicals by eating high-fat foods such as milk products, eggs, meat, and some fish. Workplace exposures can occur in industries that burn waste matter or that manufacture other chemical products containing these substances.

How Dioxins, Furans, and Dioxin-Like PCBs Affect People's Health: Human health effects from low environmental exposures are unclear. People who have been unintentionally exposed to large amounts of these chemicals have developed a skin condition called chloracne, liver problems, and elevated blood lipids (fats). Laboratory animal studies have shown various effects, including cancer and reproductive problems.

Levels of Dioxins, Furans and Dioxin-Like PCBs in the U.S. Population: In the Fourth National Report on Human Exposure to Environmental Chemicals (Fourth Report), CDC scientists measured 26 of these chemicals in the blood serum (the clear part of blood) of at least 1,800 participants aged 12 years and older who took part in the National Health and Nutrition Examination Survey (NHANES) during 2003–2004. Prior survey periods of 1999–2000 and 2001–2002 are also included in the Fourth Report. By measuring these chemicals in serum, scientists can estimate the amounts of these chemicals that have entered people's bodies.

In the Fourth Report, CDC researchers found low levels of these 26 chemicals in the U.S. population. The findings are consistent with other studies that found that the levels of most of these chemicals have decreased by more than 80% since the 1980s.

Finding a measurable amount of one or more of these chemicals in serum does not mean that the level of one or more of these chemicals causes an adverse health effect. Biomonitoring studies of these chemicals provide physicians and public health officials with reference values so that they can determine whether people have been exposed to higher levels than are found in the general population. Biomonitoring data can also help scientists plan and conduct research on exposure and health effects.

For More Information:

Agency for Toxic Substances and Disease Registry (ATSDR)

ToxFAQs for Chlorinated Dibenzo-p-Dioxins, http://www.atsdr.cdc.gov/tfacts104.html;
http://www.atsdr.cdc.gov/tfacts32.html

Environmental Protection Agency

Dioxins and Furans, http://www.epa.gov/pbt/pubs/dioxins.htm

Center for Disease Control and Prevention

National Biomonitoring Program,
http://www.cdc.gov/biomonitoring/DioxinLikeChemicals_FactSheet.html

Polybrominated Diphenyl Ethers (PBDEs) and Polybrominated Biphenyls (PBBs)

Polybrominated diphenyl ethers (PBDEs) and polybrominated biphenyls (PBBs) belong to a class of chemicals that are added to certain manufactured products in order to reduce the chances that the products will catch on fire. Finished products that may contain PBDEs are furniture foam padding; wire insulation; rugs, draperies, and upholstery; and plastic cabinets for televisions, personal computers, and small appliances. PBBs were used in the past, and one in particular, BB-153, has not been produced in the U.S. since the 1970's.

These chemicals can get into the air, water, and soil during their manufacture; they can leak from products that contain them or escape when the products that contain them break down. They do not dissolve easily in water; they stick to particles and settle to the bottom of rivers or lakes. Some PBDEs can build up in certain fish and mammals when they eat contaminated food or water.

How People Are Exposed to PBDEs and PBBs: People can be exposed to PBDEs and PBBs by eating contaminated foods, especially those with a high fat content, such as fatty fish. Another source of exposure results from breathing contaminated air or swallowing contaminated dust. Working in industries that make these chemicals or that make, repair, or recycle products containing these chemicals flame retardants can result in exposure.

How PBDEs and PBBs Affect People's Health: Human health effects from PBDEs and PBBs at low environmental exposures are unknown. In animal studies, these chemicals have shown some effects on the thyroid and liver, as well as on brain development. More research is needed to assess the human health effects of exposure to PBDEs and PBBs.

Levels of PDBEs and PBBs in the U.S. Population: In the Fourth National Report on Human Exposure to Environmental Chemicals (Fourth Report), CDC scientists measured ten different PBDEs in the blood serum (the clear portion of blood) of at least 1,985 participants aged 12 years and older who took part in the National Health and Nutrition Examination Survey (NHANES) during 2003–2004. In addition, BB-153, which is one of the PBBs, was measured in 2,032 participants aged 12 years and older. By measuring PBDEs and PBBs in blood serum scientists can estimate the amounts of these chemicals that have entered people's bodies.

