

# The Green Housing Study

## Supporting Statement (Part B)

October 20, 2011

Project Official:  
Ginger L. Chew, ScD  
Principal Investigator  
Healthy Homes and Lead Poisoning Prevention Branch  
National Center for Environmental Health  
U.S. Centers for Disease Control and Prevention (CDC)  
4770 Buford Hwy., N.E., MS-F60  
Atlanta, GA 30341  
Tel: (770) 488-3992  
Fax: (770) 488-3635  
[gjc0@cdc.gov](mailto:gjc0@cdc.gov)

## B. Collections of Information Employing Statistical Methods

### B.1. Respondent Universe and Sampling Methods

The purpose of this study is to provide insight into the potential implications of green renovations for the health of young asthmatics who live multifamily HUD-subsidized housing in the United States and US territories. According to HUD, 970,532 households live in public housing in the United States (HUD 2009). The number of M2M properties is in flux according to market forces and other factors such as landlord motivations for participation; however, it is estimated that since 1997, 1600 developments (with approximately 100 units each) have been renovated through the M2M Green Initiative <http://www.hud.gov/offices/hsg/omhar/paes/greenini.cfm>. Collecting data from asthmatic children in all housing units being renovated would be too burdensome, expensive, and logistically impractical. We will include a targeted non-probability sample of 832 homes in 13 cities for this study methodology. HUD had selected housing developments for green renovations projects prior to the inception of this proposed study based upon specific requirements (e.g., use of low VOC materials, use of energy efficient appliances). Figure 8 illustrates the sampling process. Since the housing developments were already selected based on grant awards, random assignment of the green intervention was not possible for this study.

The selection of the cities is based upon the following:

1. City must have one or more housing developments which are receiving a HUD-subsidized green-renovation.
  - a. These renovations must occur within the timeline of our study period (5 years, although, we will ask OMB for a continuation prior to the expiration of the initial 3-year OMB approval)
  - b. Housing developments should have many apartments which will undergo the green renovations. Smaller housing developments would severely hamper recruitment of our targeted sample size in each city. However, we will consider cities which have several housing developments with a smaller number of apartments, given that the housing developments will undergo renovations within 6 months of each other.
  - c. Green renovations must meet inclusion criteria: Low VOC materials and Integrated Pest management (IPM).
2. The housing renovations within the city must occur in areas with high prevalence (i.e., greater than the national average, currently 9.1%) of childhood asthma (based upon National Health Interview Survey data, (Akinbami et al. 2009)). This is to enhance the potential pool of study participants. Areas of lower asthma prevalence would severely hamper recruitment of our targeted sample size in each city.
3. Cities are located in different regions of the country and/or represent different types of housing stock.

The design being used allows us to provide insight into the societal benefits of green housing on low income families with asthmatic children. However, it will not be generalizable to

respondents or even geographically or demographically defined subgroups due to the fact that both the applicants of the HUD awards and the households themselves are self-selected. Specifically, the design does not allow generalizations based on city, type of locations (rural, suburban), climactic regions (e.g., desert, arctic), or ethnicities. Furthermore, this study will systematically exclude certain subsets of the population for logistical reasons. Specifically:

i. Public housing is comprised mostly of 3 main ethnicities: white, African-American, and Latino (HUD, 2009).

ii. Our main health endpoint, asthma, is highest among Latinos and African-Americans. While several childhood asthma studies have focused on some minority populations in the United States (African American and Latino), only recently have investigators focused studies of Asian populations. In the Boston Chinatown neighborhood, researchers found a higher prevalence of asthma for children born in the US as compared to those who were foreign-born in an Asian population, enriched with recent Chinese immigrants (Brugge et al., 2007). These results confirm findings in a similar Asian population from the same community (Greenfield et al., 2005).

iii. We do not have the capacity to translate into all languages. However, we determined that it would be beneficial to include Spanish and Chinese translations for the reasons mentioned above. In meetings with stakeholders at our first potential study site, Boston, we found that they have a substantial Chinese population (along with Latino and African-American). The tenants' organization asked if we would recruit the Chinese residents too and if we could translate all of our materials into Chinese. We believe that the tenants' organization's request is reasonable. Furthermore, in other potential study site locations (e.g., Los Angeles, New York, San Francisco), Chinese language translation might also be relevant.

We assume an 80% participation rate for the eligible residents for the collection as a whole (as described in Part B, section 3).

The design is stratified by city. As discussed below, one pair of housing developments will be chosen in each of 13 cities that meet the criteria delineated in section B1. We will frequency match green intervention and comparison homes by HUD-subsidized housing development, asthma status of children, age group of asthmatic children (7-12 years) and primary language spoken by mother/primary caregiver of the asthmatic child. We are not matching on ethnicity *per se*; however, much of the low-income housing in inner-city communities tend to be segregated to some extent, by race/ethnicity (Acevedo-Garcia and Lochner 2003). We will record race/ethnicity in our questionnaire and adjust accordingly in our analysis. As mentioned earlier, this selection will be limited by the availability of the ongoing HUD renovation efforts. There are no other problems requiring specialized sampling procedures. The data collection plan requires only one series of data collection within a one-year follow-up period.

Sample size overviews: Our calculations estimate that 416 subjects/study arm (i.e., green vs. comparison homes) must be recruited in order to achieve sufficient statistical power to statistically differentiate between the study arms (this paragraph outlines the calculations supporting this estimate, with details in subsequent paragraphs of this section; see also figure below). In order to have sufficient power to detect meaningful differences in both environmental measurements and health outcomes between the arms, we began by calculating sample sizes based on each of these measures.

1. Our sample calculations for environmental measurements (see Table 21) were based on cockroach allergen data in a repeated measures study of the effect of an integrated pest management (IPM) intervention, which indicate that 13 buildings would be necessary in each of the two arms of the study, assuming that 25 subjects could be recruited for each building, yielding 325 subjects/study arm to provide adequate statistical power for environmental measurements.
2. Our sample calculations for health outcomes (see Table 22) were based on asthma in children subjected to a multifactorial intervention (i.e., education, mattress covers, IPM, and HEPA filter units) in a repeated measures study. These data indicate that 274 subjects/study arm would be needed to provide adequate statistical power for asthma outcomes.

Therefore, since we desired sufficient power to detect meaningful differences in both 1) environmental measurements and 2) health outcomes, we selected the larger of the two estimates — 325 subjects/study arm — as the minimum sample size (see Figure 7). In addition, we augmented this number in order to account for an anticipated 20 percent loss to follow-up over a one-year period. After rounding up where necessary, this increased the sample size to 416 subjects/study arm, comprising 32 subjects (one subject per apartment) in each of 13 buildings in each study arm. The total sample size for the study across both study arms is 832 subjects. The details of our sample size calculations are listed below and the equations that were used were from a book on longitudinal data analysis by Diggle, Liang, and Zeger (1994).

Figure 7. Summary of sample size

Sample size calculations for the overall difference between environmental exposures in green vs. comparison homes can be given as a simple test of two proportions and means; however a specific difficulty arises when trying to adjust for temporal and spatial correlations between measurements. A study that had enough measurements to assess spatial and temporal correlation was an integrated pest management (IPM) study conducted in New York City (Chew et al. 2006; Kass et al. 2009). We have used the design effect from this study to estimate the number of clusters (or buildings) needed to detect differences in cockroach allergen because IPM is also one of the main green characteristics in the Green Housing Study. Assumptions from the aforementioned study comparing IPM to non-IPM homes are listed below:

- 1 13 buildings
  - 2 About half were treatment and the other half comparison
  - 3 3 repeated measures at: baseline (before IPM), 3months later (post-IPM), and 6months later (post-IPM)
  - 4 On average, 25 apartments within each building were measured
  - 5 For the comparison homes, the correlations between baseline and 3-month follow-up cockroach allergen measurements and 3-month and 6 months follow-up measurements were approximately equal to 0.5.
- Design effect due to clustering = 3.62

Equation 1. Sample size for repeated measures – cockroach allergen.

$$m = D * [2(z_{\alpha} + z_{\beta})^2 \{1 + (n - 1)\rho\}] / (n\Delta^2)$$

m = number in each group (e.g., intervention and non-intervention)

n = number of repeated measurements (equals 3 in this scenario)

$z_{\alpha}$  = Z score for alpha = 0.05

$z_{\beta}$  = Power, set at 0.80

$\rho$  = correlation among repeated observations

$\Delta = d / \sigma$  where d is the smallest meaningful difference and  $\sigma$  is the standard deviation

D = Design effect due to clustering of apartments within buildings (This was not in the formula used by Diggle et al. 1994, but was added to adjust for clustering expected in our study)

Note: we also assumed a design effect (e.g., increase (multiplicative) sample needed because of the effects of clustering) equal to 3.62. This is the ratio in clustering sampling variance divided by the simple random variance (of the same size) (i.e., the denominator without clustering taken into account).

