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EXPOSURES TO MULTIPLE ENVIRONMENTAL CHEMICALS (LEAD, METHYLMERCURY AND POLYCHLORINATED BIPHENYLS) AMONG CHILDBEARING-AGED WOMEN IN THE U.S.

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EXPOSURES TO MULTIPLE ENVIRONMENTAL CHEMICALS (LEAD, METHYLMERCURY AND POLYCHLORINATED BIPHENYLS) AMONG CHILDBEARING-AGED WOMEN IN THE U.S.

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ABSTRACT

It is estimated that 5 to 20% of neurodevelopmental disabilities in children are caused by environmental toxic exposures. Lead, methylmercury and polychlorinated biphenyls (PCBs) are known to have neurobehavioral and neurodevelopmental consequences in animal models and human population studies. Bioaccumulation and exposures during gestation transfer from mother to fetus via the placenta and to an infant and young child through lactation. Little is known about multiple environmental chemical exposures, especially among childbearing-aged women.

This descriptive and exploratory study involved analysis of existing data from the National Health and Nutrition Examination Survey (NHANES), a national probability sample. Lead, methylmercury and the summed value of four lipid-adjusted PCB congeners (118, 138/158, 153, 180) were measured in the blood or serum of childbearing-aged females aged 16 to 49 of diverse races and ethnicities who were living in the U.S. 1999 to 2004, including a subset of pregnant women. Exposure was defined as two or more xenobiotic blood levels at or above the geometric mean. Sexton, Olden and Johnson's modified environmental health paradigm (1993) guided the selection of 62 measures of vulnerability (susceptibility- and exposure-related attributes, socioeconomic factors and race-ethnicity).

Findings were reported for weighted (adjusted) data. The prevalence of exposures was widespread among childbearing-aged women, one fifth of whom had xenobiotic blood levels at or above the geometric mean for all three chemicals. Overall, pregnant women had lower prevalence rates. Best-fit logistic regression

exposure model contained 13 variables. Three were notable. Any fish consumption in past 30 days tripled the risk. A non-linear relationship was demonstrated with increasing age, exponential at ages 40 to 49. Past and current breastfeeding was protective for these women. Current pregnancy was protective with regard to individual chemical exposures only. Statistically significant two-way interactions were identified even though the paradigm could not be fully tested.

Further research on exposures to multiple environmental chemicals using the modified environmental health paradigm is needed. Xenobiotic biomonitoring in conjunction with risk communication among childbearing-aged women is encouraged. Precautionary level interventions aimed at eliminating or minimizing exposures are urgently needed. Bioaccumulation and transgenerational consequences of exposures should be addressed in public health policy.

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PREFACE

About the Author. Marcella Remer Thompson has a Master of Science degree in Occupational Health from the Harvard School of Public Health, a Master of Science degree in Occupational Health Nursing from Boston University and a Bachelor of Science degree *magna cum laude* from Salve Regina University in nursing. She is board certified as a safety professional and as an occupational health nurse specialist. Thompson is a Fellow of the Academy of American Occupational Health Nurses and a past recipient of the American Society of Safety Engineers (ASSE) Council on Practices and Standards' Safety Professional of the Year. Additionally, Thompson is a former ASSE Vice President of Finance and member of its Board of Directors. Her more than 25 years of work experience includes founding clinical director of a regional Boston hospital's occupational health service, consultant to small- and medium-sized businesses for occupational and environmental health and safety, principal safety engineer for a semiconductor fabrication facility and adjunct faculty for Salve Regina University in Newport, Rhode Island. Currently, Thompson is Assistant Professor, Adjunct at the College of Nursing, University of Rhode Island.

Origins of This Research. In 2004, Thompson became interested in environmental health when she was appointed by (former) Rhode Island Governor Donald E. Carcieri to Chair the Rhode Island Commission for Mercury Reduction and Education. While mercury was the focus of their efforts, it became apparent in discussions at commission meetings that there were broader environmental health issues. Through this experience, she became keenly aware that there were inequities

and gaps in the research of exposures to multiple environmental neurotoxins, particularly among women of childbearing-age. In 2005, while writing protocols for an umbilical cord blood study of lead, mercury and cadmium, there emerged one pivotal question: "Why are we waiting nine months to find out about maternal and fetal exposures to environmental chemicals?"

Future. Thompson's penultimate goals are to make a lasting contribution to the profession through mentoring future generations of occupational and environmental health and safety professionals, conducting environmental health research and improving the public's health by impacting environmental health policy, practice and actively engaging in public dialogue. Her vision for the profession includes a global perspective for managing the built environment, mastery of transdisciplinary knowledge and implementation of the precautionary principle.

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CHAPTER 1

INTRODUCTION

Women of childbearing age should be of great public health concern because their fetuses, infants and young children are vulnerable to the health effects associated with maternal exposures to certain environmental chemicals. Environmental risk factors account for 25 to 33% of the total global burden of disease (Smith, Corvalan, & Kjellstrom, 1999). Seventeen percent of all U.S.-born children are reported to have at least one neurodevelopmental disability (Boyle, Decoufle, & Yeargin-Allsopp, 1994). It is estimated that 5 to 20% of these disabilities are caused by toxic environmental exposures with annual projected costs to diagnose and treat them at $240 billion or 2.8% of all U.S. healthcare expenditures (Landrigan, Schechter, Lipton, Fahs, & Schwartz, 2002). To date, few studies have examined exposures to multiple environmental chemicals among childbearing-aged women. There is a paucity of information about population subgroups who may be disproportionately exposed and/or impacted. Additionally, these exposures may differ between pregnant and non-pregnant women.

Lead, methylmercury and polychlorinated biphenyls (PCBs) were selected for this study because they are pervasive, persistent and co-occur in the environment and each has been shown to have neurobehavioral and neurodevelopmental consequences in animal models and human population studies with these health effects occurring at concentrations below so-called “safe” levels. One would expect that the health effects

from a combination of these chemicals would be more severe than the health effects from exposure to any individual chemical.

Currently, interaction models evaluate chemicals with common health outcomes that is, neurodevelopment and/or single exposure sources such as breast milk. To evaluate the influence of binary interactions on neurotoxicity, the U.S. Agency for Toxic Substances and Disease Registry (ATSDR) examined the scientific literature for the "mechanistic" understanding for each of these chemicals with special attention as to whether these chemicals have the same or similar toxic action.

The ATSDR estimated the direction of toxicological interaction to be greater-than-additive for methylmercury on PCBs and PCBs on methylmercury and additive for lead on methylmercury and methylmercury on lead (Agency for Toxic Substances and Disease Registry, 2004, 2006). However, limitations and inconsistencies with these models may underestimate effects of chemical interactions (Wilkinson et al., 2000). Additionally, the biologically-effective dose from exposures to multiple environmental chemicals may be lower than those associated with exposure for any single environmental chemical. To date, ATSDR has not evaluated interactions for PCBs on lead or lead on PCBs.

As specific environmental chemicals bioaccumulate, the body burden from past exposures has the potential for transgenerational consequences. As a result, childbearing-aged women – not just those who are pregnant – should be of great public health concern. In addition to bioaccumulation, these neurotoxins have adverse health effects if exposure occurs in a sensitive neurodevelopmental period during gestation. Preconceptual, periconceptual and prenatal exposures transfer to fetuses via

the placenta and to infants and young children through lactation. As a result of these transfers, there may be differences in xenobiotic (biomarker for a specific chemical) levels between pregnant or lactating and non-pregnant women.

Exposure to specific environmental chemicals is compounded by vulnerability. It is highly likely that some subgroups of childbearing-aged women have higher exposures than others. It may be possible to identify these at-risk population subgroups by susceptibility- and exposure-related attributes as well as socioeconomic factors and race-ethnicity (Sexton, Olden & Johnson, 1993a; Turner et al., 2003a). Since the health impact of exposures to multiple environmental chemicals may be greater than the impact of exposure to a specific chemical, this impact may be magnified even more among these vulnerable population subgroups. For those who are most vulnerable, a safe exposure level may be zero.

Despite what is known about the hazards of exposure to these specific environmental chemicals, little is known about exposures to combinations of these chemicals. To date, few studies have examined exposures to combinations of environmental chemicals known to have neurological and neurodevelopmental consequences among women of childbearing age.

Conceptual Framework

Exposure has been defined as "the contact between an agent and a target with contact taking place at an exposure surface over an exposure period by an exposure route" (International Programme on Chemical Safety, 2000, p. 21). Exposure is strongly related to the concepts of environment, human and health – all phenomena of interest to nursing science (Fawcett & Malinski, 1996). Exposure and health are

related to vulnerability. Vulnerability is defined most broadly as a "susceptibility to harm" (Turner et al., 2003a). Sexton, Olden and Johnson (1993a) referred to four categories of vulnerability: susceptibility-related attributes, exposure-related attributes, socioeconomic factors and race-ethnicity. It is generally thought that the more fragile, less resilient and/or less resourceful, the more vulnerable (Aday, 2001; Kasperson, 2001; Kasperson, Kasperson, Turner, Dow, & Meyer, 1995; Sexton, 1997).

In nursing, the focus is on the (human) client (Kim, 2000). As a result, exposure is measured by the presence of biomarkers in blood, tissue and body excretions. The presence of a xenobiotic (biomarker of specific chemical) or its metabolites within a compartment of an organism confirms exposure to that specific environmental substance. A biomarker of exposure reflects the relationship between external contaminant (amount available for contact from all potential sources) and body burden (internal dose). The internal dose or bioavailability of an agent is dependent upon the distribution, bioaccumulation, storage and elimination capabilities and capacities of the human body (National Research Council, 2006). Xenobiotic levels are chemical-specific biomarkers of exposure that estimate body burden most closely.

The modified environmental health paradigm (Sexton et al., 1993a, p. 714) was the overarching theoretical frame of reference and deliberative construct for this research as it described the complex relationship between the physical and biological effects of environmental hazards and vulnerability. This dissertation sought to define and explore these interrelationships.

Aim

The aim of this research was to examine childbearing-aged and pregnant childbearing-aged women's exposures to specific environmental chemicals known to have neurobehavioral and neurodevelopmental consequences in animal models and human population studies. This dissertation focused on exposures to each of these chemicals individually and in four different combinations and permutations. Additionally, this dissertation identified those population subgroups at highest risk for two or more xenobiotic (chemical-specific) blood levels at or above the geometric mean. This research used existing data from the National Health and Nutrition Examination Survey (NHANES), a national probability sample.

Research Questions. This study had three research questions:

1. What was the prevalence of childbearing-aged and pregnant childbearing-aged women's exposures to each of the following environmental chemicals: lead, methylmercury and polychlorinated biphenyls (PCBs) as measured by chemical-specific (xenobiotic) levels at or above geometric mean in blood or serum of these women who were living in the United States from 1999 through 2004?

2. What combinations and permutations of chemical exposures were most common among these childbearing-aged and pregnant childbearing-aged women as evidenced by xenobiotic blood levels at or above the geometric mean?