In the Fourth Report, scientists found that one PBDE, BDE-47, demonstrated the highest levels of the ten different PBDEs measured in the Fourth Report. These levels for the U.S. population are roughly 3 to10 times higher than levels seen in participants of various studies from European countries. The following PBDEs were detected in greater than 60 percent of participants: BDE-28, BDE-99, BDE-100, and BDE-153. Also, BB-153 was detected in greater than 60 percent of participants.

Finding measurable amounts of PBDEs and/or PBBs in serum does not mean that the levels of these chemicals cause an adverse health effect. Biomonitoring studies of serum PBDEs and PBBs can provide physicians and public health officials with reference values so that they can determine whether people have been exposed to higher levels of PBDEs and/or PBBs than are found in the general population. Biomonitoring data can also help scientists plan and conduct research on exposure and health effects.

For More Information:

Agency for Toxic Substances and Disease Registry

Polybrominated Diphenyl Ethers (PBDEs), http://www.atsdr.cdc.gov/tfacts68-pbde.html
ToxFAQs for Polybrominated Biphenyls (PBBs), http://www.atsdr.cdc.gov/tfacts68.pdf

Environmental Protection Agency

Polybrominated Diphenyl Ethers (PBDEs), http://www.epa.gov/oppt/pbde/

Center for Disease Control and Prevention

National Biomonitoring Program,
http://www.cdc.gov/biomonitoring/DioxinLikeChemicals_FactSheet.html

Chlorinated Pesticides

Hexachlorobenzene (HCB)

Hexachlorobenzene (HCB) was used from the 1930's to the 1970's in the U.S. primarily as a fungicide and seed treatment until the U.S. EPA cancelled its use in 1984. Although it is not manufactured as an end-product in the U.S., HCB may be created as either a by-product or an impurity in the manufacturing process for certain chemicals and pesticides.

How People Are Exposed to Hexachlorobenzene: Hexachlorobenzene has entered the environment as a result of industrial activities and pesticide applications, and has been detected in soil, air, water, and sediment (Barber et al., 2005). It is a persistent chemical and bioaccumulates in both aquatic and terrestrial food chains (ATSDR, 2002). The general population may be exposed to HCB through diet, particularly by consuming fish, wildfowl, or game taken from areas with HCB contamination, and foods with a high fat content. The FDA dietary surveys have shown that over time, HCB has been detected in fewer foods since the 1980s (FDA, 2008; Gunderson, 1988). Workers in chemical manufacturing industries may be exposed to HCB via inhalation or dermal pathways.

HCB is well absorbed after oral administration, distributes widely throughout the body, and accumulates in fatty tissues where it persists for years. HCB is slowly metabolized, and elimination occurs by renal and fecal routes; breast milk is an additional route of elimination in nursing women. Urinary metabolites include pentachlorophenol (PCP), 2,4,5-trichlorophenol (2,4,5-TCP) and 2,4,6-trichlorophenol (2,4,6-TCP) (To-Figueras et al., 1997); these metabolites can also be produced after exposure to other chlorinated compounds (Kohli et al., 1976). Therefore, measuring HCB in serum is a specific indicator of exposure to the parent chemical.

How Hexachlorobenzene Affect People's Health: Human health effects from HCB at low environmental doses or at biomonitored levels from low environmental exposures are unknown. Chronic feeding studies in animals have demonstrated kidney injury, immunologic abnormalities, reproductive and developmental toxicities, and liver and thyroid cancers (ATSDR, 2002). In humans, very high, acute doses produce central nervous system depression and seizures. HCB interferes with normal heme synthesis, which is manifested by increased delta-aminolevulinic acid synthase activity and decreased uroporphyrinogen decarboxylase activity. With chronic exposure, a consequence of these heme abnormalities is a condition known as acquired porphyria cutanea tarda. This condition, as well as hypertrichosis, arthritis, thyromegaly, anorexia, and weakness, were seen in an epidemic of poisoning in Turkey that occurred from 1955 to 1959 when HCB-treated seed grain was diverted for bread production. Infants were exposed transplacentally and through breast milk, and many died before 2 years of age (Peters et al., 1982; Schmid, 1960).