When calculating the sample sizes, we used two standardized effect sizes based on the IPM study: 0.37 and 0.30. The effect size of 0.37 is based on the ability to detect a difference of 0.8148 ln units of the *Blattella germanica* cockroach allergen (i.e., Bla g 2) and a standard deviation of ~ 2.2. The effect size of 0.30 is based on the ability to detect a difference of 0.649 ln units of Bla g 2 and a standard deviation of 2.2. We also assumed an alpha of 0.05 and a power of 80%). The sample size based on changing the expected correlation between repeated measures would result is presented in Table 21.

Assuming a correlation of 0.5 we get the following:

- 1 With assumption 1, we would need 16 buildings (8 green and 8 comparison) with at least 25 apartments per building
- 2 With assumption 2, we would need 26 buildings (13 green and 13 comparison) with at least 25 apartments per building.

Table 21. Sample size requirements for number of buildings.

| Correlation between repeated measures | Sample size requirements<br>(number of buildings) |                     |
|---------------------------------------|---|---------------------|
|                                       | Assuming<br>Delta=0.377                           | Assuming Delta=0.30 |
| 0.2                                   | 6   | 9                   |
| 0.3                                   | 7   | 11                  |
| 0.4                                   | 8   | 12                  |
| 0.5                                   | 8   | 13                  |
| 0.6                                   | 9   | 15                  |
| 0.7                                   | 10  | 16                  |
| 0.8                                   | 11  | 17                  |

\*samples size is for each group (e.g., 8 buildings means 8 comparison and 8 green buildings)

We also estimated the sample size for detecting differences in pesticides and VOCs. To date, there is only one study of an intervention to decrease pesticide exposures that used objective measurements of pesticide levels in residential homes in a non-agricultural environment (Williams et al., 2006). This study was conducted in homes of Latina and African-American women living in low-income housing in New York. In the study, 25 homes underwent IPM as an intervention. The pesticide synergist, piperonyl butoxide, is unique to pyrethroid pesticides and this was an analyte that was measured in the study's air samples. We used their pre- and post piperonyl butoxide concentrations (pre = mean  $1.66 \pm$  s.e.  $0.71 \text{ ng/m}^3$  vs. post = mean  $0.8 \pm$  s.e.  $0.22 \text{ ng/m}^3$ ) for our calculations of sample size which are shown in Table 22.

To date, there is only one study comparing the VOC levels in newly-built green homes and conventionally-built homes; therefore, this was not a renovation like our proposed study. We calculated the sample size based on their measurements of formaldehyde in the two types of homes in their study. This study was conducted in Finland. In the study, 6 apartments in each type of building had air measurements for formaldehyde (green-built: mean =  $13 \text{ } \mu\text{g/m}^3$ , s.d. = 4 vs. conventionally-built: mean  $23 \text{ } \mu\text{g/m}^3$ , s.d. = 5). We calculated the sample size necessary to detect a decrease in 50%, 25%, and 15%, of the difference in formaldehyde levels observed in their two study groups (see Table 22).

Devos et al (1990) have suggested that the minimum level of an indoor irritant be set with a safety factor of 40 (Devos, Patte et al. 1990). Given that the American Conference of Governmental Industrial Hygienists (ACGIH) threshold limit value is  $368 \text{ } \mu\text{g/m}^3$ , a minimum level of formaldehyde below which no irritant effects are expected is  $9.8 \text{ } \mu\text{g/m}^3$  (which is the CDC/ATSDR Minimum Risk Level, <http://www.atsdr.cdc.gov/ToxProfiles/tp.asp?id=220&tid=39> ).

Table 22. Calculations of samples sizes for VOCs and pesticides

| Analyte                              | Design Effect | Effect Size ( $\Delta$ ) | Required Sample size (in each group) |
|--------------------------------------|---------------|--------------------------|--------------------------------------|
| Formaldehyde                         |               |                          |                                      |
| - 15% : 1.5 $\mu\text{g}/\text{m}^3$ | 3.6           | 0.33                     | 408                                  |
| - 25% : 3.5 $\mu\text{g}/\text{m}^3$ | 3.6           | 0.77                     | 75                                   |
| - 50% : 5 $\mu\text{g}/\text{m}^3$   | 3.6           | 1.10                     | 37                                   |
| (based on Tuomainen et al, 2003)     |               |                          |                                      |
| Piperonyl butoxide                   |               |                          |                                      |
| 0.8 $\text{ng}/\text{m}^3$ decrease  | 3.6           | 0.33                     | 418                                  |
| (based on Williams et al, 2006)      |               |                          |                                      |

\*  $\alpha = 0.05$ ,  $1-\beta = 0.80$

Summary of sample size for environmental exposures: Of the intervention studies relevant to green housing, the sample size calculations based upon the cockroach allergen intervention study provided the most information to inform our estimates of sample size required for the Green Housing Study. Because Dr. Chew was a co-author on the manuscript and had analyzed the cockroach allergen samples in her laboratory, she had access to the repeated measurements database and this helped to guide our design effect due to clustering of apartments within buildings. The other papers did not have repeated measurements (thus the variance estimates were rather wide) and they also had more restrictive groups (e.g., nonsmoking pregnant women, Finnish families living in newly-constructed apartments) than is planned in the Green Housing Study. Thus, we believe that our estimates are conservative.

Sample size for assessing asthma outcomes: The calculation for assessing differences in health markers were based upon a multi-site asthma intervention study (Morgan et al. 2004). In this study, 407 asthmatic children with the multi-factorial intervention (asthma trigger education, mattress covers, IPM, HEPA filter units) had fewer days (2.62 days  $\pm$  0.12) than those (n=414) without the intervention (3.21 days  $\pm$  0.13). Table 15 shows sample sizes with different assumptions of effect sizes using equation 2.

Equation 2. Sample size for asthma morbidity outcomes.

$$m = D * [2(z_{\alpha} + z_{\beta})^2 \{1 + (n - 1)\rho\}] / (n\Delta^2)$$

m = number in each group (e.g., intervention and non-intervention)

n = 1 (note: for differences of differences, we assumed a value of 1)

$z_{\alpha}$  = Z score for alpha = 0.05

$z_{\beta}$  = Power, set at 0.80

$\rho$  = correlation among repeated observations

$\Delta = d / \sigma$  where d is the smallest meaningful difference and  $\sigma$  is the standard deviation

D = Design effect due to clustering within 13 sites (This was not in the formula used by Diggle et al.1994, but was added to adjust for clustering expected in our study). Based upon the Kwon et al (2003) paper that showed a design effect of 1.5 was helpful for designing cluster studies to assess asthma outcomes in national surveys (e.g., BRFSS and NHANES), we assumed a slightly smaller design effect equal 1.2 due to the expected low average number of children per cluster).