3. What, if any, subsets of childbearing-aged women were disproportionately exposed to two or more of these environmental chemicals based on susceptibility-related attributes (reproductive status, age, health and nutritional status), exposure-related attributes related to acculturation, proximity (residential characteristics and

occupation), activity (diet and tap water supply) and behavior (alcohol consumption and tobacco use); socioeconomic factors (education, employment, income and marital status) and race-ethnicity?

Research Design

These research questions were addressed through secondary data analysis of existing data from the National Health and Nutrition Examination Survey (NHANES), 1999 through 2004. NHANES is a population-based survey from the Centers for Disease Control and Prevention (CDC) National Center for Health Statistics (NCHS). Data from this survey are publicly available online at http://www.cdc.gov/nchs/nhanes.htm. NHANES provides a probability sample of baseline information on the health and nutritional status of the non-military, non-institutionalized adults and children living in the United States. As part of this survey, biomonitoring data was collected for more than 116 environmental chemicals or their metabolites including all the chemicals of interest to this study (Centers for Disease Control and Prevention, National Center for Environmental Health, 2007).

Significance of the Study

It is hoped that the findings of this study will help remove critical barriers to progress in areas of environmental health, public health and nursing.

By evaluating complex and important issues regarding exposures to lead, methylmercury and polychlorinated biphenyls among childbearing-aged women, environmental health research can continue with more robust study designs such as longitudinal, prospective cohort and case-control studies.

The design of NHANES allowed for this study to provide nationally representative estimates of exposures; these estimates will be useful in future public health planning. Every decade, CDC publishes its objectives for promoting health, preventing disease and eliminating health disparities in the United States. This research is relevant to CDC's *Healthy People 2020* objective EH HP2020-21: "Reduce exposure to selected environmental chemicals in the population as measured by blood and urine concentrations of the substances or their metabolites" (Centers for Disease Control and Prevention, 2009d).

This research will help provide a clearer understanding of exposure, a newly-identified concept for nursing within the client domain. By identifying at-risk groups, precautionary-level (preconceptual and prenatal) interventions can be designed and implemented. The findings of this study will support risk communication among childbearing-aged women with regard to their multiple chemical exposures and the transgenerational consequences of these exposures. *Is it safe? Is it safe enough*?

Outline of Chapters to Follow

In Chapter Two, Literature Review, definitions of exposure and related concepts are provided. Exposure models from five disciplines are reviewed. The contextual development of Sexton, Olden, and Johnson's modified environmental health paradigm is outlined and its major constructs delineated (1993a). *In vivo* and *in vitro* mechanistic interaction studies of binary chemical combinations are described. Human studies evaluating health outcomes of childbearing-aged (non-pregnant) women's exposures to these chemicals are summarized.

In Chapter Three, Methodology, the aims and research questions are reiterated. The choice of research design is discussed followed by a description of the data source that includes a brief summary of its origins. Three potential concerns involving the use of these existing data are addressed. A description of the dataset and study population are provided. Measurements of all dependent and independent variables are described; their validity and reliability reviewed. Data processing and analytic procedures are detailed. Ethical protocols used in the original research and this dissertation are outlined.

Chapter Four, Findings, begins with a general description of the study population. This chapter addresses all data gathered with regard to each research question followed by a discussion of the results. Comparisons between the exposure model and each individual chemical model are drawn and discussed.

Chapter Five, Summary, Conclusions, Limitations and Implications, summarizes the study, draws conclusions and outlines the study's limitations before outlining implications for theory development, research, education, practice and policy.

CHAPTER 2

REVIEW OF LITERATURE

In this chapter, concepts are defined. Exposure models from five disciplines are summarized. The contextual development of Sexton, Olden, and Johnson's the modified environmental health paradigm is outlined and its major constructs are delineated (1993a). In vivo and in vitro mechanistic interaction studies of three binary chemical combinations are described as are human studies that have evaluated health outcomes of exposures to all three chemicals of interest.

The Concept of Exposure

In the Oxford dictionary, exposure is defined as "an action, a state, value or condition; the action of subjecting / the state or fact of being subjected to any external influence; a definite quantity or amount of something (as in dose); an unprotected or undefended condition." To assess whether nursing had an explicit definition of exposure, a literature search was conducted using ProQuest Dissertations and Theses titles and abstracts (1997-2008) with keywords (exposure and environmental health and nursing) and CINAHL (2004-2009) with keywords (nursing and exposure and environmental health). No explicit definition or measurement of exposure was identified in nursing. It was not listed as a keyword or indexed in texts for the nursing specialties of public health, occupational health, community health or environmental health. While this concept has not been defined explicitly, it is a term that is used frequently in the nursing literature and characterized in many different ways including

causing disease, impacting a condition and adversely affecting health (Rogers, 1994b). It has been typified as a pathway or route (Lipscomb & Sattler, 2001; Institute of Medicine, 1995) and has served as an integral part of the nursing process in exposure assessment or exposure history (Sattler, Afzal, McPhaul, & Mood, 2006; Sattler, McPhaul, Afzal, & Mood, 2004). It has been given attributes of location such as occupational or residential exposure (King & Harber, 1998), hazard category for example, chemical/physical/biological exposure (Rogers & Cox, 1998), specific agent such as pesticide or lead exposure (Grady, Harden, Moritz, & Amende, 1997; Larsson & Butterfield, 2002), time as in short-term and/or long-term exposure or acute and/or chronic exposure (Edmondson & Williamson, 1998; McPhaul & Lipscomb, 2005) and a relative degree of severity as in potential or excessive toxic exposure (Sattler & Lipscomb, 2003; Tiedje & Wood, 1995).

Existing definitions and measurements of exposure were reviewed from five disciplines central to environmental health: occupational (industrial) hygiene, exposure science, toxicology, medicine and epidemiology. The disciplines of occupational (industrial) hygiene and exposure science are utilized when assessing risk (Sattler & Lipscomb, 2003). Toxicology and epidemiology are disciplines considered essential to environmental health nursing (Institute of Medicine, 1995). The review included literature identified through CINAHL, PUBMED and Sociological Abstracts as well as textbooks, dictionaries and handbooks central to these disciplines. This unpublished concept analysis (Thompson, 2006) concluded that, historically, the concept of exposure has been explicitly or implicitly defined and measured in each of these disciplines in accordance with how each discipline

approaches the etiology and amelioration of environmentally-related disease that is, source, person, outcome or some combination.

It became clear from this analysis that interdisciplinary discordance required the use of a transdisciplinary definition of exposure. A broader internet search yielded a comprehensive criteria document on human exposure assessment which was published by a transdisciplinary group of international experts from the International Programme on Chemical Safety (IPCS) under the auspices of the World Health Organization, the United Nations Environment Programme and the International Labour Organization (International Programme on Chemical Safety, 2000). They defined exposure as "the contact between an agent and a target with contact taking place at an exposure surface over an exposure period by an exposure route" (International Programme on Chemical Safety, 2000, p. 21). This is the definition of exposure that is used in this dissertation.

Exposure-Related Concepts

This concept analysis found exposure to be strongly related to the concepts of environment, human and health – all phenomena of interest to nursing science (Fawcett & Malinski, 1996). These concepts, their interrelationships with exposure and their relative importance to nursing have fluctuated over time.

Florence Nightingale believed that the environment was the fundamental cause of suffering and disease; literally, disease came "out of the air" (Nightingale, 1860). Such emphasis on the environment fell out of favor with the advent of germ theory when biological agents – not the environment itself – were identified as the cause of disease (Henle, 1840). The "patient" became "host" to these biological agents. In the twentieth century, when the interrelationship of host-agent-environment was described

as an equilibrium state, disease was no longer "a reparative process" (Nightingale, 1860, p. 7) but a state of disequilibrium. The concept of environment became inconsequential, merely "an entity in which host and agent find themselves" (Gordon, 1949, p. 507). "Health" was defined as the absence of disease. For decades, nursing constrained the definition of environment to the personal environment that is, people, places and objects that surrounded the person (Randall, Tedrow, & Van Landingham, 1984) with almost exclusive attention to the hospital or home environment and the caring of the sick. When the environment was viewed as the "source of stimuli to which individuals respond" (Chopoorian, 1986, p. 40), nursing focused on adapting the patient (as a response) to his/her environment. "Health" and "disease" were viewed more broadly on a continuous scale of well-being (Linder, 1958, p. 1276). Over time, the concept of environment encompassed socioeconomic, political and cultural aspects and institutional elements. Health, disease and well-being were considered to be biological expressions of social relations such as poverty and health disparities (Kreiger, 2001). Despite the well-publicized environmental disasters of Love Canal, Bhopal and Chernobyl, an ecological perspective of the environment was not found in community and public health nursing literature until the mid-1990s (Neufer, 1994; Tiedje & Wood, 1995).

Because a broader conceptualization of environment that encompasses global ecological perspectives has been slow to emerge in nursing, transdisciplinary definitions of environment, human and health were sought. These and other exposure-related concepts (agent, dose and vulnerability) were incorporated into one conceptual construct (Figure 1). Their definitions are provided below.

Environment. Surprisingly, "environment" was not explicitly defined in the International Programme on Chemical Safety (IPCS) criteria document on exposure assessment (2000). Kim (2000, p. 166) defined environment as "a separate entity that exists external to a person or to humanity, conceived … as that containing many distinct elements" that is, spatial, temporal and qualitative (socio-cultural). This definition of environment is congruent with IPCS definitions of target (a biological entity), agent (specific hazard) and exposure (as contact) because it allows for spatial differentiation of agent from target and therefore exposure with regard to exposure surface and exposure period. Therefore, Kim's definition of environment is used in this dissertation.

Human. Exposure is assumed to be characteristic and a process of human nature and living; by definition, an essentialistic concept in the client domain (Kim, 2000). Within this domain, there is a traditional focus on the individual as the unit of analysis. Regardless of whether the person is a single individual or an aggregate of individuals, using the target population or some segment of it as the origin of research data is an acknowledgment that the unit of analysis is at the individual level (Khrisanopulo, 1963).

Health. The authors of the IPCS criteria document (2000) did not define health explicitly. However, adverse biological effect was defined as "a change in morphology, physiology, growth, development and/or life span resulting in impairment of functional capacity to compensate for additional stress or increase in susceptibility to the harmful effects of environmental influences" (International Programme on Chemical Safety, 2000, p. 27). The phrase "to compensate for

additional stress" infers a conceptual definition of health as an outcome of successful compensation and/or adaptation to stress or stressors in the environment and, conversely, disease as an expression of failure at compensation and/or adaptation. Therefore, health was not defined in terms of a health-or-disease dichotomy but as "a continuous scale of well-being" (Linder, 1958, p. 1276). Therefore, Linder's definition of health is used in this dissertation.

Agent. "A chemical, biological or physical entity that contacts a target" (Zartarian, Ott, & Duan, 2007, p. 58) a/k/a "a threat comprised of perturbations and stress /stressors and the consequences they produce" (Turner et al., 2003a, p. 8074). An agent is referred to as a hazard if the agent is capable of causing harm. There are five general types of hazards: chemical, physical, mechanical, biological and psychosocial (Appendix B: Hazard Categories). Chemical agents in the environment are ubiquitous. There are 90 known elements and an infinite number of inorganic and organic compounds found in nature (Blumer, 1975; Turner, 2002). Some naturally-occurring chemicals and chemical compounds have been reproduced and/or modified by humans (Silbergeld, 1995). Some hazardous environmental chemicals such as lead and mercury exist naturally in elemental, inorganic and organic (e.g., alkyl lead and methylmercury) forms. However, their proportional contributions to total environmental concentrations are insignificant when compared to their anthropogenic sources (Lindberg et al., 2007). Other chemicals like polychlorinated biphenyls (PCBs) have been synthesized.