IARC classifies hexachlorobenzene as possibly carcinogenic to humans, and NTP classifies hexachlorobenzene as reasonably anticipated to be a human carcinogen. ACGIH has developed workplace exposure limits for HCB. The U.S. EPA has established a drinking water standard, and the FDA has established a bottled water standard for HCB. More information about external exposure (i.e., environmental levels) and health effects is available from the U.S. EPA at: http://www.epa.gov/pesticides/ and from ATSDR at: http://www.atsdr.cdc.gov/toxprofiles/.

What are the Levels of Hexachlorobenzene in US Population: Serum concentrations reflect the body burden of HCB. HCB levels were generally below the limits of detection in the NHANES 1999–2000 and 2001–2002 subsamples. As a result of the lower limit of detection in NHANES 2003–2004, more HCB

levels were quantified. Age-related increases of HCB in body fat and serum have been consistently noted in general population studies (Becker et al., 2002; Bertram et al., 1986; Glynn et al., 2003). In a representative sample of the 1998 German adult population, HCB levels were directly related to age, and the geometric mean concentration of HCB in whole blood was 0.44 µg/L, lower than the limit of detection (on a lipid adjusted basis) in NHANES 1999–2000 and 2001–2002, but approximately five times higher than the overall geometric mean level in 2003–2004 (Becker et al., 2002). In the 1976–1980 NHANES subsample, HCB detection in serum also was proportional to age, but overall, only 4.9% of participants had quantifiable levels (Stehr-Green, 1989). In Spain, factory workers chronically exposed to HCB and residents near the factory had serum HCB levels that were 150 to 50 times higher, respectively, than the limits of detection (on a whole weight basis) in NHANES 1999–2000 and 2001–2002 (Herrero et al., 1999). Residency near industrial or agricultural areas has been associated with higher serum HCB levels (Barber et al., 2005; Bradman et al., 2006). Over the past two decades, however, declines in background HCB levels ranging from around 50%-90% have been documented in studies using cord blood (Dallaire et al., 2002; Lackman, 2002) and among children (Link et al., 2005); the more recent values in these studies were similar to the lipid adjusted limit of detection in NHANES 1999–2000 and 2001–2002 (Dallaire et al., 2002; Lackmann, 2002; Link et al., 2005).

Finding a measurable amount of hexachlorobenzene in serum does not mean that the level of the hexachlorobenzene causes an adverse health effect. Biomonitoring studies on levels of HCB provide physicians and public health officials with reference values so that they can determine whether people have been exposed to higher levels of hexachlorobenzene than are found in the general population. Biomonitoring data can also help scientists plan and conduct research on exposure and health effects.

For More Information and References*:

Agency for Toxic Substances and Disease Registry (ATSDR).
Toxicological profile for hexachlorobenzene, : http://www.atsdr.cdc.gov/toxprofiles/tp90.html. 4/21/09

Center for Disease Control and Prevention*
National Biomonitoring Program CDC
http://www.cdc.gov/biomonitoring/Hexachlorobenzene_BiomonitoringSummary.html

beta-Hexachlorocyclohexane and gamma-Hexachlorocyclohexane (Lindane)
Hexachlorocyclohexane (HCH), formerly referred to as benzene hexachloride, exists in several isomeric forms, including alpha, beta, gamma, and delta. The gamma isomer, commonly known as lindane, can be used as an insecticide and has been used to kill soil-dwelling and plant-eating insects. The other isomers can be formed during the synthesis of lindane, and have been used either as fungicides or to synthesize other chemicals. Technical grade HCH is a mixture of all four isomers, containing about 64% alpha and 10%-15% gamma isomers. It is no longer produced or sold in the U.S. In 2006, the U.S. EPA cancelled agricultural uses of lindane (ATSDR, 2005). Lindane (1%) lotion and shampoo are available by prescription for single-use application to treat human scabies and head lice.