Table 23. Sample size requirements for number of children with asthma.

| Effect size (i.e., delta) | Sample size requirements<br>(number of asthmatic children) |                         |
|---------------------------|--|-------------------------|
|                           | in Green buildings   | in Comparison buildings |
| 0.20                      | 274  | 274                     |
| 0.232*                    | 274  | 274                     |
| 0.30                      | 206  | 206                     |
| 0.35                      | 151  | 151                     |
| 0.40                      | 116  | 116                     |

\* Based on observed effect size from Morgan et al (2004) study.

We used Equation 3 to calculate the sample size based on binary outcomes. The assumptions for the equation were based upon an intervention study in Seattle Public housing (Krieger et al., 2005). The Seattle researchers had n= 110 in a high-intensity intervention group and n = 104 in a low-intensity intervention group follow. They assessed the percentage of children in each group with urgent health service use in the past 2 months. Taking the difference between baseline and exit measurements of the two proportions for high-intensity (23.4% - 8.4% = 15% difference) and low-intensity (20.2% - 16.4% = 3.8% difference), we calculated n= 102 in each study group.

Equation 3. Sample size for binary asthma morbidity outcomes.

$$m = \left[ \left[ (z_{\alpha} \{ 2\bar{p}\bar{q} (1 + (n - 1)\rho) \})^{\frac{1}{2}} + z_Q \{ (1 + (n - 1)\rho) (p_A q_A + p_B q_B) \}^{\frac{1}{2}} \right]^2 \right] / nd^2$$

m = number in each group (e.g., intervention and non-intervention)

n = 1 (note: for differences of differences, we assumed a value of 1)

$z_{\alpha}$  = Z score for alpha = 0.05

$z_Q$  = Power, set at 0.80

$\rho$  = correlation among repeated observations

$p_A$  = proportion of Group A

$p_B$  = proportion of Group B

$q_A$  = 1- proportion of Group A

$q_B$  = 1- proportion of Group B

$\bar{p}$  =  $(p_A + p_B) / 2$

$\bar{q}$  =  $1 - \bar{p}$

d = is the smallest meaningful difference between proportions

D = Design effect due to clustering (This was not in the formula used by Diggle et al. 1994, but was added to adjust for clustering expected in our study). Based upon the Kwon et al paper (2003) that showed a design effect of 1.5 was helpful for designing cluster studies to assess asthma outcomes in national surveys (e.g., BRFSS and NHANES), we assumed a slightly smaller design effect equal 1.2 due to the expected low number of expected of average children per cluster).



## B.2. Procedures for the Collection of Information

The characteristics of study participants that will be included are: 1) Children age 7-12 years with asthma (note: The child must have been diagnosed with asthma by a physician and have had asthma-related symptoms (wheezing, slow play or night awakening) during the past 6 months), and 2) mothers/ primary caregivers of enrolled children. Also, the mother/ primary caregiver must speak English, Spanish, or Chinese to be included in the study and the enrolled participants must live in the home (from which environmental samples will be collected) on average 7 days per week.

Upon notification from HUD that a participating housing complex is about to begin rehabilitation, CDC will contact local academic institutions and departments of health in order to mobilize the Green Housing Study in that location. We envision that together with HUD and local academic investigators at the selected sites, CDC will convene town meetings at each participating complex to describe the study to residents, answer questions, and invite their participation. Depending upon the number of residents who initially volunteer at the town hall, we will convene additional town hall meetings to augment participation. Residents who express interest in the study can contact the site projector coordinator either at the town hall meetings or by telephone. Subsequently, the trained staff will schedule a home visit with the residents. For quality control purposes, teams of two trained staff will visit the home to collect questionnaire data via an in-person interview and perform environmental sampling. The environmental sampling technician will review the questionnaire information that the other technician obtained during the interview with the study participant. Also, the database entry screen will have validation checks (e.g., number of reported asthma symptoms cannot equal a negative number)

**Statistical analysis:** The main variable of interest is the type of home (green vs. comparison); however, there may be different permutations within green housing. For example, HUD has two levels of green which are based upon the acceptance of HUD-approved recommendations: Level 1) landlord agrees to implement at least 75% of the dollar amount of green repairs and improvements; and Level 2) landlord agrees to implement at least 50% of the dollar amount. While discretizing the green rehabilitation into Level 1 and Level 2 categories could simplify our analysis, we acknowledge that the two different levels do not necessarily capture green materials or practices that are potentially related to health. For example, a green home could have low VOC paint, or low VOC carpet, or replace the kitchen cabinets with low VOC materials, or have some combination of these activities.

**Allergens in the homes:** Variables related to indoor allergens in the homes may take the form of continuous measures of specific allergens or of indicator variables for the presence or absence of certain allergens or combinations of allergens. Allergen concentrations will be reported as  $\mu\text{g}$  of allergen per g of collected dust and  $\mu\text{g}$  of allergen per unit area vacuumed.

**VOCs and pesticides in the homes:** Variables related to VOCs (whether total or speciated) and pesticides (pyrethroids, propoxur, and piperonyl butoxide) in the homes may take the form of continuous measures or indicator variables for the presence or absence of certain chemicals or combinations of chemicals. Concentrations will be reported as ppm (and also  $\mu\text{g}/\text{m}^3$ ) in the case of the VOCs and  $\mu\text{g}/\text{g}$  in the case of the pesticides.

**Conditions of the home environments:** Factors that may influence the presence and levels of allergens, VOCs, and pesticides include: the presence of carpets; pests; housing type and age,

average winter temperature and relative humidity, air exchange rates.

Wheeze /asthma severity: This information may be used in the form of categorical and continuous variables (number of emergency room visits for asthma, use of asthma medications, lost school days). Nights awakened by asthma, and spirometry measurement such as FEV1 and FEF<sub>25-75%</sub>).

Additional environmental and host factors for disposition to wheeze/asthma: Other risk factors for the main outcomes of interest include: environmental tobacco smoke; acute respiratory illnesses; gender; socioeconomic status of primary caregiver; degree of acculturation (operationalized); and deficiencies in access to and quality of health care. Many of these factors allow for a variety of formulations. Environmental tobacco smoke, for example, may be analyzed as an indicator variable for the presence or absence of smoking in the home, as count data for the number of smokers in the home, or as a continuous variable for the number of cigarettes smoked per day in the home. The choice of formulation of risk factors will be driven by the aim of clarifying the main relationships of interest, for example the role of allergens in the development of early allergic sensitization and asthmatic airways disorders.

Descriptive statistics: Study participants will be characterized with regard to demographic variables such as age, gender, and race; clinical variables such as symptom/medication use frequency, healthcare utilization, allergy sensitivity and pulmonary function, and environmental variables such as indoor allergens (cockroach, mouse, cat, and dust mite). Categorical variables will be summarized by frequencies, while continuous variables will be summarized by mean, standard deviation, median, and range. Levels of mold, indoor allergens, pesticides, and VOCs will be log-transformed to compute geometric means and geometric standard deviations. Where appropriate, other transformations or non-parametric analysis methods will be used.

Regression models: In general, for the regression analyses, primary interest lies in the coefficients for the binary "exposure" variable (green vs. comparison). The regressions will also include background variables such as pesticide, VOC, and allergen levels; these variables are included to adjust for differences between households, and we are particularly interested in the coefficients. We will also include interactions between exposure and the background variables. Significant coefficients for these interactions are important because they imply that the exposure has a larger effect under some conditions in comparison to others. In addition, it will be important to consider nonlinear models to allow, for example, for a threshold of allergen exposure.

In the case of dichotomous outcomes, multiple logistic regression will be used to calculate odds ratios (in the case of rare events such as overnight hospitalizations due to asthma attacks). When rare events exceed 10%, then risk ratios will be calculated from the logistic regression (J. Zhang & Yu, 1998). Hierarchical linear modeling will be used for evaluating effects of individual apartment, neighborhood and regional factors on levels of environmental agents. The main outcomes are allergen, VOC, and pesticide levels in the home; however, several factors should be adjusted in the analysis, including but not limited to smoking in the home, proximity to major roadways, and region of the country. For example, researchers in Baltimore found a low prevalence of both cockroach exposure and sensitization among children in high SES African American families (Sarpong, Hamilton, Eggleston, & Adkinson, 1996). This observation highlights a possible mechanism through which factors operating at the social/environmental

level (e.g., deteriorated built environment) might contribute to asthma among disadvantaged urban children, i.e., via increased exposure to indoor allergens (Rauh et al., 2002). Conceivably, the greenest of homes could still have poor indoor air quality due to some of the aforementioned factors.