Dose. Exposure is aligned closely with the concept of dose. While exposure involves contact between an agent and a target, dose is the amount of agent that enters

a target by crossing an exposure surface or absorption barrier (International Programme on Chemical Safety, 2000). "While there can be no dose without a corresponding exposure, there can be exposure without a corresponding dose" (Zatarian, Ott & Duan, 2007, p. 45). This distinction is of paramount importance when measuring exposure and extrapolating dose. Dose equals exposure only when one assumes total absorption of the agent by the target (National Research Council, 1991). The internal dose or bioavailability of an agent is dependent upon the target's distribution, bioaccumulation, storage capacity and elimination capability (International Programme on Chemical Safety, 2000).

Vulnerability. Exposure and health are related to vulnerability. Vulnerability is defined most broadly as a "susceptibility to harm" (Turner et al., 2003a, p. 8074) with reference to physical, psychological and/or social health of individuals and/or populations (de Chesnay, 2005). The U.S. Environmental Protection Agency (2003b, p.39) defined vulnerability as "the intrinsic propensity of an exposed entity to experience adverse effects from external agents, events, perturbations or stresses." While vulnerability is variable over time at the individual level, it is more stable at the population level (Burbank, 2006). Kasperson (2001), Lee (2005), and the U.S. Environmental Protection Agency (EPA) National Environmental Justice Advisory Council (2004) have referred to four broad overlapping categories of vulnerability: susceptibility/sensitivity, differential exposure, differential preparedness and differential ability to recover. Although the term susceptibility had been used synonymously with vulnerability by the International Programme on Chemical Safety (2000), susceptibility remained a subcategory of vulnerability in this dissertation.

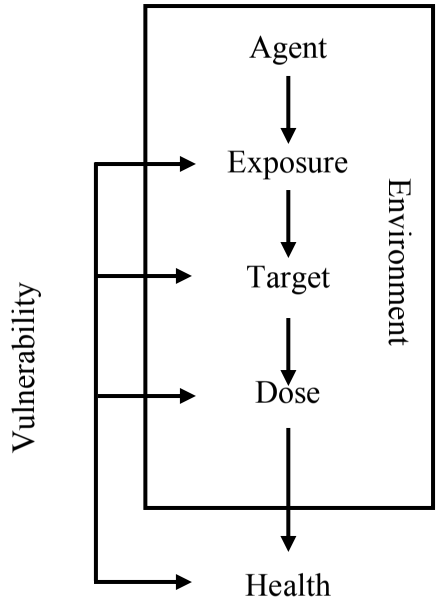


Figure 1. Exposure and Related Concepts. Adapted from *The Concept of Exposure,* by M. R. Thompson, 2006, unpublished manuscript, College of Nursing, University of Rhode Island, Kingston, RI.

Susceptibility-Related Attributes. Susceptibility or sensitivity is defined as the combination of intrinsic and acquired attributes of an individual, group of individuals or subpopulation that modify the biological response to exposure. Intrinsic attributes include genetic predisposition, gender, age and developmental stage while acquired attributes include reproductive status, health status, nutritional status and psychosocial stress or allostatic load (Grassman, 1996; Sexton, 1997). These attributes are systematically different from exposure-related attributes that increase the likelihood of exposure to environmental contaminants (Lee, 2005; Sexton, 1997).

Exposure-Related Attributes. Exposure-related attributes are acquired through differences in proximity, activity and behavior. Specifically, these attributes encompass proximity to environmental contamination sources such as residential characteristics, occupation, non-occupational activities, drinking water supply, diet and consumption of tobacco, drugs and/or alcohol (Lee, 2005; Sexton, 1997).

Socioeconomic Factors. Socioeconomic factors affect the ability to be prepared and/or to recover. Factors such as education, employment and income influence health indirectly through complex interactions with susceptibility- or exposure-related attributes or both (Sexton et al., 1993a). These interactions involve inequalities in access to adequate healthcare, nutrition, safe and healthy housing and personal, family and community resources (Flaskerud & Winslow, 1998; Mechanic & Tanner, 2007; Nyamathi, Koniak-Griffen, & Greengold, 2007).

Race-Ethnicity. The most controversial attribute of vulnerability is that of race-ethnicity. In the U.S., rates of morbidity and mortality vary significantly among racial and ethnic groups (Perlin, Wong, & Sexton, 2001; Sexton, Kleffman, & Callahan,

1995b) and the cause of these health disparities are not well elucidated (Sexton et al., 1993b). To some extent, racial and ethnic identity represents genetic and phenotypic homogeneity within a common geography and/or culture (Molnar, 1998). Sykes (2001) and others have analyzed mitochondrial DNA sequences and traced *Homo sapiens*' 150,000-year family tree to just 33 clans worldwide. While decoding the human genome and discovering epigenetic mechanisms will elucidate genetic commonalities and phenotypic distinctions (Vineis, Khan, Vlaanderen, & Vermeulen, 2009), race and ethnicity are social and not biological constructs. As such, these "bioethnic conscriptions" may act as indirect surrogates for socioeconomic disadvantage (Montgomery & Carter-Pokras, 1993; Montoya, 2007), serve as proxy variables for residential segregation and social isolation (Acevedo-Garcia & Osypuk, 2008) and/or reflect institutional environmental discrimination (Gelobter, 1992; Lee, 1992). All of these factors could influence susceptibility, exposure and health. However, even when these factors are controlled for confounding, racial and ethnic differences have persisted (Lieu, Newacheck, & McManus, 1993). Whatever the causal determinants, racial and ethnic minorities remain vulnerable and therefore, race-ethnicity was included in this dissertation.

The definition of vulnerability is congruent with the concept of health as an outcome of an adaptive process; the more fragile, less resilient and/or less resourceful, the less adaptive and consequentially, the more vulnerable (Aday, 2001; Institute of Medicine, 1995; Kasperson, 2001; Kasperson, Kasperson, Turner, Dow, & Meyer, 1995; Sexton, 1997). It is crucially important to identify those vulnerable individuals

and/or groups of individuals who are at-risk for adverse health effects as a result of exposures to multiple environmental chemicals.

Definitions for the major concepts discussed in this chapter and other exposure-related concepts can be found in Appendix C. Conceptual Definitions.

Review of Exposure Models by Discipline

Once the major concepts were defined, a review was conducted of existing exposure models in five disciplines central to environmental health: occupational (industrial) hygiene, exposure science, toxicology, medicine and epidemiology. The results of this search are summarized here.

Occupational (Industrial) Hygiene Model. The ecological model of occupational (industrial) hygiene (Cralley & Cralley, 1985) illustrates the inseparability of environment and health and describes this interrelationship as an ecological balance maintained through co-adaptation. The major assumption of this model is that man and environment are indivisible and each reacts upon the other as moving through and changing each other simultaneously (Clayton, 1973). There are three sources of environmental stressors capable of impacting health: macrocosmic stressors, microcosmic stressors and those individual stressors associated with lifestyle, work and off-the-job (Cralley & Cralley, 1985). The goal of occupational (industrial) hygiene is the protection of health through the recognition, evaluation and control of that which is both measureable and controllable in the environment (Clayton, 1973; Irish, 1973). Its central concept is surveillance with an emphasis on environmental monitoring. As a result, this model was not appropriate for this dissertation.

Exposure Science Models. Ott's (1985) full risk model is illustrated by five links from source to effect. Each link is dependent upon those links that precede it; each link is equally important in assessing overall risk.

The source-to-dose model (Lioy, 1990) begins at the point where a chemical enters the environment and tracks its movement to exposure or target contact; individual characteristics are inconsequential. Since environmental regulations seek to control specific sources of contamination, this type of model addresses each source separately. The source-to-dose model calculates exposure and potential dose for an individual within a population of interest. According to Price and Chaisson (2005), this source-to-dose model does not address aggregate exposures (total dose from a single substance received from multiple sources), cumulative exposures (total doses from multiple substances received from multiple sources), or intra- or inter-individual variation. To account for uncertainty, the model intentionally overestimates the average exposure.

The person-oriented exposure model (Price & Chaisson, 2005) places the concept of "person" at the center of the design with the focus on the population of interest rather than the sources of exposure. Price and Chaisson based their framework on a series of four nested loops which they referred to as the exposure event loop, the time-step loop, the inter-individual variation loop and the uncertainty loop. Assumptions of the model include: a chemical dose from each source is constant for a specified (short) duration of time; a chemical level in the microenvironment is constant; and person-characteristics such as physiology, demographics, housing, activities, and microenvironment are constant for a specified (short) duration of time. Distribution

sampling among the population of interest determines the person-characteristics for a specified (short) duration of time. Since the probability of exposure to each chemical source is dependent upon these person-characteristics, there appears to be a seemingly unlimited opportunity to introduce any number of human-related variables. The model allows for individuation when exposures are different, even if the chemical is the same. Exposures to multiple chemicals can be concurrent, successive or mutually exclusive. Route-specific and source-specific doses for each chemical and for each person are calculated, thus providing a more accurate population profile for a specific duration at a specific point in time. Between the exposure and inter-individual loops, the time-step loop provides insight into how a person's exposures vary over time with changes in characteristics, microenvironment and source. As a result, this model is more dynamic than the source-to-dose model. Because it is person-centered, exposure is more broadly conceptualized as a characteristically-driven process with many dimensions.

The goal of exposure science is to characterize quantitatively the relationships among all identified sources and exposure contacts with a specific target or representative population (Ott, 2007). One model is predominantly observational and performed in the field within normal living and working situations or microenvironments. The other is to construct exposure profiles mathematically (Ott, 1985). These aforementioned models of exposure were created for generalization to populations, particularly those at high risk from environmental contaminants. However, these models generate large amounts of data which could lead to "paralysis

by analysis" so there are some doubts as to their empirical application. As a result, these exposure science models were not selected for this dissertation.

Toxicological Models. There were two toxicological models examined: toxicokinetic and toxicodynamic. Toxicokinetic models trace the physiology involved with transport, metabolism and disposition of an agent internally. "What does the target do?" Toxicodynamic models describe the influence of agents on the target. "What does the agent do?" (Rozman, Doull, & Hayes, 2001).

Toxicology is the study of the adverse effects of chemicals on living organisms. It is a mechanistically-oriented discipline that identifies cellular, biochemical, and molecular mechanisms by which chemicals exert specific effects on living organisms. These mechanisms are identified through laboratory experiments and observations as well as mathematical modeling (Klaassen & Watkins, 2003). The goal of toxicology is to define dose-response, the correlative relationship between exposure and effect. Toxicology does not consider the source of exposure or the environment in which the agent exists and the target lives. Thus, these models had limited application to this dissertation.