How People Are Exposed to HCH: HCH isomers, particularly alpha and gamma have been detected widely in air, soil, water, and sediment as a result of historic production and use. As pesticide applications of HCH were increasingly restricted or eliminated, environmental levels declined. Lindane has a half-life of about two weeks in soils and water. HCH does not bioaccumulate to an appreciable extent in plants (ATSDR, 2005). However, HCH isomers are lipophilic, so they can accumulate in fatty tissues of animals. General population exposure to HCH is through the diet. The U.S. FDA pesticide monitoring program has shown a temporal decline in the detection of lindane, from 6% of samples in 1982–1984 to 2% in 1994 (FDA, 2008; Gunderson 1988). Pesticide applicators or agricultural workers could be exposed to HCH by inhalation and dermal pathways.

HCH isomers are absorbed after inhalation, ingestion, or dermal exposure. Distribution is mainly to fatty tissues. After dermal application of lindane 1% lotion, the serum half-life was about 20 hours among children (Ginsburg et al., 1977). The beta isomer accumulates in fatty tissues and is metabolized more slowly, resulting in a half-life of about seven years. HCH isomers are metabolized to chlorophenol metabolites that are excreted in the urine (Angerer et al., 1983). HCH crosses the placenta and is also excreted in breast milk (Radomski et al., 1971; Rogan, 1996; Saxena et al., 1981).

How Can HCH Affect Human Health: Human health effects from HCH isomers at low environmental doses or at biomonitored levels from low environmental exposures are unknown. Acute high dose toxicity in rodents affects the central nervous system, producing decreased activity, ataxia, and seizures. When animals were chronically fed lindane at high doses, enlarged livers, hepatic enzyme induction, and nephropathy developed (IPCS, 2002). Acute high doses of lindane after ingestion or excessive skin application of the 1% lotion have produced seizures in humans, probably by blocking inhibitory neurotransmitters in the central nervous system. Workers who directly handled HCH have complained of headache, paresthesias, tremors, and memory loss (Nigam et al., 1986). OSHA and ACGIH have established workplace standards and guidelines, respectively, for lindane. U.S. EPA has established a drinking water standard, and FDA has established a bottled water standard and food residue tolerances for lindane. IARC classifies hexachlorocyclohexane isomers as possibly carcinogenic to humans, and NTP classifies hexachlorocyclohexane isomers as reasonably anticipated to be human carcinogens. More information about external exposure (i.e., environmental levels) and health effects is available from the U.S. EPA at: http://www.epa.gov/pesticides/ and from ATSDR at: http://www.atsdr.cdc.gov/toxprofiles/.

What are the Levels of HCH in US Population: Because of its longer half-life, beta-HCH may be detected in a higher percentage of the general population than are the other HCH isomers. Studies of general populations have shown declining beta-HCH levels since the 1970s (ATSDR, 2005; Kutz et al., 1991; Link et al., 2005; Radomski et al., 1971; Stehr-Green, 1989; Sturgeon et al., 1998). Additional factors associated with higher beta-HCH levels include rural residence, older age, male sex, and a diet that includes meat (Becker et al., 2002; Kutz et al., 1991; Stehr-Green, 1989).

In NHANES 1999–2000, 2001–2002, and 2003–2004, serum levels of lindane were generally below the limits of detection, which were considerably lower (as much as twentyfold) than mean levels reported in small studies of adults in Spain (Botella et al., 2004) and India (Bhatnagar et al., 2004). In recent years, studies in populations with environmental exposure have reported lindane levels below the limit of detection in most persons (Anderson et al., 1998; Bates et al., 2004; Becker et al., 2002). In population-based studies of New Zealand adults and German adults and children, the maximum and 95th percentile beta-HCH values, respectively, were similar to the 95th percentiles in the Fourth Report. In an earlier (1996–1997) sample of German children, aged 9-11 years, the 95th percentile of beta-HCH levels was twofold to threefold higher than the 95th percentile of 12-19 year olds in the comparable NHANES 2001–2002 survey period (Link et al., 2005). In a small study of adults who consumed sport fish from the Great Lakes, the median beta-HCH levels were similar or slightly higher than the 95th percentile in the Fourth Report (Anderson et al., 1998). A study of Swedish women aged 54 years and older reported a median beta-HCH level that was slightly higher than the geometric mean for women reported in the NHANES 1999–2000 survey period (Glynn et al., 2003). beta-HCH and lindane levels in workers involved in HCH production have been more than 1000-fold higher than the 95th percentile and limit of detection (lipid adjusted), respectively, in the Fourth Report (Nigam et al., 1986; Radomski et al., 1971).