The analytical plan for specific hypotheses are:

Hypothesis 1: Green housing will lead to 1) lower levels of environmental contaminants compared with those of comparison housing, and 2) lower levels of related biomarkers in the residents of green vs. comparison housing. (Note: Hypotheses are abbreviated here for brevity. For complete wording of hypotheses see Part A)

The longitudinal study here outlined will permit estimation of:

Geometric mean (GM) and standard deviation (GSD) for each of the environmental analytes (e.g., pesticides, VOCs, mold, and indoor allergens) by rehabilitation type (green vs. comparison).

Geometric mean (GM) and standard deviation (GSD) for each of the biomarkers for pesticides and VOCs by rehabilitation type (green vs. comparison).

Correlations between environmental measurements and biomarkers (stratified by several characteristics including but not limited to age and gender).

Proportion of green vs. comparison homes that have pesticides that are currently banned for residential use by EPA.

*Hypothesis 2:* If irritants and allergens are lower in green vs. comparison housing, children with asthma (ages 7-12) living in green housing should experience fewer and less severe asthma exacerbations. (Note: Hypothesis is abbreviated here for brevity. For complete wording of hypothesis see Part A)

The longitudinal study here outlined will permit estimation of:

Odds ratios (OR) or Rate Ratios (RR) for exposures to environmental agents and cumulative incidence of wheeze and/or other asthma-related morbidity measurements (among children ages 7-12 with asthma).

Missing data: We anticipate the inevitable occurrence of missing data, including dropouts. First, if the missingness of the data is sufficiently small and the associations of interest are sufficiently large, the simple device of imputing upper and lower bound data, if possible, will suffice. That is, a small amount of missing data and a large effect size will allow a unique inference to stand no matter whether the missing data are imputed at their minimum or maximum possible values and used as such. This is consistent with the most conservative approaches adopted in clinical trials wherein subjects lost to follow-up are assumed to have died or to have otherwise suffered the worst possible endpoint. In general however, we must anticipate that we may be facing larger missingness and/or smaller effect sizes and/or impractical upper and lower bounds, such that primary inference changes between the extremes. In this case we will use the multiple imputation procedure of Rubin (Rubin, 1985) to address the problem. In this

technique, a fair amount of effort is devoted to the construction of an imputation model or set of models to provide best estimates of missing endpoints. These best estimates may include the best case or worst case scenarios; the point is that they should most fairly represent data that are missing given the observable information at hand. The imputation models may need to assume data missing at random or they may need further specification to allow for non-ignorable missingness. Each analysis be developed using the best imputation model for missing data for that analysis, using available observed covariates and non-missing endpoints.

### B.3. Methods to Maximize Response Rates and Deal with Nonresponse

Two large-scale housing intervention studies in low-income neighborhoods that had a 1-year follow-up have reported response rates of 92-93% (Morgan et al. 2004; Persky et al. 2009). We anticipate that once enrolled into the Green Housing study, participants will have at least an 80% response rate for completion of the 1-yr study.

We have two strategies to maximize response rates of the enrolled participants: 1) Study participants (mothers/ primary caregivers of children enrolled in study) will receive compensation for their participation as they complete the required study activities throughout the 1-year duration. (see section A.9 INCENTIVES FOR RESPONDENTS for details) and 2) We will also give study results to the participants. Other investigators have found that study participants often wish to know their results (Brody et al. 2007). By offering an in-person discussion of their results during their last home visit, we hope to maximize the chance for completion of their 1-yr follow-up. If we experience a loss-to-follow-up greater than 80%, our contingency plan is to meet with HUD partners to possibly add another study site.

We have the following instructions for trying to contact difficult-to-reach participants: 1) At least 10 attempts will be made and documented in an effort to reach the participant; 2) Calls and visits to the participants will be made at various times of days (mainly between 10am- 8pm) and on different days of the week at a time convenient to the study participant; 3) When leaving a message, the trained technician will leave his/her name, the name of his/her institution, the reason for the call (i.e., housing study, and the call-back number; and 4) The technician will try calling “alternate contacts” to reach the study participants.

### B.4. Tests of Procedures or Methods to be Undertaken

The Green Housing Study questionnaires were primarily based on questions from national health and housing surveys and different epidemiologic studies (e.g., The Inner-City Asthma Study, ICAS) that were conducted in different parts of the country among similar low-income, inner city children with asthma. The national surveys include the following:

1. The National Children’s Study (NCS)
2. The National Health and Nutrition Examination Survey (NHANES)
3. The National Health Interview Survey (NHIS)
4. The Behavioral Risk Factor Surveillance System (BRFSS)
5. The Current Population Survey (CPS)
6. The American Healthy Homes Survey (AHHS)
7. The American Housing Survey (AHS)

Results from the research studies have been extensively published in peer-reviewed environmental health journals that provided scientific basis for home-based asthma intervention studies (Wilson et al. 2009). Some questions from these studies were included verbatim in the Green Housing Study baseline questionnaire, some were modified to fit our study framework, and some additional questions were added (Table 24). CDC epidemiologists modified some of the existing questions and developed new questions in consultation with academic peers and subject matter experts.

**Table 24.** Examples of questions used in the Green Housing Study and their provenance.

| Questions                         | Questionnaire Type   | Question  | Name of the study   | Reference article   |
|-----------------------------------|--|---|---|---|
| Included verbatim                 | Baseline (Home characteristics)  | In the last 3 days: today or yesterday or the day before yesterday, have you either breathed fumes from <b>gasoline</b> or had it on your skin?   | NHANES  | n/a   |
|                                   | Baseline (Child with asthma age 7-12)  | Is [Child's name] currently covered by any kind of health insurance or some other health care plan?   | NCS   | n/a   |
|                                   | Illness checklist  | Did you receive Tamiflu® or oseltamivir [ <i>o sel TAM i veer</i> ] or an inhaled medicine called Relenza® or zanamivir [ <i>za NA mi veer</i> ] to treat this illness?   | BRFSS   | n/a   |
| Included with minor modifications | 6 and 12 month follow-up (Child with asthma age 7-12)<br>* note: the mother or primary caregiver answers this question, not the child. | <u>Green Housing Study version:</u> In the last 3 months, did [Child's name] receive Tamiflu® or oseltamivir [ <i>o sel TAM i veer</i> ] or an inhaled medicine called Relenza® or zanamivir [ <i>za NA mi veer</i> ] to treat this illness?<br><u>BRFSS version:</u> Last month, did you receive Tamiflu® or oseltamivir [ <i>o sel TAM i veer</i> ] or an inhaled medicine called Relenza® or zanamivir [ <i>za NA mi veer</i> ] to treat this illness? | BRFSS<br><br>And also recent H1N1 flu pandemic surveillance | Cauchemez S, Donnelly CA, Reed C, Ghani AC, Fraser C, Kent CK, Finelli L, Ferguson NM. Household transmission of 2009 pandemic influenza A (H1N1) virus in the United States. <i>N Engl J Med.</i> 2009 Dec 31;361(27):2619-27. |

After development of initial draft, the baseline questionnaire was distributed among CDC, NIH, EPA, and HUD colleagues and five non-federal academic peers (Drs. Gary Adamkiewicz, Brett Singer, Mark Mendell, Doug Brugge, and Tiina Reponen) for face and content validation. Based on repeated feedback received from peers, the questionnaire underwent multiple revisions before a final draft was prepared. Cognitive interviews with nine or fewer college-educated CDC colleagues were conducted in a controlled environment. The questionnaire underwent a final revision based on the responses from participants. Some of the results from this pilot testing are shown below.