Spectrum of Disease Model. The spectrum of disease model (Leavell & Clark, 1958) describes the prepathogenesis and pathogenesis of disease that occurs over time. The spectrum begins with the host's exposure to the etiological agent (prepathogenesis) and concludes with symptom development (pathogenesis). The spectrum encompasses subclinical manifestations of the host's response to the agent. The period of pathogenesis begins with the development of overt symptoms of illness

through the diagnosis of disease. It concludes with death, disability or recovery (Hussey, 2002).

With this model, emphasis is on diagnosis and treatment of symptoms and disease. Diagnosis involves categorization of findings from the health history, physical examination and laboratory evaluation into broad classes or so-called "toxic syndromes." Diagnosis initiates treatment for presenting symptoms based upon the most likely category of toxin responsible for those symptoms (Klaassen & Watkins, 2003.) There are many factors that confound the process of making an accurate and/or early diagnosis. Most environmentally-related illnesses either manifest as nonspecific symptoms or mimic other common illnesses in clinical presentation. Often, subclinical manifestations go unnoticed. Documenting a patient's environmental health history is rarely a routine component of primary care. It is employed only when there is already a suspicion of environmental etiology. By the time an accurate diagnosis is made, irreversible harm may have already occurred (Paranzino, Butterfield, Nastoff, & Ranger, 2005).

By emphasizing diagnosis and treatment of symptoms and disease, the spectrum of disease model is incongruent with environmental health nursing practice which focuses on the "prevention of illness and injury and protection from work-related and environmental hazards" (Association of American Occupational Health Nurses, 2008). As a result, it was not appropriate for this dissertation.

Epidemiological Models. Single causation models, multiple causation models and multi-dimensional causation models were considered.

Single Causation Models. According to miasma theory, the universal source of morbidity and mortality was the "foul emanations" from the environment (soil, water and air). A human contracted disease directly from the environment. To reduce disease, one had to control the environment (Lancisi, 1717). Germ theory identified a distinct and single *contagium animatum* responsible for each disease. To reduce disease, one had to control the infectious agent (Henle, 1840 as cited in Rosen, 1936). Gordon's (1949) epidemiological model represented interactions among host, agent and environment. To control disease, one had to maintain equilibrium among the host, agent and environment. These models have applicability to infectious and mechanical agents only. Thus, a single causation model was not appropriate for this dissertation.

Multi-Causation Models. Three multi-causation models were considered. MacMahon and Pugh (1970) broadened the single etiological model to explain how more diverse aspects of host-agent-environment were involved in disease causation. This "web of causation" model represented complex interrelationships among risk factors and disease. Cassel's (1976) psychosocial theory proposed that the host's neuroendocrine reaction to environmental stressors specifically in the social environment causes an increase in susceptibility to disease. Detrimental aspects of the social environment included dominance hierarchies, social disorganization, rapid social change, marginal status in society and bereavement. McEwen (1998) introduced the concept of "allostatic load" in which psychosocial factors were not merely contributing to an increased susceptibility to disease as Cassel had proposed, but that these reactions were directly pathogenic to the host. A combination of biological evolution, behavior and experience influences the host's perception of stress

and subsequently causes neuroendocrine stress which results in adverse health over time. These multiple causation models introduced the concept of individual susceptibility on a biological level. Inherent in this biological focus was the assumption that individual perception and behavior changes were sufficient to attain and retain health. However, there is substantial uncertainty about the relative contribution of allostatic load (Sexton et al., 1993a). These models were too limited in scope for this dissertation.

Multi-Dimensional Systems Causation Models. Three multi-dimensional systems causation models were examined. Causation models with multiple levels of interactive and dynamic systems shifted the focus from the individual to the "social production of disease" and the "political economy of health" (Krieger, 2001, p. 670). Vulnerability was viewed from a systems perspective. Krieger's (1994) ecosocial theory visualized fractals intertwined with inseparable levels of health, disease and well-being as biological expressions of social relations. "Social structure, cultural norms, ecologic milieu and genetic variability must be systematically addressed" (Krieger, 1994, p. 897). Similarly, the theory of eco-epidemiology focuses on systems analyses, specifically, pathogenesis and causality at the molecular level and causal pathways at the societal level (Susser & Susser, 1996b). In the socio-ecologic model (McMichael, 1999) interpersonal and intrapersonal aspects were illuminated to include physical, social, cultural and institutional elements such as organizational culture. These multi-dimensional systems causation models introduced the concept of sociopolitical and economically-related vulnerability. Inherent in this social focus was

the assumption that social changes on the population level were sufficient to attain and retain health. These models were too broad in scope for this dissertation.

Historically, epidemiology is defined as the study of the distribution and determinants of disease frequency and its goal is to determine the etiology of disease (MacMahon & Pugh, 1970). As such, these models were more useful in effects assessment – not exposure assessment – which was the focus of this dissertation.

Environmental Health Nursing. Salazar and Primomo's (1994) ecological systems model for environmental health nursing practice was adapted from Bronfenbrenner's (1979) ecology of human development theory. According to Bronfenbrenner's theory, an individual's environment was comprised of four concentric sets of structures (macro-, exo-, meso-, micro-systems) that reflect relative proximity or conversely, distance to/from the individual. These systems interacted through progressive mutual accommodation to shape an individual's temperament, personality, belief system and behavior (Johnson, 2002). Salazar and Primomo selected this theory because the construct of multi-dimensional systems was useful for describing the complexities of physical, cultural, social, political and economic factors that contribute to environmental hazards for application in environmental health nursing practice (Salazar & Primomo, 1994). However, their adaptation of Bronfenbrenner's theory was exploratory and, therefore, it was of limited use for this dissertation.

Conclusions. As a result of this multidisciplinary exposure models review, it was concluded that each of these models was structured in accordance with how each discipline approached the etiology and amelioration of environmentally-related

disease. These findings were consistent with findings of the concept analysis described earlier. Some of these models were congruent with conceptual definitions; none of these models addressed all key concepts. As a result of this analysis, it became clear that a single unifying conceptual framework for this dissertationn was needed – one that would address both exposure *and* vulnerability. A reference found in the IPCS criteria document on human exposure assessment (International Programme on Chemical Safety, 2000, p. 25) led to the modified environmental health paradigm (Sexton et al., 1993a).

Modified Environmental Health Paradigm

The conceptual framework selected for this dissertation was the modified environmental health paradigm by Sexton, Olden and Johnson (1993a, p. 714).

Figure 2. Modified Environmental Health Paradigm

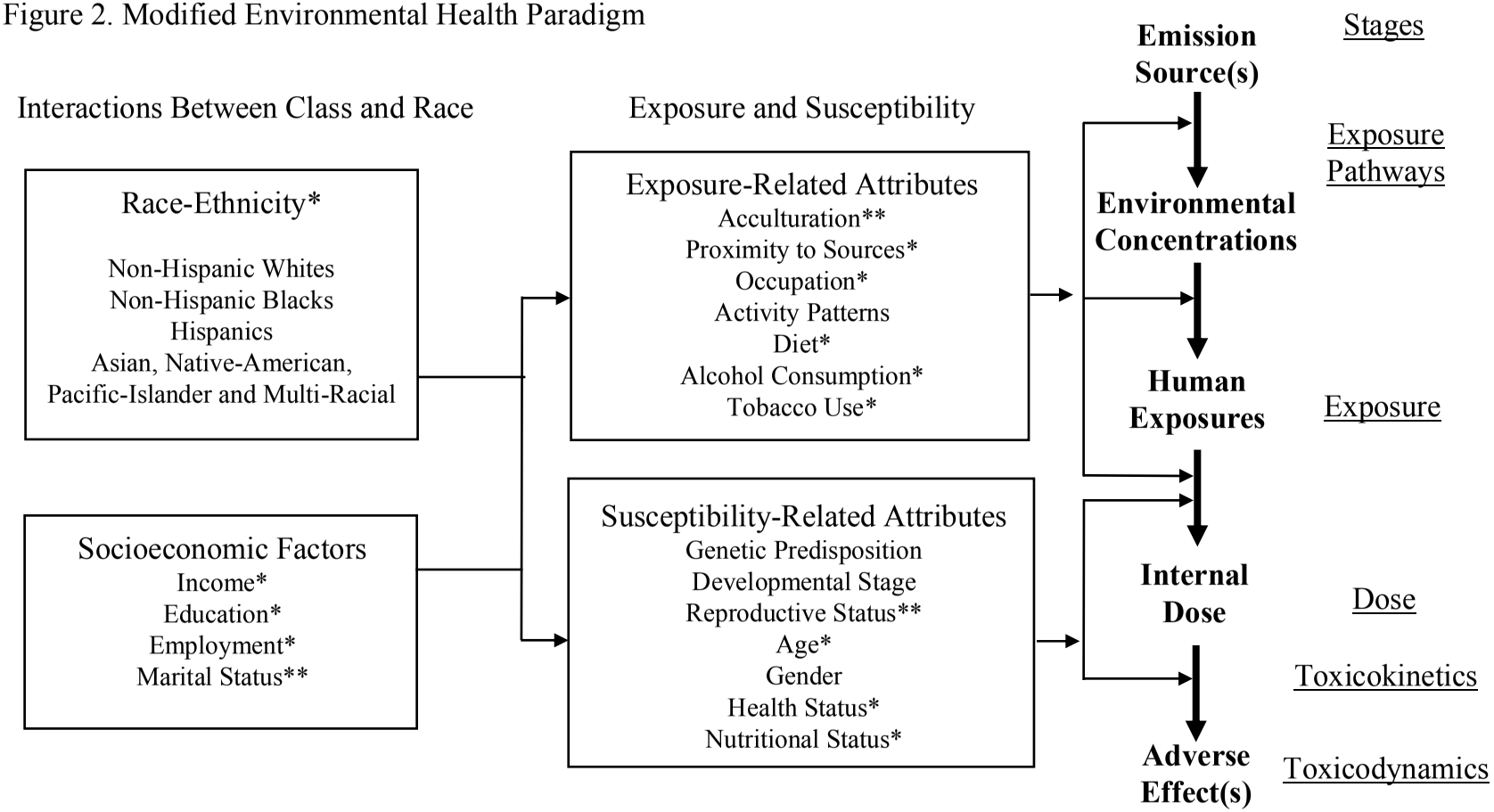


Figure 2. Modified Environmental Paradigm. bold = Original Environmental Paradigm; * = original variables; ** = variables added. Adapted from "Environmental Justice": The Central Role of Research in Establishing a Credible Scientific Foundation for Informed Decision Making, by K. Sexton, K. Olden, & B. Johnson, 1993a, *Toxicology and Industrial Health, 9*(5), p. 714. Copyright 1993 by SAGE Publications, London, UK.

Selection Criteria. Selection of this paradigm was based upon the following criteria:

1) Congruence with environmental health nursing practice which "focuses on promotion and restoration of health, prevention of illness and injury and protection from work-related and environmental hazards" (Association of American Occupational Health Nurses, 2008);

2) Focus and level of analysis is on the individual human being (Kim, 2000);

3) Addresses the essentialistic characteristics and processes of human nature and living (Kim, 2000);

4) Inclusion seven major concepts: environment, agent, exposure, target (human), dose, vulnerability and health;

5) Congruence with definition of exposure as a characteristic and process of human nature and living (Kim, 2000);

6) Conceptualization of a dynamic interactive process between/among major concepts; and

7) Incorporation of conceptual definitions consistent with all other criteria.