Finding a measurable amount of HCH isomers in serum does not mean that the level of HCH isomers causes an adverse health effect. Biomonitoring studies on levels of HCH isomers provide physicians and public health officials with reference values so that they can determine whether people have been exposed to higher levels of HCH isomers than are found in the general population. Biomonitoring data can also help scientists plan and conduct research on exposure and health effects.

For More Information and References*:

Agency for Toxic Substances and Disease Registry (ATSDR).
Toxicological profile for hexachlorocyclohexanes, http://www.atsdr.cdc.gov/toxprofiles/tp43.html. 4/21/09

Center for Disease Control and Prevention*
National Biomonitoring Program
http://www.cdc.gov/biomonitoring/Hexachlorocyclohexane_BiomonitoringSummary.html

Dichlorodiphenyltrichloroethane (DDT) and dichlorodiphenyldichloroethene (DDE)
Dichlorodiphenyltrichloroethane (DDT) is an insecticide used in agriculture. The United States banned the use of DDT in 1972, but some countries still use the chemical. DDT has also been used in the past for the treatment of lice. It is still in use outside the United States for the control of mosquitoes that spread malaria. DDT and its related chemicals persist for a long time in the environment and in animal tissues.

How People Are Exposed to DDT: People are most likely to be exposed to DDT from foods, including meat, fish, and dairy products. DDT can be absorbed by eating, breathing, or touching products contaminated with DDT. In the body, DDT is converted into several breakdown products called metabolites, including the metabolite dichlorodiphenyldichloroethene (DDE). DDT and DDE are stored in the body's fatty tissues. In pregnant women, DDT and DDE can be passed to the fetus. Both chemicals are found in breast milk, resulting in exposure to nursing infants.

How DDT Affects People's Health: Human health effects from DDT at low environmental doses are unknown. Following exposure to high doses, human symptoms can include vomiting, tremors or shakiness, and seizures. Laboratory animal studies showed effects on the liver and reproduction. DDT is considered a possible human carcinogen.

Levels of DDT and DDE in the U.S. Population: In the Fourth National Report on Human Exposure to Environmental Chemicals (Fourth Report), CDC scientists measured DDT and its metabolite DDE in the serum (a clear part of blood) of at least 1,956 participants aged 12 years and older who took part in CDC's National Health and Nutrition Examination Survey (NHANES) during 2003–2004. Prior survey periods of 1999–2000 and 2001–2002 are also included in the Fourth Report. By measuring DDT and DDE in the serum, scientists can estimate the amounts of these chemicals that have entered people's bodies.
A small portion of the population had measureable DDT. Most of the population had detectable DDE. DDE stays in the body longer than DDT, and DDE is an indicator of past exposure. Blood serum levels of DDT and DDE in the U.S. population appear to be five to ten times lower than levels found in smaller studies from the 1970s.
Finding measurable amounts of DDT and DDE in serum does not mean that the levels of these chemicals cause an adverse health effect. Biomonitoring studies of serum DDT and DDE can provide physicians and public health officials with reference values so that they can determine whether people have been exposed to higher levels of DDT and DDE than are found in the general population. Biomonitoring data can also help scientists plan and conduct research on exposure and health effects.

For More Information:
Agency for Toxic Substances and Disease Registry
Public Health Statement for DDE and DDT, http://www.atsdr.cdc.gov/toxprofiles/phs35.html

E**nvironmental Protection Agency**
DDT Fact Sheet, http://www.epa.gov/pbt/pubs/ddt.htm

Center for Disease Control and Prevention
National Biomonitoring Program**,** http://www.cdc.gov/biomonitoring/DDT_BiomonitoringSummary.htm

Chlordane and Heptachlor

Chlordane and heptachlor are pesticides that were used in agriculture in the United States from the 1950's until the 1980's. Chlordane was used in homes and for termite control. Heptachlor was used as a soil and seed treatment and for termite control. Since 1992, the use of heptachlor has been limited to treatment of fire ants near utility equipment.