Each of the questionnaires was pilot-tested at CDC on nine or fewer (in some cases not all 9 were available to participate) predominantly college-educated CDC employee-volunteers during non-work hours. The pilot tests were administered by two Green Housing Study researchers. The results of our pilot testing are shown in Table 25. Based upon pilot testing, the questionnaires were revised to increase ease of understanding and speed of response. We conservatively estimated the response times for our study participants (low-income mothers/primary caregivers living in multifamily, urban housing) based on the average response times recorded during our pilot tests.

**Table 25.** Pilot test of each questionnaire and estimated response time for study participants

| Form name | Average response time (minutes) | Minimum response time (minutes) | Maximum response time (minutes) | Estimated response time for study participants |
|-----------|---------------------------------|---------------------------------|---------------------------------|--|
|           |                                 |                                 |                                 |  |

|  |      |      |      |    |
|--|------|------|------|----|
| Screening questionnaire  | 4:52 | 2:16 | 7:57 | 10 |
| Baseline Questionnaire (Home Characteristics)                            | 6:03 | 4:37 | 7:15 | 15 |
| Baseline Questionnaire (Part 2: Home Characteristics)                    | 2:56 | 2:26 | 3:31 | 5  |
| Baseline Questionnaire (Mother/primary caregiver)                        | 0:58 | 0:50 | 1:15 | 5  |
| Baseline Questionnaire (for Children with asthma 7-12 years)             | 6:38 | 6:20 | 6:50 | 15 |
| 3 and 9-month Phone contact  | 2:30 | 2:15 | 2:45 | 5  |
| 6 and 12-month Follow-up Questionnaire (for environment)                 | 3:52 | 3:10 | 4:20 | 10 |
| 6 and 12-month Follow-up Questionnaire (for children with asthma 7-12)   | 3:07 | 3:00 | 3:15 | 10 |
| Time/Activity form (for Mothers/primary caregivers of enrolled children) | 1:45 | 1:40 | 2:00 | 5  |
| Time/Activity form (for Children with asthma 7-12 yrs)                   | 0:40 | 0:35 | 0:50 | 5  |
| Illness Checklist  | 1:05 | 0:45 | 1:25 | 5  |

B.5. Individuals Consulted on Statistical Aspects and Individuals Collecting and/or Analyzing Data

*Individuals Consulted on Statistical Aspects of the Design*

|                              |                     |                |
|------------------------------|---------------------|----------------|
| Curtis Blanton, MS           | CDC/NCEH            | (770) 488-7114 |
| Dana Flanders, Ph.D.         | CDC/NCEH            | (770) 488-3472 |
| Rey DeCastro, ScD.           | CDC/NCEH            | (770) 488-0162 |
| Carol Gotway Crawford, Ph.D. | CDC/OD              | (404) 498-6023 |
| Andrew Gelman, Ph.D.         | Columbia University | (212) 851-2142 |

*Contractors Responsible for Collecting Information for the Agency*

Contractor Name: TBD

Contractor Address: TBD

*Contractors Responsible for Analyzing Information for the Agency*

Not applicable. CDC will analyze data.



## References

- Acevedo-Garcia, D. and K. A. Lochner (2003). Residential Segregation and Health. *Neighborhoods and Health*. I. Kawachi and L. F. Berkman. New York, NY, Oxford University Press: 265-287.
- Acevedo-Garcia D. 2004. Acculturation. In: Encyclopedia of Health and Behavior (Anderson NB, ed). Thousand Oaks, CA: Sage Publications, 1-6.
- Adgate JL, Church TR, Ryan AD, Ramachandran G, Fredrickson AL, Stock TH, et al. 2004. Outdoor, indoor, and personal exposure to VOCs in children. *Environmental health perspectives* 112(14): 1386-1392.
- Akinbami LJ, Rhodes JC, Lara M. 2005. Racial and ethnic differences in asthma diagnosis among children who wheeze. *Pediatrics* 115(5): 1254-1260.
- Akinbami LJ, Moorman JE, Garbe PL, Sondik EJ. 2009. Status of childhood asthma in the United States, 1980-2007. *Pediatrics* 123 Suppl 3: S131-145.
- Alp H, Yu BH, Grant EN, Rao V, Moy JN. 2001. Cockroach allergy appears early in life in inner-city children with recurrent wheezing. *Ann Allergy Asthma Immunol* 86(1): 51-54.
- Amdur, M. O., J. Doull, et al., Eds. (1991). *Casarett and Doull's Toxicology: The basic science of poisons*. Elmsford, NY, Pergamon Press.
- Arbes SJ, Sever M, Archer J, Long EH, Gore JC, Schal C, et al. 2003. Abatement of cockroach allergen (Bla g 1) in low-income, urban housing: A randomized controlled trial. *J Allergy Clin Immunol* 112(2): 339-345.
- Arshad SH, Tariq SM, Matthews S, Hakim E. 2001. Sensitization to common allergens and its association with allergic disorders at age 4 years: a whole population birth cohort study. *Pediatrics* 108(2): E33.
- Baker SE, Barr DB, Driskell WJ, Beeson MD, Needham LL. 2000. Quantification of selected pesticide metabolites in human urine using isotope dilution high-performance liquid chromatography/tandem mass spectrometry. *Journal of exposure analysis and environmental epidemiology* 10(6 Pt 2): 789-798.
- Barnes PJ. 1995. Is asthma a nervous disease? The Parker B. Francis Lectureship. *Chest* 107(3 Suppl): 119S-125S.
- Belanger K, Beckett W, Triche E, Bracken MB, Holford T, Ren P, et al. 2003. Symptoms of wheeze and persistent cough in the first year of life: associations with indoor allergens, air contaminants, and maternal history of asthma. *American journal of epidemiology* 158(3): 195-202.
- Bornehag CG, Sundell J, Bonini S, Custovic A, Malmberg P, Skerfving S, et al. 2004. Dampness in buildings as a risk factor for health effects, EUROEXPO: a multidisciplinary review of the literature (1998-2000) on dampness and mite exposure in buildings and health effects. *Indoor air* 14(4): 243-257.
- Bradman A, Eskenazi B, Barr DB, Bravo R, Castorina R, Chevri er J, et al. 2005. Organophosphate urinary metabolite levels during pregnancy and after delivery in women living in an agricultural community. *Environmental health perspectives* 113(12): 1802-1807.
- Breyse PN, Buckley TJ, Williams D, Beck CM, Jo SJ, Merriman B, et al. 2005. Indoor exposures to air pollutants and allergens in the homes of asthmatic children in inner-city Baltimore. *Environmental research* 98(2): 167-176.
- Brody, J. G., R. Morello-Frosch, et al. (2007). "Improving disclosure and consent: "is it safe?": new ethics for reporting personal exposures to environmental chemicals." *Am J Public Health* 97(9): 1547-54.
- Brugge, D., J. Vallarino, et al. (2003). "Comparison of multiple environmental factors for asthmatic children in public housing." *Indoor Air* 13(1): 18-27.
- Brugge D, Lee AC, Woodin M, Rioux C. 2007. Native and foreign born as predictors of pediatric asthma in an Asian immigrant population: a cross sectional survey. *Environ Health* 6: 13.
- Brugge, D., M. Woodin, et al. (2008). "Community-level data suggest that asthma prevalence varies between U.S. and foreign-born black subpopulations." *J Asthma* 45(9): 785-9.
- Brunekreef B, Janssen NA, de Hartog J, Harssema H, Knape M, van Vliet P. 1997. Air pollution from truck traffic and lung function in children living near motorways. *Epidemiology (Cambridge, Mass)* 8(3): 298-303.
- Brunekreef B, Dockery DW, Speizer FE, Ware JH, Spengler JD, Ferris BJ. 1989. Home dampness and respiratory morbidity in children. *Am Rev Resp Dis* 140: 1363-1367.
- Buchvald F, Baraldi E, Carraro S, Gaston B, De Jongste J, Pijnenburg MW, et al. 2005. Measurements of exhaled nitric oxide in healthy subjects age 4 to 17 years. *The Journal of allergy and clinical immunology* 115(6): 130-1136.
- Bush RK, Prochnau JJ. 2004. Alternaria-induced asthma. *The Journal of allergy and clinical immunology* 113(2): 227-234.
- Call RS, Smith TF, Morris E, Chapman MD, Platts-Mills TAE. 1992. Risk factors for asthma in inner city children. *J Pediatr* 121: 862-866.