The following section provides historical and political contexts of its development and outlines its purpose and goals, focus, scope and basic assumptions. It includes a brief review of published research studies and critical analyses that test the theory's concepts and operational constructs.

Development of Paradigm. The historical and political context in which this framework was developed provides much insight into its philosophical foundations and construction. At the time of its original publication in 1993, Ken Sexton was

director of the U.S. Environmental Protection Agency's Office of Health Research. Barry L. Johnson was administrator of the Agency for Toxic Substances and Disease Registry and assistant surgeon general. Kenneth Olden was the director of the National Institute of Environmental Health Sciences and the National Toxicology Program, the principal federal agency responsible for assessing the toxicity of environmental substances. A decade earlier, *Risk Assessment in the Federal Government: Managing the Process* (National Research Council, 1983) had been adopted by these and other U.S. federal agencies in an effort to unify their evaluative methodologies in conducting research, assessing risks and making risk management decisions (Williams, 1995). This risk assessment process was comprised of four elements: hazard identification, exposure assessment, dose-response assessment and risk characterization (National Research Council, 1983). Health policy formulation was based upon a risk assessment's strength of evidence and the benefits and costs of different command-and-control strategies (Johnson, 2007). By employing this quantitative methodology, policy decisions were assumed to be based on "impartial" and rational choice (Bartell, 2005). Intrinsic assumptions of this process included the existence of an acceptable level of risk, the existence of a "safe" level that was possible to determine empirically and an overarching belief in the ability of biological entities to recover, if not immediately, then in the future. These policies resulted from a U.S. Supreme Court decision (Industrial Union Department v. American Petroleum Institute, 448 U.S. 607, 1980) that nullified efforts by the Occuptional Safety and Health Administration to promulgate regulations aimed at reducing occupational exposures to benzene as far as technologically possible. With this court decision,

cognizant agencies had the burden to prove harm "beyond a reasonable doubt" (Cranor, 2004). As a result, agencies' research efforts concentrated upon understanding the specific mechanisms of exposure, the determinants of health, and the links between exposure and health (Sexton & Reiter, 1989). Satisfying this fairly stringent standard of proof required detailed, science-intensive risk assessments which often combined multiple studies from the five disciplines central to environmental health.

The original environmental health paradigm (Sexton et al., 1993a, pp. 706, 719; Sexton, Callahan & Bryan, 1995a, p. 18) was conceptualized simply to represent the continuum of exposure between hazard source and health outcome and to serve as a unifying, transdisciplinary model for risk assessment (K. Sexton, personal communication, September 22, 2009). Believing that "exposure, not toxicity, is the ultimate means by which we regulate use or release of hazardous agents" (Graham et al., 1992, p. 409), risk assessment identified and evaluated adverse outcomes that could occur in well-defined scenarios resulting in narrowly-constructed hypotheses that included only well-defined variables. Unfortunately, the less defined the event or outcome, the more uncertainty existed. This epistemological uncertainty created default model assumptions which produced overly conservative risk estimates. Most often, an "uncertainty factor" or "margin of safety" ranging from 10 to 1,000 times was "calculated" into the risk assessment at the last step. Risk assessment remained a quantitative framework based on probability theory and empirical causation. It evaluated and combined evidence from various scientifically-based disciplines to determine an acceptable level of risk with which there was an associated willingness-

to-pay value (Bartell, 2005). Cost-benefit analyses of economically-related indicators provided the sole basis for determining a willingness-to-pay value associated with that risk. Another type of cost-benefit analysis used was the "quality-adjusted-life-years" calculation which compared positive outcomes with negative outcomes associated with a comparison of relative risks in terms of life expectancy. Under this original risk-based environmental health paradigm, the health policy formulation process preserved the status quo until there was sufficient certainty of evidence present or until sufficient uncertainty was removed – "innocent until proven guilty" (Cranor, 2004). To a great extent, the promulgation process remained a quagmire. The demand for strong empirical justification led to regulatory "paralysis by analysis" and a regulatory process that responded to existing health problems only when a high certainty of severe (irreversible) harm existed. If risks were small, they were considered insignificant and therefore acceptable. The goal of risk assessment was to realize the greatest good by balancing the interests of all affected persons. Risk assessment was viewed as an objective assessment of everyone's interests and a tool to guide an impartial choice to maximize good for all affected parties (Beauchamp & Childress, 2001). However, there was no independent weighting of values in the process of regulating environmental risk and that resulted unintentionally in unjust social distribution.

By 1993, there was evidence of inequitable distribution of the costs and benefits associated with environmental regulations among vulnerable communities; specifically, placement of hazardous waste sites, landfills, incinerators and polluting industries in communities inhabited mainly by racial and ethnic minorities and low

income groups (Bullard, 1990; Johnson, Harris, & Williams, 1992; United Church of Christ Commission for Social Justice, 1987; U.S. Government Accounting Office, 1983; U.S. Environmental Protection Agency, 1992a).

In 1983, the U.S. General Accounting Office (GAO) assessed the correlation between the location of hazardous waste landfills in southern states and the racial and economic status of their surrounding communities. Using 1980 U.S. census data, the GAO found African Americans were the majority population in three out of four communities and their mean family income was below that of all races within the same community. Five years later, the United Church of Christ Commission for Social Justice (1987) conducted two cross-sectional studies to determine the extent to which racial and ethnic minorities were exposed to commercial hazardous waste transfer, storage and disposal facilities (TSDFs) and uncontrolled toxic waste sites in their communities. Using racial, ethnic and income classifications from the 1980 U.S. census, their analysis indicated race was a stronger predictor of the location of TSDFs than income, education and all other socioeconomic indicators. *Dumping in Dixie: Race, Class and Environmental Quality* (Bullard, 1990) chronicled the concerns and potential health risks of those residing near hazardous waste landfills and industrial chemical facilities. This seminal work galvanized the environmental equity movement in the same way Sinclair's *The Jungle* (1906) gave impetus to food safety 84 years earlier.

In response, the Centers for Disease Control and Prevention and the Agency for Toxic Substances and Disease Registry sponsored a national minority health conference which focused on:

1. demographics (i.e., special problems in determining exposure of minority populations to hazardous substances in the environment);
2. health perspectives (i.e., factors such as nutritional status, lifestyle, and socioeconomic influences that may cause exposure to hazardous substances to affect minority populations disproportionately); and
3. health communication and health education (i.e., the effectiveness of public health messages for minority populations about preventing exposures to hazardous substances).

(Centers for Disease Control and Prevention, 1990, p. 825)

Barry L. Johnson was co-editor for the conference proceedings (Johnson, Harris, & Williams, 1992). Similarly, the EPA formed an Environmental Equity Working Group to review the impact of hazardous environmental substances on minority and low income populations. Ken Sexton was a member of this working group. *Environmental Equity: Reducing Risk For All Communities* concluded that "there are clear differences between racial groups in terms of disease and death rates" (U.S. Environmental Protection Agency, 1992a, p. 3). One recommendation was to "provide an objective basis for assessment of risks by income and race, beginning with the development of a research and data collection plan" (U.S. Environmental Protection Agency, 1992a, p. 4). This inclusion of social, economic and behavioral factors in risk assessment heralded a shift to the concept of "total risk" among populations (U.S. Environmental Protection Agency, 2003b, p. 2). It is within these contexts that Sexton, Olden and Johnson developed the modified environmental health paradigm.

Stated Purpose and Goals. "We outline a risk-based framework for analyzing issues of environmental justice … to improve our understanding of fundamental mechanisms of environmentally-related disease … and to underscore the critical

importance of identifying and evaluating groups potentially at greater risk … ” (Sexton et al., 1993a, p. 687).

Focus and Scope. The modified environmental health paradigm described the interrelationships among socioeconomic class and race, exposure- and susceptibility-related attributes and environmental health risk (Sexton et al., 1993a, p. 713.) This paradigm allowed exploration of the interrelationships among these components. Demonstrated interrelationships among socioeconomic class, race, exposure- and susceptibility-related attributes indicated whether certain demographic groups were disproportionately represented in at-risk categories. This dissertation sought to define and explore some of these interrelationships.

Theoretical and Philosophical Foundations. As stated previously, the original environmental health paradigm (Sexton et al., 1993a, pp. 706, 719; Sexton et al., 1995a, p. 18) was conceptualized simply to represent the continuum of exposure from hazard source to health outcome (“exposure-disease continuum”) and to serve as a unifying, transdisciplinary model for risk assessment (K. Sexton, personal communication, September 22, 2009). Its theoretical and philosophical foundations were post-positivistic, deduced from quantitative-based research among disciplines central to environmental health. Of note, the International Programme on Chemical Safety and other organizations selected this continuum to serve as the foundation for the domain of exposure assessment (Gee & Payne-Sturges, 2004; International Programme on Chemical Safety, 2000, p. 25; Weis et al., 2005). In an effort to explain health disparities, Sexton, Olden and Johnson modified this paradigm in 1993.

These modifications were made after careful consideration of conceptual models by Freeman, Wagener, Williams and Wilson; and Polednak.

Freeman (1989, 1991) postulated that socioeconomic factors accounted for racial differences observed in cancer incidence, mortality and survival. He cited numerous studies as evidence. Regardless of race, poor Americans had a higher incidence of cancer and lower five-year survival rates. Freeman argued that "race is a gross variable for culture, tradition, belief systems and lifestyle" and "poverty acts through this cultural prism" (Freeman, 1989, p. 329). Similar to race, poverty was a proxy variable for specific elements of living such as inadequate physical and social environments (substandard housing, social isolation); inadequate information and knowledge (lack of education); risk-promoting behaviors (smoking, alcohol consumption, substance abuse and inadequate nutrition); and inaccessible or inadequate healthcare. In his model, all of these factors contributed to decreased cancer survival.

Wagener, Williams and Wilson's (1993) model emphasized broader psychosocial contexts of environmental risks. In this model, race was a composite measure of biological, cultural, socioeconomic and sociopolitical factors as well as racial discrimination. Racial discrimination was not elaborated further. They postulated that these factors were interrelated and mitigated health status through intermediary variables such as medical care (need, access and quality); psychosocial resources (social ties, perceptions of control and coping patterns); environmental stress (residential and occupational); psychosocial stress (family, financial and residential);

and health practices (smoking, alcohol and nutrition). In turn, these intermediary variables affected health through one or more unidentified biological processes.

Polednak's (1989) model of acculturation described the interrelationships among determinants of health status and acculturation. Polednak defined acculturation as "a reciprocal process that encompasses those phenomena which result when groups of individuals having different cultures come into continuous first-hand contact with subsequent changes in the original culture patterns of either or both groups" (Polednak, 1989, p. 26). The acculturation process has four possible outcomes: assimilation (total acceptance of new culture and total rejection of the original culture); integration (partial retention of original culture with partial incorporation of the new culture); reaffirmation (total rejection of new culture and total retention of original culture); or marginalization (rejection of both original and new cultures) (Page, 2006; Maxwell, 2009). Depending upon which aspects of culture are accepted, retained or rejected, the impact of acculturation on health outcome varies (Gibson, Diaz, Mainous, & Geesey, 2005; Grant, Hamer, & Steptoe, 2009; Negy, Schwartz & Reig-Ferrer, 2009; Polednak, 1989; Weis & Bellinger, 2006).