Both pesticides can remain in treated soils, in agricultural runoff water, and near factories where they were manufactured. Chlordane and heptachlor can be found in the air and dust of buildings long after treatment for termites or insects was performed.

How People Are Exposed to Chlordane and Heptachlor: People are usually exposed to these chemicals by eating foods high in fat, such as meat, fish, and dairy products. Pregnant women may pass these chemicals to the fetus, and after birth, chlordane and heptachlor may be passed to infants through breast milk. Chlordane and heptachlor are converted in the body into chemicals called metabolites. These chemicals leave the body slowly over a period of months to years.

How Chlordane and Heptachlor Affect People's Health: The human health effects from low environmental exposures to these chemicals are unknown. Short-term large exposures to either chlordane or heptachlor can cause seizures and injure the liver. Both chlordane and heptachlor are considered possible cancer-causing chemicals in humans.

Levels of Chlordane and Heptachlor Metabolites in the U.S. Population:In the Fourth National Report on Human Exposure to Environmental Chemicals (Fourth Report), CDC scientists measured the metabolites of chlordane and heptachlor in the blood serum (the clear part of blood) of 1,955 participants aged 12 years and older who took part in the National Health and Nutrition Examination Survey (NHANES) during 2003–2004. Prior survey periods of 1999–2000 and 2001–2002 are also included in the Fourth Report. By measuring the metabolites of chlordane and heptachlor in serum, scientists can estimate the amounts of these chemicals that have entered people's bodies. The Fourth Report shows that the U.S. population continues to have measureable amounts of chlordane and heptachlor metabolites in their bodies.

Finding measurable amounts of the metabolites of chlordane and heptachlor in serum does not mean that the levels cause an adverse health effect. Biomonitoring studies on levels of the metabolites of chlordane and heptachlor can provide physicians and public health officials with reference ranges so that they can determine whether people have been exposed to higher levels of heptachlor and chlordane than are found in the general population. Biomonitoring data can also help scientists plan and conduct research on exposure and health effects.

We will measure: Oxychlordane**,** *trans*-Nonachlor**,** Heptachlor epoxide

For More Information:
Agency for Toxic Substances and Disease Registry
Public Health Statement for Chlordane, http://www.atsdr.cdc.gov/toxprofiles/phs31.html
Public Health Statement for Heptachlor/Heptachlor Epoxide; http://www.atsdr.cdc.gov/toxprofiles/phs12.html

Environmental Protection Agency

Technology Transfer Network Air Toxics Web Site: Chlordane,
http://www.epa.gov/ttn/atw/hlthef/chlordan.html

Center for Disease Control and Prevention
National Biomonitoring Program
http://www.cdc.gov/biomonitoring/ChlordaneHeptachlor_BiomonitoringSummary.html

Aldrin and Dieldrin

Aldrin and dieldrin are no longer produced or used in the U.S. From the 1950s to 1970, both chemicals were applied mainly as a soil insecticide or seed dressing for food and commodity crops. Dieldrin was also used for mothproofing clothes and carpets. In tropical countries, dieldrin was used as a residual spray in residential dwellings to control vector-borne diseases such as malaria. The U.S. EPA cancelled agricultural uses of both pesticides in 1970; termiticide uses were cancelled in 1987. Aldrin is readily converted to dieldrin in the environment and in plants that take up the chemical. Aldrin volatilizes after agricultural soil applications or is converted to dieldrin, which volatilizes more slowly. These chemicals persist in the environment and bioaccumulate in foods (Jorgenson 2001; USGS, 2007). Aldrin is rarely detected in plants or animal tissues, but dieldrin has been detected in meats, dairy products, and in crops grown in soils that have been contaminated, usually by application, manufacturing, or disposal.