- Cardinale F, de Benedictis FM, Muggeo V, Giordano P, Loffredo MS, Iacoviello G, et al. 2005. Exhaled nitric oxide, total serum IgE and allergic sensitization in childhood asthma and allergic rhinitis. *Pediatr Allergy Immunol* 16(3): 236-242.
- Cauchemez S, Donnelly CA, Reed C, Ghani AC, Fraser C, Kent CK, et al. 2009. Household transmission of 2009 pandemic influenza A (H1N1) virus in the United States. *The New England journal of medicine* 361(27): 2619-2627.
- Chew, G. L., H. B. Burge, et al. (1998). "Limitations of a home characteristics questionnaire as a predictor of indoor allergen levels." *Am. J. Respir. Crit. Care Med.* 157: 1536-1541.
- Chew, G. L., K. M. Higgins, et al. (1999). "Monthly measurements of indoor allergens and the influence of housing type in a northeastern US city." *Allergy* 54(10): 1058-1066.
- Chew, G. L., M. S. Perzanowski, et al. (2003). "Distribution and determinants of mouse allergen exposure in low-income New York City apartments." *Env. Health Perspect.* 111(10): 1348-1351.
- Chew, G. L., E. Carlton, et al. (2006). "Determinants of cockroach and mouse exposure and associations with asthma among families and the elderly living in New York City public housing." *Ann. Allergy Asthma Immunol.* 97(4): 502-513.
- Chew, G. L., M. S. Perzanowski, et al. (2008). "Cockroach allergen levels and associations with cockroach-specific IgE." *J Allergy Clin Immunol* 121(1): 240-5.
- Cho, S. H., T. Reponen, et al. (2006). "The effect of home characteristics on dust antigen concentrations and loads in homes." *Sci Total Environ* 371(1-3): 31-43.
- Clarke CW. Relationship of bacterial and viral infections to exacerbations of asthma. *Thorax.* 1979 Jun;34(3):344-7.
- Cohn, R. D., S. J. Arbes, et al. (2004). "National prevalence and exposure risk for mouse allergen in US households." *J. Allergy Clin. Immunol.* 113(6): 1167-1171.
- Cohn RD, Arbes SJ, Jaramillo R, Reid LH, Zeldin DC. 2006. National prevalence and exposure risk for cockroach allergen in U.S. households. *Environ Health Perspect* 114(4): 522-526.
- Crain EF, Walter M, O'Connor GT, Mitchell H, Gruchalla RS, Kattan M, et al. 2002. Home and allergic characteristics of children with asthma in seven U.S. urban communities and design of an environmental intervention: the Inner-City Asthma Study. *Environ Health Perspect* 110(9): 939-945.
- Custovic A, Simpson BM, Simpson A, Hallam CL, Marolia H, Walsh D, et al. 2003. Current mite, cat, and dog allergen exposure, pet ownership, and sensitization to inhalant allergens in adults. *The Journal of allergy and clinical immunology* 111(2): 402-407.
- Dales, R. and M. Raizenne (2004). "Residential exposure to volatile organic compounds and asthma." *J Asthma* 41(3): 259-70.
- Devos, M., F. Patte, et al., Eds. (1990). *Standardized Human Olfactory Thresholds*. New York, IRL Press at Oxford University Press.
- Diaz-Sanchez D. 1997. The role of diesel exhaust particles and their associated polyaromatic hydrocarbons in the induction of allergic airway disease. *Allergy* 52(38): 52-56.
- Ding YS, Blount BC, Valentin-Blasini L, Applewhite HS, Xia Y, Watson CH, et al. 2009. Simultaneous determination of six mercapturic acid metabolites of volatile organic compounds in human urine. *Chemical research in toxicology* 22(6): 1018-1025.
- Dietz RN, Cote EA. 1982. Air infiltration measurements in a home using a convenient perfluorocarbon tracer technique. *Environ Int* 8: 419-433.
- Diggle, PJ, Liang, KY, Zeger, SL. (1994). *Analysis of Longitudinal Data*. New York. Oxford University Press
- Esposito S, Molteni CG, Daleno C, Valzano A, Tagliabue C, Galeone C, et al. 2010. Collection by trained pediatricians or parents of mid-turbinate nasal flocked swabs for the detection of influenza viruses in childhood. *Virology journal* 7(1): 85.
- Franchi M, Carrer P, Kotzias D, Rameckers EM, Seppanen O, van Bronswijk JE, et al. 2006. Working towards healthy air in dwellings in Europe. *Allergy* 61(7): 864-868.
- Garry VF, Kelly JT, Sprafka JM, Edwards S, Griffith J. 1994. Survey of health and use characterization of pesticide applicators in Minnesota. *Archives of environmental health* 49(5): 337-343.
- Gent JF, Ren P, Belanger K, Triche E, Bracken MB, Holford TR, et al. 2002. Levels of household mold associated with respiratory symptoms in the first year of life in a cohort at risk for asthma. *Environ Health Perspect* 110(12): A781-A786.
- Gold, D. R. and D. Acevedo-Garcia (2005). "Immigration to the United States and acculturation as risk factors for asthma and allergy." *J Allergy Clin Immunol* 116(1): 38-41.