Polednak substantiated his conceptual framework by citing many multidisciplinary studies comparing disease rates and patterns of developing countries with those countries already developed (Polednak, 1989). These studies demonstrated that improvements in sanitation, nutrition, control of infectious diseases and access to medical care have led to reductions in infant mortality and an increased average life expectancy. Conversely, increases in hypertension, diabetes mellitus, cardiovascular disease and certain cancers were the result of a combination of negative factors:

environmental (increased pollution, noise and population density); psychosocial stress (language barriers, decreased social interaction and low self-perception of health); and health practices (smoking, alcohol use, poor dietary habits, risky sexual behavior and decreased physical activity levels). This is referred to as the "immigrant paradox" where immigrant health is better upon arrival in the U.S. then declines inversely to time spent in the United States (Gallo, Penedo, Espinosa de los Monteros, & Arguelles, 2009; Lee, Nguyen, & Tsui, 2009; Markides & Coreil, 1986; Mendoza, 2009). "Both diversity and similarity across populations need to be recognized, whether one is dealing with sociocultural characteristics, biological characteristics or risk of disease" (Polednak, 1989, p. 295).

These three theories added insight into the differences in health status among racial, ethnic and socioeconomic groups and assisted Sexton, Olden and Johnson in developing their framework. However, citing "substantial uncertainty about the relative contribution of this factor" (p. 702), they chose not to address acculturation as a separate entity in their model. Despite this uncertainty, it was decided to include measures of acculturation in this dissertation as it was considered a contributing factor to exposure-related activities and behavior choices.

Basic Assumptions. Sexton, Olden and Johnson were not explicit about the assumptions for their model. Based upon readings, this author has deduced the following assumptions about the modified environmental health paradigm:

1. Human existence cannot be considered out of an environmental context (Kim, 2000, p. 167);

2. For an exposure to occur, an agent and a target must be in contact with each other, both spatially and temporally (Zartarian et al., 2007, p. 34);

3. Exposure is an integral and necessary component in a sequence of events having potential health consequences (World Health Organization, 1990, p. 23);

4. Without exposure, there can be no dose (Zartarian et al., 2007, p. 34);

5. The concentration of each agent generated from each source and the resulting dose are constant for a specific period of time (Zartarian et al., 1997);

6. Vulnerability increases the risk of adverse health effects from a given exposure (Sexton et al., 1993a); and

7. Vulnerability impacts compensation and recovery from these adverse health effects (Sexton, 1992b).

Tests for Validity and Reliability. The original environmental health paradigm by Sexton, Olden and Johnson is widely accepted. The International Programme on Chemical Safety and the Environmental Protection Agency selected the original environmental health paradigm to serve as the foundation for exposure assessment and human health environmental exposure research (International Programme on Chemical Safety, 2000, p. 25; U.S. Environmental Protection Agency, 2003c). It has been used by various investigators and organizations to conceptualize their research directions and strategies (K. Sexton, personal communication, October 13, 2009). Twelve articles referencing Sexton, Olden and Johnson's paradigm were identified using the keywords (environmental health paradigm and Sexton or Sexton) in CINAHL, PUBMED, Sociological Abstracts and the ProQuest Dissertations and Theses database (1993-2009). These publications were reviewed. Only one by

Murray (2003) specifically evoked the modified environmental health paradigm. To date, no known tests for validity and reliability have been performed on this paradigm.

Relational Propositions. The original environmental health paradigm consists of five stages: exposure pathways, exposure, dose, toxicokinetics, toxicodynamics. Vulnerability was added in the modified version. These relationships are delineated here within the context of this study's three chemicals of interest: lead, methylmercury and PCBs and a review of the scientific literature.

Stage One: Exposure Pathways. There are three possible exposure pathways that an agent takes from its source to the target:

1. directly from the sources via one or more environment media;
2. indirectly after undergoing transformation by biotic or abiotic means; and
3. accumulating in the environment (Lioy, 1990).

When quantifying exposure pathways, data are collected non-invasively through chemical inventories, environmental monitoring and personal monitoring as an agent's concentration in a particular medium. Measurement of specific agents in a target's environment establishes whether and to what extent the individual is potentially exposed to such agents. Fate and transport models identify and evaluate exposures most accurately when specific agents, sources and concentrations of agents, and exposure pathways are known a priori such as in occupational settings (Price & Chaisson, 2005).

Lead, methylmercury and PCBs are pervasive, persistent and co-occur in the environment. Because of the hemispheric distribution of global emissions, these chemicals have been detected at elevated levels in all remote areas of the globe. For

example, lead has been found in Icelandic salt marshes (Marshall, Clough, & Gehrels, 2009; Shotyk & LeRoux, 2005), mercury in the tundra ecosystem (Poissant, Zhang, Canario, & Constant, 2008), and PCBs in Arctic and Antarctic air (Choi et al., 2008). Even if it were possible to cease all new emissions of these chemicals, their biogeochemical cycles would extend for years to decades or longer (U.S. Environmental Protection Agency, 2008a). Their environmental persistence is affected by air and sea temperatures, wind speeds, variation in precipitation patterns (Lindberg et al., 2007) and secondary effects of climate change (McMichael & Martins, 2002) such as soil acidification (Navratil, Skrivan, Vach, Dobesova, & Langrova, 2004). Irrespective of source, it is generally accepted that co-occurrence of environmental chemicals in general and these chemicals in particular exist due to their common spatial and temporal distributions (Altenburger, 2008; Agency for Toxic Substance and Disease Registry, 2004, 2006). As a result of this pervasiveness, persistence and co-occurrence, humans have daily contact with these three environmental chemicals. They are present at or above detectable levels:

1. in air, water, soil/rock and food (Clayton, Pellizzari, Whitmore, Perritt, & Quackenboss, 1999; Kawahara, Horikoshi, Yamaguchi, Kumagai, & Yanagisawa, 2005; Sunderland, 2007; Macintosh, Kabiru, Echols, & Ryan, 2001; Mahaffey, Clickner, & Bodurow, 2004; Roy, Georgopoulos, Ouyang, Freeman, & Lioy, 2003; Schecter et al., 2001);

2. where people live, work, play and learn (Herrick, McClean, Meeker, Baxter, & Weymouth, 2004; Herrick, Meeker, Hauser, Altshul, & Weymouth, 2007;

Krieger, Bernard, Dinoff, Ross, & Williams, 2001; Lauwerys & Hoet, 1993; Lawson et al., 2006; Rudel, Seryak, & Brody, 2008; Van Hemmen et al., 2001);

3. in consumer products purchased and equipment used (MacGregor, 2004; McRill, Boyer, Flood, & Ortega, 2000; Sällsten, Thorén, Barregård, Schütz, & Skarping, 1996; Weldon et al., 2000); and,

4. in some instances, these chemicals are incorporated into social behaviors and ritual practices (JSI Center for Environmental Health Studies, 2003; Riley, Newby, Leal-Almeraz, & Thomas, 2001; U.S. Environmental Protection Agency, Office of Emergency and Remedial Response, 2002).

Stage Two: Exposure. There may be multiple and/or sustained contacts with one or more agents. An exposure assessment estimates the exposure concentration for an individual. Such an assessment assumes that the concentration of agent generated from each source is constant for a specified duration of time (Zartarian et al., 1997). Since a target's specific activities affect exposure, measures of contact can be based on activity patterns from diaries, questionnaires and direct observation (International Programme on Chemical Safety, 2000). While adding a time-step loop may provide insight into intra-individual variation, it does not address inter-individual variation adequately. A large population sample is required to compensate for these variations (Price & Chaisson, 2005). This dissertation used a large population sample.

Stage Three: Dose. The amount of agent that enters a target in a specified time duration after crossing an exposure surface and/or absorption barrier is a function of the exposure concentration (International Programme on Chemical Safety, 2000). The rate and extent to which an agent can be absorbed by a target is dependent upon the

agent-in-medium's molecular weight and chemical properties specifically, pH, degree of ionization, water or lipid solubility and the target's physiology. Lead, methylmercury and PCBs are lipophilic and unbound to plasma protein and, as such, either simply diffuse across or are transported through an absorption barrier by specialized carrier systems (Dix, 2001). Absorption of an agent can be impacted by pre-systemic ("first-pass") extraction whereby some or all of it is eliminated quickly (Eaton, 2005).

Stage Four: Toxicokinetics. Once the agent is absorbed, it is subject to a myriad of toxicokinetic processes involving distribution, bioaccumulation and elimination (International Programme on Chemical Safety, 2001a). Distribution of the agent among anatomical or physiologic compartments via systemic circulation that is, blood and/or lymph, may or may not result in different concentrations of xenobiotics in various tissues and/or organs over time. Distribution is dependent upon volume and clearance, the agent's affinity for the medium and the target's elimination efficiency. Toxicokinetic mechanisms that may be affected include enzyme and active transport induction, competitive inhibition, modification of uptake and elimination (Sexton & Hattis, 2007). In a single-compartment model, the xenobiotic equilibrates quickly in all body tissues that is, xenobiotic concentrations and subsequent changes in concentrations are proportional throughout the body even though the concentrations may not be identical numerically. The elimination rate is linear and affected by dose and the limits of the target's capacity to respond (Medinsky & Valentine, 2003). In a two-compartment model, there are central compartments (blood/plasma/lymph) and peripheral compartments (tissues). Distribution is rapid but concentrations are not

proportional between compartments. Elimination rate is slower and independent of dose (Caraccio & Mofenson, 1993).

Exposure, dose and these toxicokinetic processes determine the body burden. Repeated exposures to an agent result in cumulative storage and a corresponding increase in body burden until a steady state is achieved (Dix, 2001, p. 569). An agent with a long biological half-life bioaccumulates with each successive dose, reaches steady-state concentration slowly and continues to be excreted slowly even after exposure has ceased (Medinsky & Valentine, 2003). Plasma proteins, fat tissue, bone and organ systems that are responsible for elimination (liver, kidney) store these xenobiotics. The agent is released very slowly from these storage sites as it undergoes biotransformation and excretion (Rozman & Klaassen, 2003).

A biomarker of exposure reflects the relationship between external contaminant (i.e., amount available for contact from all potential sources) and body burden (i.e., internal dose). Biomarkers of exposure do not provide information on timing, sources or routes of exposure. While timing and duration of exposure are more critical for chemicals with shorter half-lives, it is less critical for those with longer half-lives as is the case with lead, methylmercury and PCBs. For agents that produce developmental defects at low dosages or concentrations, biomarkers may be the only available indicators of exposure (National Research Council, 2000a). Xenobiotic levels are chemical-specific biomarkers of exposure that estimate body burden most closely. It is for these reasons that xenobiotic levels in blood were used to assess exposures in this dissertation. Exposures to multiple environmental chemicals could produce any one of four toxicokinetic interactions: independence, antagonism, additivity or

synergism (Appendix C: Conceptual Definitions). Chemical interaction models are addressed further in this chapter.