How People Are Exposed to Aldrin and Dieldrin: General population exposure to these chemicals occurs through the diet, and detection of dieldrin residue in foods has decreased over time (FDA, 2008). Inhalation exposure may occur among people living in residences where aldrin was applied historically as a pesticide. Aldrin and dieldrin are absorbed following ingestion, inhalation, and dermal application. After absorption, aldrin is metabolized to dieldrin so rapidly that aldrin is rarely detected. Dieldrin accumulates in fatty tissues, and its metabolites are excreted in bile and feces (ATSDR, 2002). It is also excreted in breast milk and can cross the placenta. The elimination half-life of dieldrin is approximately 1 year (IPCS, 1989; Jorgenson 2001).

How Can Aldrin and Dieldrin Affect Human Health: Human health effects from aldrin and dieldrin at low environmental doses or at biomonitored levels from low environmental exposures are unknown. At high doses, aldrin and dieldrin block inhibitory neurotransmitters in the central nervous system (Narahashi et al., 1992). This blocking action can cause abnormal excitation of the brain, leading to symptoms such as headache, confusion, muscle twitching, nausea, vomiting, and seizures. When fed to experimental animals, both aldrin and dieldrin caused liver enlargement and liver tumors; dieldrin at higher doses caused irritability, tremors, and occasionally, seizures (Smith, 1991). When dieldrin was fed to pregnant rodents, the offspring had altered CNS neurotransmitter levels (Sanchez-Ramos et al., 1998) and behavioral changes (Carlson and Rosellini, 1987). Studies done in vitro showed that dieldrin binds to estrogen receptors (Soto et al., 1995), but no estrogenic effect was noted in a study that used cultured cells (Tully et al., 2000). Epidemiologic and animal studies have not conclusively associated dieldrin exposure with risk for developing Parkinson's disease (Corrigan et al., 2000; Kanthasamy et al., 2005; Li et al., 2005).

The U.S. EPA has established environmental standards for aldrin and dieldrin, and the FDA monitors foods for pesticide residues. OSHA has established workplace exposure standards for aldrin and dieldrin. IARC has determined that aldrin and dieldrin are not classifiable with regard to human carcinogenicity. Information about external exposure (i.e., environmental levels) and health effects is available from ATSDR at: http://www.atsdr.cdc.gov/toxprofiles/.

What are the Levels of Aldrin and Dieldrin in US Population: In the NHANES 2001–2002 and 2003–2004 subsamples, serum aldrin levels were below the limit of detection, similar to results in a subsample of NHANES II (1976–1980) (Stehr-Green, 1989). Levels of aldrin also were not detectable in 1996–1997 pooled samples from New Zealand adults (Bates et al., 2004).

Serum dieldrin levels at the 95th percentile in NHANES 2001–2002 and 2003–2004 subsamples were approximately ten times lower than the corresponding percentile measured in NHANES II (1976–1980), in which only 10.6% of the subsample had dieldrin levels above the limit of detection (Stehr-Green 1989). The median level in pooled samples from New Zealand adults obtained in 1996–1997 was generally similar to the 90th percentile for adults in the Fourth Report (Bates et al., 2004). In samples obtained between 1973 and 1991 from Norwegian women, the median serum dieldrin level was generally similar to the 90th percentile for females in the Fourth Report (Ward et al., 2000). Danish women whose serum was collected in 1976 had a median dieldrin level near the 95th percentile for females in the Fourth Report (Hoyer et al., 1998). In a study of pesticide applicators with occupational exposure to aldrin, median levels of dieldrin were more than thirtyfold higher than the 95th percentile in the NHANES 2001–2002 and 2003–2004 subsamples (Edwards and Priestly 1994).

Finding a measurable amount of aldrin or dieldrin in serum does not mean that the level of aldrin or dieldrin causes an adverse health effect. Biomonitoring studies on levels of aldrin and dieldrin provide physicians and public health officials with reference values so that they can determine whether people have been exposed to higher levels of aldrin or dieldrin than are found in the general population. Biomonitoring data can also help scientists plan and conduct research on exposure and health effects.