- Gotzsche PC, Johansen HK, Schmidt LM, Burr ML. 2004. House dust mite control measures for asthma. *Cochrane database of systematic reviews (Online)*(4): CD001187.
- Gruchalla RS, Pongracic J, Plaut M, Evans R, Visness CM, Walter M, et al. 2005. Inner City Asthma Study: Relationships among sensitivity, allergen exposure, and asthma morbidity. *J Allergy Clin Immunol* 115(3): 478-485.
- Gunnbjornsdottir MI, Franklin KA, Norback D, Bjornsson E, Gislason D, Lindberg E, et al. 2006. Prevalence and incidence of respiratory symptoms in relation to indoor dampness: the RHINE study. *Thorax* 61(3): 221-225.
- Hankinson JL, Odenchantz JR, Fedan KB. 1999. Spirometric reference values from a sample of the general U.S. population. *American journal of respiratory and critical care medicine* 159(1): 179-187.
- Henderson CE, Ownby DR, Trumble A, DerSimonian R, Kellner LH. 2000. Predicting asthma severity from allergic sensitivity to cockroaches in pregnant inner city women. *The Journal of reproductive medicine* 45(4): 341-344.
- HUD. (2009). "Resident Characteristics Report " Retrieved November 13, 2009, from <http://www.hud.gov/offices/pih/systems/pic/50058/rcr/>.
- Horner WE, Helbling A, Salvaggio JE, Lehrer SB. 1995. Fungal allergens. *Clin Microbiol Rev* 8(2): 161-179.
- Huss K, Adkinson NF, Jr., Eggleston PA, Dawson C, Van Natta ML, Hamilton RG. 2001. House dust mite and cockroach exposure are strong risk factors for positive allergy skin test responses in the Childhood Asthma Management Program. *The Journal of allergy and clinical immunology* 107(1): 48-54.
- Institute of Medicine, Division of Health Promotion and Disease Prevention, Ed. (2000). Clearing the Air: Asthma and Indoor Air Exposures. Washington, D.C., National Academy Press.
- Institute of Medicine (2004). Damp Indoor Spaces and Health. Washington, D.C.: The National Academies Press.
- Jaakkola JJ, Verkasalo PK, Jaakkola N. 2000. Plastic wall materials in the home and respiratory health in young children. *American journal of public health* 90(5): 797-799.
- Jaakkola JJ, Hwang BF, Jaakkola N. 2005. Home dampness and molds, parental atopy, and asthma in childhood: a six-year population-based cohort study. *Environ Health Perspect* 113(3): 357-361.
- Jacobs, D. E., T. Kelly, et al. (2007). "Linking public health, housing, and indoor environmental policy: successes and challenges at local and federal agencies in the United States." *Environ Health Perspect* 115(6): 976-82.
- Jacobson, J. S., R. B. Mellins, et al. (2008). "Asthma, body mass, gender, and Hispanic national origin among 517 preschool children in New York City." *Allergy* 63(1): 87-94.
- Janssen NA, Brunekreef B, van Vliet P, Aarts F, Meliefste K, Harssema H, et al. 2003. The relationship between air pollution from heavy traffic and allergic sensitization, bronchial hyperresponsiveness, and respiratory symptoms in Dutch schoolchildren. *Environmental health perspectives* 111(12): 1512-1518.
- Kass, D., W. McKelvey, et al. (2009). "Effectiveness of an integrated pest management intervention in controlling cockroaches, mice, and allergens in New York City public housing." *Environ Health Perspect* 117(8): 1219-25.
- Kattan, M., H. Mitchell, et al. (1997). "Characteristics of inner-city children with asthma: the National Cooperative Inner-City Asthma Study." *Pediatr. Pulmonol.* 24(4): 253-262.
- Khetsuriani, N., et al., Prevalence of viral respiratory tract infections in children with asthma. *J Allergy Clin Immunol*, 2007. 119(2): p. 314-21.
- Krieger JW, Takaro TK, Song L, Weaver M. 2005. The Seattle-King County Healthy Homes Project: a randomized, controlled trial of a community health worker intervention to decrease exposure to indoor asthma triggers. *Am J Public Health* 95(4): 652-659.
- Kwon HL, Belanger K, Bracken MB. 2003. Asthma prevalence among pregnant and childbearing-aged women in the United States: estimates from national health surveys. *Annals of epidemiology* 13(5): 317-324.
- Lara, M., L. Akinbami, et al. (2006). "Heterogeneity of childhood asthma among Hispanic children: Puerto Rican children bear a disproportionate burden." *Pediatrics* 117(1): 43-53.
- Liao D, Peuquet DJ, Duan Y, Whitsel EA, Dou J, Smith RL, et al. 2006. GIS approaches for the estimation of residential-level ambient PM concentrations. *Environmental health perspectives* 114(9): 1374-1380.
- Matsui, E. C., R. A. Wood, et al. (2003). "Cockroach allergen exposure and sensitization in suburban middle-class children with asthma." *J. Allergy Clin. Immunol.* 112(1): 87-92.
- Matsui, E. C., E. Simons, et al. (2005). "Airborne mouse allergen in the homes of inner-city children with asthma." *J. Allergy Clin Immunol.* 115(2): 358-363.

- Matsui EC, Eggleston PE, Buckley TJ, Krishnan JA, Breyse PN, Rand CS, et al. 2006. Household mouse allergen exposure and asthma morbidity in inner-city preschool children. *Ann Allergy Asthma Immunol* 97(4): 514-520.
- Matt GE, Wahlgren DR, Hovell MF, Zakarian JM, Bernert JT, Meltzer SB, et al. 1999. Measuring environmental tobacco smoke exposure in infants and young children through urine cotinine and memory-based parental reports: empirical findings and discussion. *Tobacco control* 8(3): 282-289.
- Maurya V, Gugnani HC, Sarma PU, Madan T, Shah A. 2005. Sensitization to *Aspergillus* antigens and occurrence of allergic bronchopulmonary aspergillosis in patients with asthma. *Chest* 127(4): 1252-1259.
- Miller EK, Griffin MR, Edwards KM, Weinberg GA, Szilagyi PG, Staat MA, Iwane MK, Zhu Y, Hall CB, Fairbrother G, Seither R, Erdman D, Lu P, Poehling KA; New Vaccine Surveillance Network. Influenza burden for children with asthma. *Pediatrics*. 2008 Jan;121(1):1-8.
- Monto AS. 2002. Epidemiology of viral respiratory infections. *The American journal of the medical sciences* 112(Suppl. 6A): 4S-12S.
- Morgan, W. J., E. F. Crain, et al. (2004). "Results of a home-based environmental intervention among urban children with asthma." *N. Engl. J. Med.* 351(11): 1068-1080.
- Morgenstern V, Zutavern A, Cyrus J, Brockow I, Koletzko S, Kramer U, et al. 2008. Atopic diseases, allergic sensitization, and exposure to traffic-related air pollution in children. *American journal of respiratory and critical care medicine* 177(12): 1331-1337.
- Munir AK, Einarsson R, Dreborg S. 2003. Variability of airborne cat allergen, Fel d1, in a public place. *Indoor air* 13(4): 353-358.
- NHLBI (2007). *Morbidity and Mortality: 2007 Chart Book on Cardiovascular, Lung, and Blood Diseases*, National Institutes of Health (NIH), National Heart, Lung, and Blood Institute.
- NIH. 2005. *Proceedings from the Surgeon General's Workshop on Healthy Indoor Environment*. Bethesda: United States Department of Health and Human Services.
- O'Connor GT, Walter M, Mitchell H, Kattan M, Morgan WJ, Gruchalla RS, et al. 2004. Airborne fungi in the homes of children with asthma in low-income urban communities: The Inner-City Asthma Study. *J Allergy Clin Immunol* 114(3): 599-606.
- Park JH, Cox-Ganser JM, Kreiss K, White SK, Rao CY. 2008. Hydrophilic fungi and ergosterol associated with respiratory illness in a water-damaged building. *Environmental health perspectives* 116(1): 45-50.
- Peat JK, Salome CM, Woolcock AJ. 1990. Longitudinal changes in atopy during a 4-year period: relation to bronchial hyperresponsiveness and respiratory symptoms in a population sample of Australian schoolchildren. *The Journal of allergy and clinical immunology* 85(1 Pt 1): 65-74.
- Perry T, Matsui E, Merriman B, Duong T, Eggleston P. 2003. The prevalence of rat allergen in inner-city homes and its relationship to sensitization and asthma morbidity. *J Allergy Clin Immunol* 112(2): 346-352.
- Persky, V., J. Piorkowski, et al. (2009). "The effect of low-cost modification of the home environment on the development of respiratory symptoms in the first year of life." *Ann Allergy Asthma Immunol* 103(6): 480-7.
- Phipatanakul, W. (2006). "Environmental factors and childhood asthma." *Pediatr Ann* 35(9): 646-56
- Phipatanakul, W., P. A. Eggleston, et al. (2000). "Mouse allergen I: The prevalence of mouse allergen in inner-city homes." *J. Allergy Clin. Immunol.* 106: 1070-1074.
- Phipatanakul W, Eggleston PA, Wright EC, Wood RA, Study TNCI-CA. 2000. Mouse allergen I: The prevalence of mouse allergen in inner-city homes. *J Allergy Clin Immunol* 106: 1070-1074.
- Phipatanakul W, Cronin B, Wood RA, Eggleston PA, Shih MC, Song L, et al. 2004. Effect of environmental intervention on mouse allergen levels in homes of inner-city Boston children with asthma. *Ann Allergy Asthma Immunol* 92(4): 420-425.
- Phipatanakul W. 2002. Rodent allergens. *Curr Allergy Asthma Rep* 2(5): 412-416.
- Pijnenburg MW, Hofhuis W, Hop WC, De Jongste JC. 2005. Exhaled nitric oxide predicts asthma relapse in children with clinical asthma remission. *Thorax* 60(3): 215-218.
- Platts-Mills TA, Vervloet D, Thomas WR, Aalberse RC, Chapman MD. 1997. Indoor allergens and asthma: report of the Third International Workshop. *J Allergy Clin Immunol* 100(6 (part1)): S2-S24.
- Platts-Mills TA, Vaughan JW, Carter MC, Woodfolk JA. 2000. The role of intervention in established allergy: avoidance of indoor allergens in the treatment of chronic allergic disease. *The Journal of allergy and clinical immunology* 106(5): 787-804.
- Quandt SA, Arcury TA, Rao P, Snively BM, Camann DE, Doran AM, et al. 2004. Agricultural and residential pesticides in wipe samples from farmworker family residences in North Carolina and Virginia. *Environmental health perspectives* 112(3): 382-387.