Stage Five: Toxicodynamics. At the molecular level, an agent biochemically alters the target's cell regulation and/or cell maintenance. If and when the degree and rate of the target's compensatory biochemical mechanisms are absent, insufficient and/or overwhelmed, then cellular dysfunction, dysregulation and/or destruction at the tissue, organ and/or organism level results (International Programme on Chemical Safety, 2001b). A target's response can be measured in biomarkers of effect and detected as subclinical and/or clinical manifestations of morbidity and mortality.

Exposure to any one of these three chemicals has been shown to have neurobehavioral and/or neurodevelopmental consequences in animal models and human population studies (Collaborative on Health and the Environment's Learning and Development Disabilities Initiative, 2008a, 2008b). These effects are well documented and have been reviewed elsewhere (Costa, Aschner, Vitalone, Syversen, & Soldin, 2004; Faroon, Jones, & deRosa, 2000; Wigle et al., 2007).

For each dose response, there should be a corresponding conceptual biological plausibility that may or may not be understood fully. The correlative relationship between dose and a defined response can be graphically depicted as *s*-curved or biphasic (Calabrese & Baldwin, 2003). A biphasic or hormetic response may reflect data variability (Thayer, Melnick, Burns, Davis, & Huff 2005) or the presence of two or more different biochemical mechanisms with parallel or non-parallel overlapping dosages (Rozman, Doull, & Hayes, 2001). The slope of a dose response relationship may or may not be constant. An inconsistent slope may reflect two or more different

biochemical mechanisms as well. Most importantly, a low or no-dose threshold may reflect a target's vulnerabilities more than an agent's toxicity. Several epidemiologic studies have indicated that health effects occur at concentrations below "safe" levels (Grandjean, Budtz-Jørgensen, Kieding, & Weihe, 2004a; Lanphear, Dietrich, Auinger & Cox, 2000) with a cumulative impact on health (Sexton & Hattis, 2007). The degree to which a target is likely to experience "harmful effects of environmental influences" results from the intersection of an agent's toxicity and the target's vulnerability (International Programme on Chemical Safety, 2000, p. 27).

Vulnerability. There are four components to vulnerability: susceptibility-and exposure-related attributes, socioeconomic class and race-ethnicity. Vulnerability can influence the magnitude of biological response to environmentally-related exposures, the type of response or both (Grassman, 1996; Sexton, 1997). The health effects of vulnerability may be cumulative (Nyamathi, Koniak-Griffin, & Greengold, 2007; Shi & Stevens, 2005; Shi, Stevens, Lebrun, Faed, & Tsai, 2008).

Susceptibility-Related Attributes. These attributes are comprised of intrinsic and acquired attributes that modify the response to exposure. Intrinsic attributes are physiologic in nature and include genetic predisposition, developmental stage, age and gender. Acquired attributes include health status and nutritional status (Grassman, 1996; Sexton, 1997; Sexton et al., 1993b).

Genetic Predisposition. Certain interactions among genes, proteins and metabolites may modify biologic response to environmental exposure (Cascorbi, 2006; Neri et al., 2006). Human epidemiological and animal studies have demonstrated associations between genetic variations, phenotypic expressions and

disease etiologies (Cummings & Kavlok, 2004). Current understanding of the gene-environment interactions involved in toxicokinetics are rudimentary (Gundacker, Gencik, & Hengstschläger, 2010). Few population studies include biomarkers of susceptibility. As a result, this type of intrinsic susceptibility was not included in this research.

Developmental Stage. Fifty percent of all females living in the United States are of childbearing-age (U.S. Census Bureau, 2000). Since these specific environmental chemicals bioaccumulate, the body burden from past exposures has the potential for transgenerational consequences. In addition to bioaccumulation, these neurotoxins have adverse health effects if exposure occurs in a sensitive neurodevelopmental period during gestation. During pregnancy, environmental chemicals are easily transferred from maternal blood through the placenta to the fetus by simple diffusion since the placenta is a permeable plasma membrane (Goyer, 1990). Since the blood-cerebrospinal fluid-brain barrier does not mature until the infant is six months old, plasma proteins easily transfer through this "barrier" to the developing brain (Adinolfi, 1985). There exists structural and functional windows of vulnerability during which environmental exposures have the potential to alter neurodevelopment and neurobehavior permanently (Wilson, 1973). On a cellular level, crucial stages of neurodevelopment include: neuronal and glial proliferation, neuronal and glial differentiation, cellular migration, neurite outgrowth of axonal and dendrite processes, synaptogenesis (formation of neurotransmitters and receptors), myelination and apoptosis or programmed cell death (Radio, Freudenrich, Robinette, Crofton, & Mundy, 2010; Suñol, 2010). All of these stages occur with precision timing during

the prenatal period with connectivity and synaptic reorganization occurring into adolescence (Connors et al., 2008). Xenobiotic disruption of neurodevelopment may occur at one or more of these morphological and/or functional maturational stages. Further detail regarding these specific mechanisms is beyond the scope of this dissertation.

Reproductive Status. Preconceptual, periconceptual and prenatal exposures to these chemicals transfer to fetuses via the placenta and umbilical cord, and to infants and young children through lactation (Axelrad, Bellinger, Ryan, & Woodruff, 2007; Bellinger, Leviton, Waternaux, Needleman, & Rabinowitz, 1987; Daniels et al., 2003; Dewailly et al., 1996; Gundacker et al., 2002; Pilsner et al., 2009;Vreugdenhil, Van Zanten, Brocaar, Mulder, & Weisglas-Kuperus, 2004). As a result of these transfers, there may be differences in xenobiotic blood levels between pregnant and non-pregnant women.

Age. Susceptibility is age-dependent. An infant's rapid respiratory rate and higher skin permeability increases the amount of agent inhaled or absorbed while those agents ingested through lactation diffuse freely through gastrointestinal mucosa into blood circulation to target organs (Weiss & Bellinger, 2006). The endocrine, reproductive, immune, visual and nervous systems are particularly vulnerable (Butterfield, 2002). Children absorb a larger dose per unit of body weight. Functional immaturity of the liver and kidneys lowers the ability to metabolize and eliminate certain agents (Bruckner, 2000). With aging, subtle changes in synapses, receptors, neurotransmitters and other mechanisms can lead to cognitive dysfunction (Shankar, 2010). There are structural and functional changes in the liver and kidney as well

(Cory-Slechta, 1990; Esposito & Dal Canton, 2010). Loss of bone mineral density (Theppeang et al., 2008) and changes in body fat composition (Mitchell, Haan, Steinberg, & Visser, 2003) may increase xenobiotic blood levels.

Health Status. Health status is multidimensional and dynamic, incorporating physical and mental well-being as well as recovery capability (Robine, Jagger, & Egidi, 2000). Co-morbid disease adds prognostic burden to exposure outcomes by impacting toxicokinetic and toxicodynamic processes. Conversely, environmental exposures may exacerbate pre-existing disease conditions (Herzstein, 2005). Inadequate access to healthcare may delay diagnosis and treatment of environmentally-related disease (Sexton, 1997).

The ability to recover and/or maintain health is closely tied to access and use of healthcare and social services (Lee, 2000). In the United States, a lack of health insurance is responsible for approximately 18,000 unnecessary deaths annually (Institute of Medicine, 2004). For the most part, health insurance coverage in the United States is provided to an individual and/or an individual's immediate family through the private sector (typically employer-based) or government-funded programs. Those who do not qualify for health insurance benefits are unemployed, employed part-time or engaged in seasonal or temporary work. Others cannot afford insurance premiums, copayments and co-insurance fees. One in five childbearing-aged women are uninsured (U.S. Census Bureau, 2009).

Nutritional Status. Nutritional balance is important to overall health. Mineral, vitamin and/or protein deficiency leads to dysfunction, disease and/or impaired recovery from illness or injury (Morón & Viteri, 2009). Some micronutrients prevent

bioactivation of specific environmental agents and conversely, some agents can impair the bioavailability of micronutrients (Ralston, Ralston, Blackwell, & Raymond, 2008; Twaroski, O'Brien, & Robertson, 2001). Mineral and elemental deficiencies can result in increased absorption of specific environmental chemicals (Soeters et al., 2008). Deficiency in iron and calcium may increase absorption of lead (Kwong, Friello, & Semba, 2004). Selenium can inhibit absorption of methylmercury (Ralston, Ralston, Blackwell, & Raymond, 2008). The lipophilic chemical body burden is related to total body fat. When weight loss, vigorous physical activity, pregnancy or lactation mobilizes fat stores, lipophilic chemicals are released into the blood (Herzstein, 2005). Food insecurity or the lack of enough nutritious food has been related to increased risk of fair or poor child health (Chilton et al., 2009) and depression and poor health in adults (Chilton & Rose, 2009).

Exposure-Related Attributes. These attributes are acquired through differences in proximity, activity and behavior (Lee, 2005; Sexton, 1997). These exposure-related attributes are systematically different from susceptibility-related attributes because they increase the likelihood of exposure to environmental contaminants.

Acculturation. As discussed previously in this chapter, since acculturation is considered to be a contributing factor to exposure-related activities and behavior choices, acculturation was included in this dissertation.

Proximity to Sources. "Built" environments (e.g., residences, schools and workplaces) are primary sources of environmental contaminants. Statistical relationships have been found among race, poverty, age and residential proximity to industrial sources of pollution (Perlin, Wong, & Sexton, 2001). Relative proximity to

stationary sources (e.g., industries, landfills and hazardous waste sites), increases the likelihood and magnitude of exposure (Aelion, Davis, McDermott, & Lawson, 2009). The degree of environmental contamination has been correlated with population density as well as industrial and agricultural intensity (Schwela, 2000). Sixty-eight percent of the U.S. population lives within an urbanized area with a population density of 50,000 people or more (U.S. Census Bureau, 2000). Models predict North American urban intake fractions to be one order of magnitude higher than rural intake fractions (Humbert et al., 2009).

Americans spend 87% of their time indoors in residences, schools and workplaces with an additional 5% spent in transit (Klepeis et al., 2001). Indoor environmental contaminants have been estimated to be 1,000 times more likely to result in exposure than outdoor sources (Ilacqua, Hanninen, Kuenzli, & Jantunen, 2007) and persist over longer periods of time (Carpi & Chen, 2001). As a result, this dissertation addressed indoor sources of exposure only.