We will measure: Dieldrin

For more information and References*:

Agency for Toxic Substances and Disease Registry (ATSDR)
Toxicological profile for aldrin/dieldrin [online], http://www.atsdr.cdc.gov/toxprofiles/tp1.html. 4/21/09

Center for Disease Control and Prevention*
National Biomonitoring Program
http://www.cdc.gov/biomonitoring/AldrinDieldrin_BiomonitoringSummary.html

Mirex

Mirex has not been produced or used in the U.S. since 1977. Formerly, its major uses were as a flame retardant additive and as a pesticide to kill fire ants and yellow jackets in the southeastern U.S., where it was applied directly to soil and by aerial spraying. Mirex binds strongly to soil, where it has a half-life of 12 years; it is a highly persistent chemical in the environment. Mirex has been detected in air, soil, sediments, water, aquatic organisms, animals, and foods. Mirex contamination of Lake Ontario and adjacent waterways has been well documented (ATSDR, 1995). The most likely sources of human exposure to mirex are eating fish from contaminated water or living in areas with soil contaminated by historic mirex manufacturing, disposal, or pesticide application. Some states and the U.S. EPA have issued public health advisories or warnings that fish from contaminated lakes and rivers may contain mirex. Occupational exposure is limited to workers at sites where mirex contamination is present.

How People Can Get Exposed to Mirex: Mirex is absorbed through the skin and from the gastrointestinal tract, after which it is widely distributed in the body and stored in fat. Ingested mirex that is not absorbed is eliminated in the feces within about 48 hours. Mirex is not metabolized in the body. In studies conducted in the 1970's and 1980's, mirex was detected in human adipose samples, especially those from persons living in the southeastern U.S. (Kutz et al., 1985, 1991). Mirex can cross the placenta and be excreted in breast milk, resulting in exposure to newborns and nursing infants.

How Can Mirex Affect Human Health: Human health effects from mirex at low environmental doses or at biomonitored levels from low environmental exposures are unknown. Laboratory animals fed high doses developed liver enlargement and liver tumors; reproductive toxicity included decreased fertility and testicular damage. In addition, developmental abnormalities including cataracts and edema in the offspring have been reported (ATSDR, 1995; Smith, 1991). The U.S. EPA has established environmental standards for mirex, and the FDA monitors foods for pesticide residue and has established an action level for mirex in fish tissue. IARC classifies mirex as possibly carcinogenic to humans, and NTP classifies mirex as reasonably anticipated to be a human carcinogen. More information about external exposure (i.e., environmental levels) and health effects is available from the ATSDR at: http://www.atsdr.cdc.gov/toxprofiles/.

What are the Levels of Mirex and in US Population: In the NHANES 1999–2000, 2001–2002, and 2003–2004 subsamples, as well as in a subsample of NHANES II (1976–1980) participants, serum mirex levels were generally below the limits of detection (Stehr-Green, 1989). Fishermen in New York who consumed Great Lakes sport fish had median levels of lipid-adjusted serum mirex that were lower than the 95th percentile value among males the NHANES 2001–2002 subsample (Bloom et al., 2005). In samples obtained between 1994 and 1997, Inuit mothers from three Arctic areas had geometric mean serum mirex levels that were threefold to sevenfold higher than non-Inuit mother from other Arctic regions. The geometric mean mirex levels of the Inuit mothers were 8, 7.8, and 4.7 ng/g of lipid, which is approximately twofold to threefold lower than the 90th percentile for females in the NHANES 2001–2002 subsample but similar to 95th percentile for females in the NHANES 2003–2004 subsample (Van Oostdam et al., 2004).

Finding a measurable amount of mirex in serum does not mean that the level of mirex causes an adverse health effect. Biomonitoring studies on levels of mirex provide physicians and public health officials with reference values so that they can determine whether people have been exposed to higher levels of mirex than are found in the general population. Biomonitoring data can also help scientists plan and conduct research on exposure and health effects.

For More Information and References*:

Agency for Toxic Substances and Disease Registry (ATSDR).
Toxicological profile for mirex and chlordecone, http://www.atsdr.cdc.gov/toxprofiles/tp66.html. 4/21/09

Center for Disease Control and Prevention*
National Biomonitoring Program,
http://www.cdc.gov/biomonitoring/Mirex_BiomonitoringSummary.html
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