- Rauh, V. A., G. L. Chew, et al. (2002). "Deteriorated housing contributes to high cockroach allergen levels in inner-city households." *Environ Health Perspect* 110(2): 323-327.
- Rosenstreich, D. L., P. Eggleston, et al. (1997). "The role of cockroach allergy and exposure to cockroach allergen in causing morbidity among inner-city children with asthma." *N. Engl. J. Med.* 336: 1356-1363.
- Rubin DB. 1985. Multiple imputation for Non-response in Surveys. New York: John Wiley & Sons.
- Rudel RA, Camann DE, Spengler JD, Korn LR, Brody JG. 2003. Phthalates, alkylphenols, pesticides, polybrominated diphenyl ethers, and other endocrine-disrupting compounds in indoor air and dust. *Environmental science & technology* 37(20): 4543-4553.
- Rumchev, K., J. Spickett, et al. (2004). "Association of domestic exposure to volatile organic compounds with asthma in young children." *Thorax* 59(9): 746-51.
- Salam MT, Li YF, Langholz B, Gilliland FD. 2004. Early-life environmental risk factors for asthma: findings from the Children's Health Study. *Environmental health perspectives* 112(6): 760-765.
- Sarpong, S., R. Hamilton, et al. (1996). "Socioeconomic status and race as risk factors for cockroach allergen exposure and sensitization in children with asthma." *J. Allergy Clin. Immunol.* 97: 1393-1401.
- Savilahti R, Uitti J, Laippala P, Husman T, Roto P. 2000. Respiratory morbidity among children following renovation of a water-damaged school. *Archives of environmental health* 55(6): 405-410.
- Sears, M. R., B. Burrows, et al. (1991). "Relation between airway responsiveness and serum IgE in children with asthma and in apparently normal children." *N. Eng. J. Med.* 325: 1067-1071.
- Senthilselvan A, McDuffie HH, Dosman JA. 1992. Association of asthma with use of pesticides. Results of a cross-sectional survey of farmers. *The American review of respiratory disease* 146(4): 884-887.
- Sever ML, Arbes SJ, Jr., Gore JC, Santangelo RG, Vaughn B, Mitchell H, et al. 2007. Cockroach allergen reduction by cockroach control alone in low-income urban homes: a randomized control trial. *The Journal of allergy and clinical immunology* 120(4): 849-855.
- Skorge TD, Eagan TM, Eide GE, Gulsvik A, Bakke PS. 2005. Indoor exposures and respiratory symptoms in a Norwegian community sample. *Thorax* 60(11): 937-942.
- Sobottka A, Thriene B. 1996. Sanitation programmes for living spaces and health risks involved. *Toxicology letters* 88(1-3): 365-368.
- Sparrow D, O'Connor GT, Basner RC, Rosner B, Weiss ST. 1993. Predictors of the new onset of wheezing among middle-aged and older men. The Normative Aging Study. *The American review of respiratory disease* 147(2): 367-371.
- Spengler JD, Jaakkola JJ, Parise H, Katsnelson BA, Privalova LI, Kosheleva AA. 2004. Housing characteristics and children's respiratory health in the Russian Federation. *American journal of public health* 94(4): 657-662.
- Sporik, R., S. T. Holgate, et al. (1990). "Exposure to house-dust mite allergen (*Der p I*) and the development of asthma in childhood: A prospective study." *N. Engl. J. Med.* 323: 502-507.
- Stark PC, Burge HA, Ryan LM, Milton DK, Gold DR. 2003. Fungal levels in the home and lower respiratory tract illnesses in the first year of life. *American journal of respiratory and critical care medicine* 168(2): 232-237.
- Stark PC, Celedón JC, Chew GL, Ryan LM, Burge HA, Muilenberg ML, et al. 2005. Fungal levels in the home and allergic rhinitis by age five years. *Env Health Perspect* 113: 1405-1409.
- Sunesson AL, Rosen I, Stenberg B, Sjostrom M. 2006. Multivariate evaluation of VOCs in buildings where people with non-specific building-related symptoms perceive health problems and in buildings where they do not. *Indoor air* 16(5): 383-391.
- Sunyer J, Torrent M, Garcia-Esteban R, Ribas-Fito N, Carrizo D, Romieu I, et al. 2006. Early exposure to dichlorodiphenyldichloroethylene, breastfeeding and asthma at age six. *Clin Exp Allergy* 36(10): 1236-1241.
- Takaro TK, Krieger J, Song L, Sharify D, Beaudet N. 2011. The Breathe-Easy Home: the impact of asthma-friendly home construction on clinical outcomes and trigger exposure. *American journal of public health* 101(1): 55-62.
- Thrasher JD, Madison R, Broughton A. 1993. Immunologic abnormalities in humans exposed to chlorpyrifos: preliminary observations. *Archives of environmental health* 48(2): 89-93.
- Tolbert PE, Klein M, Peel JL, Sarnat SE, Sarnat JA. 2007. Multipollutant modeling issues in a study of ambient air quality and emergency department visits in Atlanta. *Journal of exposure science & environmental epidemiology* 17 Suppl 2: S29-35.
- Tolbert PE, Mulholland JA, MacIntosh DL, Xu F, Daniels D, Devine OJ, et al. 2000. Air quality and pediatric emergency room visits for asthma in Atlanta, Georgia, USA. *American journal of epidemiology* 151(8): 798-810.
- Turyk M, Curtis L, Scheff P, Contraras A, Coover L, Hernandez E, et al. 2006. Environmental allergens and asthma morbidity in low-income children. *J Asthma* 43(6): 453-457.

- van Vliet P, Knape M, de Hartog J, Janssen N, Harssema H, Brunekreef B. 1997. Motor vehicle exhaust and chronic respiratory symptoms in children living near freeways. *Environmental research* 74(2): 122-132.
- Voorhorst R, Spijksma FT. 1969. Recent progress in the house dust mite problem. *Acta allergologica* 24(2): 115-123.
- Whyatt RM, Rauh V, Barr DB, Camann DE, Andrews HF, Garfinkel R, et al. 2004. Prenatal insecticide exposures and birth weight and length among an urban minority cohort. *Environmental health perspectives* 112(10): 1125-1132.
- Williams, MK, DB Barr, DE Camann, LA Cruz, EJ Carlton, M Borjas, A Reyes, D Evans, PL Kinney, RD Whitehead, Jr., FP Perera, S Matsoanne, and RM Whyatt (2006). An intervention to reduce residential insecticide exposure during pregnancy among an inner-city cohort. *Env. Health Perspectives*. 114: 1684-1689.
- Wilson J, Dixon SL, Breyse P, Jacobs D, Adamkiewicz G, Chew GL, et al. 2009. Housing and allergens: a pooled analysis of nine US studies. *Environmental research* 110(2): 189-198.
- Zhang, J. and K. F. Yu (1998). "What's the relative risk? A method of correcting the odds ratio in cohort studies of common outcomes." *Jama* 280(19): 1690-1.
- Zota A, Adamkiewicz G, Levy JI, Spengler JD. 2005. Ventilation in public housing: implications for indoor nitrogen dioxide concentrations. *Indoor air* 15(6): 393-401.