Sixty-nine percent of all occupied housing units are owner-occupied with 79% as single-family homes (U.S. Census Bureau, 2000). Housing quality has been shown to be correlated with environmental contaminant levels (Jacobs, Wilson, Dixon, Smith, & Evens, 2009). Continuous contamination sources include emissions from building materials with intentional agent additives (e.g., lead and methylmercury in paint, PCBs in piping and caulking) or unintentional contaminants such as lead dust or airborne PCBs and mercury (Harrad, Hazrati, & Ibarra, 2006). Discontinuous contaminated sources are associated with smoking and household maintenance such as cooking, cleaning and vector control agents (Whyatt et al., 2003). Even though lead in new

house paint was banned in 1978 (U.S. Environmental Protection Agency, 2010a), PCB manufacturing was banned in 1979 (U.S. Environmental Protection Agency, 2009d), and mercury in new latex paint was banned in the early 1990s (Weschler, 2009), these three chemicals can be found in most older homes. Agocs et al. (1990) measured potentially hazardous levels of mercury in homes painted with interior latex paint. In 2002, a cross-sectional study by Kim, Staley, Curtis, and Buchanan found a direct correlation between the age of the house and the mean blood lead level of resident children. And in 2004, Herrick, McClean, Meeker, Baxter and Weymouth found extensive PCB contamination in schools and other buildings. This study used age of residence as an indicator of potential environmental chemical contamination.

Occupation. Each workplace and each occupation has both a commonality to product and process and a unique combination of hazards, varying potential for exposures and a continued risk for injury or illness or exacerbation of a pre-existing injury or illness. A working population is generally healthier than the overall population, so prevalence rates of disease conditions may differ between these two groups. Those employees most affected by an acute occupational exposure will most likely request transfer to a different position or self-terminate employment. This is referred to as the "healthy worker effect" (Monson, 1980). Adults spend 24 to 36% of their time at work. A working lifetime spans many decades and, as such, working adults are more likely to experience long-term health effects from lower levels of exposures to environmental contaminants. Many reproductive toxicants are found in traditionally female-dominated employment sectors specifically, healthcare and service (McDiarmid & Gehle, 2006). Minorities represent 28% of the workforce.

Hispanic men and African-American women represent the largest subgroups of minority workers (Lusk, Connon, Dirksen & Miller, 2001). Minorities are employed disproportionately in high-risk occupations and they hold lower paying jobs than Non-Hispanic White coworkers (U.S. Census Bureau, 2003b). While past veteran/military service may present a potential source of exposure, it was not included in this study. Additionally, all military personnel were excluded from participating in the NHANES survey.

Activity Patterns. Those exposure-related attributes associated with activity include recreational or subsistence hunting and/or fishing and vigorous physical activity (Lee, 2005; Sexton, 1997). Non-occupational or recreational activities such as hunting and fishing are potential sources of environmental chemical exposure if what is hunted and caught is contaminated and consumed. Immigrant, poor and indigenous populations are known to engage in subsistence fishing and hunting (Dellinger, 2004; Mariën & Patrick, 2001; Tsuji et al., 2008; Weintraub & Birnbaum, 2008). While these activities may be important sources of exposure among certain population subgroups (Dellinger, 2004; Mariën & Patrick, 2001; Tsuji et al., 2008; Weintraub & Birnbaum, 2008), this variable could not be included in this study because the number of study participants engaged in these activities was too small. Vigorous physical activity can mobilize fat stores, thus releasing lipophilic chemicals into the blood (Herzstein, 2005). Physical activity was not included in this study.

Those exposure-related attributes associated with behavior include diet, drinking water supply, alcohol consumption and tobacco use (Lee, 2005; Sexton, 1997).

Diet. Domestic and imported produce, meats, dairy, seafood and freshwater fish are primary sources of these environmental chemical exposures for adults (Clarkson, Amin-Zaki, & Al-Tikriti, 1976; Curley et al., 1971; Dórea, 2008; Mahaffey, Clickner, & Jeffries, 2009; Schecter & Piskac, 2001; Stewart et al., 1999). These persistent chemicals biomagnify in wild piscivorous fish, mammals and birds at relatively higher levels than non-predatory species with intraspecies variability occurring with habitat diversity (Scheuhammer, Meyer, Sandheinrich, & Murray, 2007).

Drinking Water Supply. Drinking water becomes contaminated as a result of industrial effluent, agricultural runoff, sewage treatment discharge, storm water, urban street runoff, atmospheric deposition, naturally-occurring inorganic and organic substances and residential water delivery systems (Ritter et al., 2002). Municipalities are required by the U.S. Environmental Protection Agency to test potable water for certain environmental chemicals and to initiate proper mitigation procedures when maximum contaminant levels are exceeded (U.S. Environmental Protection Agency, 2009c). However, this does not address lead and PCB contamination from water delivery systems inside residences or private-owned drinking water sources (Kim & Herrera, 2010; Palmer, Wilson, Casey, & Wagner, 2010). Fifteen percent of the U.S. population may be at increased risk for environmental chemical exposure since they rely on privately-owned drinking water sources not regulated by the EPA. Commercially available water treatment devices may remove some but not all contaminants (U.S. Environmental Protection Agency, 2002).

Alcohol Consumption. The prevalence of alcohol consumption among U.S. childbearing-aged women is 53% (Tsai & Floyd, 2004). Of those women surveyed,

29% reported consuming an average of five or more drinks on typical drinking days and 12% reported binge drinking. On average, 21% consumed 45 drinks per month (Tsai, Floyd, Green, & Boyle, 2007). Since the liver is the primary organ of detoxification and elimination by metabolism of many chemicals, interaction between alcohol and an environmental chemical may be toxicokinetic or toxicodynamic (Alessio, Apostoli, & Crippa, 1995; Mumenthaler, Taylor, & Yesavage, 2000; Toffoletto, Crippa, & Torri, 2007). Alcohol impairs two micronutrients important to fetal development: folate (Hamid, Wani, & Kaur, 2009) and calcium (Nagy, 2000). Increasing frequency of drinking in late pregnancy has been associated with increasing umbilical cord blood lead levels relative to maternal blood lead levels (Harville et al., 2005). Additionally, alcohol consumption has been associated with increased concentrations of PCBs in breast milk fat (Dewailly et al., 1996). Alcohol potentiation of prenatal methylmercury- and lead-related toxicities has been demonstrated in animal studies (Gupta & Gill, 2000; Turner, Bhatnagar, & Yamashiro, 1981; Maia et al., 2009a), but not of PCBs (Krampl, Kontskova, & Kramplova, 1980). However, PCBs have been shown to be hepatotoxic in animal studies (Agency for Toxic Substances and Disease Registry, 2000).

Tobacco Use. In 2000, 21% of U.S. women smoked cigarettes (Trosclair, Husten, Pederson, & Dhillon, 2002). More women than men are exposed to environmental tobacco smoke that is, "secondhand smoke" (Wipfli et al., 2008). Tobacco contains over 400 identified chemical compounds (Kutlu, Karagozler, & Gozurkara, 2006) including nicotine, cadmium, lead, chromium, nickel (Pereg, Lagueux, DeWailly, Poirier, & Ayotte, 2001) and dioxin-like PCBs (Uehara,

Nakamura, Matsuura, Kondo, & Tada, 2007). Blood lead levels increase with both active and passive smoking (Kutlu, Karagozler, & Gozurkara, 2006; Willers, Gerhardsson, & Lundh, 2005). However, smoking has been associated with decreased levels of dioxin-like PCBs (Uehara et al., 2007). When cigarettes become contaminated through airborne deposition of chemicals and/or insufficient handwashing, secondary inhalation of environmental chemicals can occur as well (Askin & Volkmann, 1997).

Socioeconomic Factors. These factors affect the ability to be prepared and/or to recover. Factors such as education, employment and income influence health indirectly through complex interactions with susceptibility- or exposure-related attributes or both (Sexton et al., 1993a). These interactions may result in inequalities in safe and healthy housing, nutritional status, health status and risk-related behaviors (Flaskerud & Winslow, 1998; Mechanic & Tanner, 2007; Nyamathi et al., 2007).

Education. In 2000, 81% of all U.S. women had completed high school and approximately 23% had earned a bachelor's degree (Bauman & Graf, 2003). Higher educational attainment has been associated with better physical health (Winkleby, Jatulis, Frank, & Fortmann, 1992; Zajacova & Hummer, 2009). Johnson et al. (2009) speculated that more educated people may manage their environments better to protect their health. Health literacy may influence one's ability to make healthcare-related decisions (Smith, Trevena, Nutbeam, Dixon, & McCaffery, 2009). Overall, education is viewed as the key to increased opportunities for employment and higher income potential (Adler & Newman, 2002).

Employment. In 2000, 50% of all childbearing-aged women worked (Clark & Weismantle, 2003). Of these women, 50% worked usually fewer than 35 hours per week and 5.5% held more than one job (U.S. Bureau of Labor Statistics, 2005). When workers become unemployed, their health is likely to suffer. Based on data from the U.S. Panel Study of Income Dynamics (1999-2003), involuntary job separation increased the odds of reporting fair or poor health by 56% and the odds of reporting a new health problem by 84%, regardless of race-ethnicity (Strully, 2009). However, with voluntary job separation, the odds of reporting fair or poor health increased to 84% for Non-Hispanic Black and Hispanic respondents as compared to Non-Hispanic White respondents. It was not possible to determine whether poor health was the primary reason for voluntary job separation. Gender differences were not examined. In 2002, the unemployment rate was 9.5% for mothers who were single, widowed, divorced or separated with children under 18 years – twice that of married mothers of similar age with children under 18 years (U.S. Bureau of Labor Statistics, 2003). Among women, unemployment has been associated with increased tobacco use (Novo, Hammarström, & Janlert, 2000) but not increased alcohol consumption (Gore, Harris, & Firestone, 2004).

Income. In 2000, 17% of women aged 18-64 and 36% of female heads-of-household with children under 18 had incomes below the federal poverty threshold (U.S. Census Bureau, 2008). Women comprise more than 61% of minimum wage workers (Lichtenwalder, 2005). Mortality and morbidity rates as well as self-assessments of poor health are substantially higher among the poor (Lu, Samuels, & Wilson, 2004; Mackenbach et al., 2008; Montgomery & Carter-Pokras, 1993) and

especially poor women (Nagahawatte & Goldenberg, 2008). Larson and Halfon (2009) determined income was strongly and significantly related to health outcomes in children. The percentage of children in poor health increased with decreasing family income for 15 health indicators with the steepest income-to-health gradient at 100% below the federal poverty level.

Marital Status. While this variable was not included in the modified environmental health paradigm, marital status is an important socioeoconomically-related factor for women and their overall health (Schoenborn, 2004; Skalická & Kunst, 2008; Wickrama et al., 2006). As a result, it was included in this dissertation.

Race-Ethnicity. Health disparities among racial and ethnic minorities are well known (Morello-Frosch & Lopez, 2006a; Morello-Frosch & Shenassa, 2006b; Payne-Sturges & Gee, 2006). As stated previously, there is considerable evidence of inequitable distribution of the costs and benefits associated with environmental regulations among vulnerable communities. The placement of hazardous waste sites, landfills, incinerators and polluting industries is more common in communities inhabited mainly by low income groups and racial and ethnic minorities (Bullard, 1990; Johnson, Harris, & Williams, 1992; Mohai, & Bryant, 1992b; United Church of Christ Commission for Racial Justice, 1987; U.S. Government Accounting Office, 1983; U.S. Environmental Protection Agency, 1992a). Race-ethnicity may serve as proxy variables for residential segregation and social isolation (Acevedo-Garcia & Osypuk, 2008) and/or reflect institutional environmental discrimination (Gelobter, 1992; Lee, 1992). Each of these factors could influence susceptibility, exposure and health (Merkin et al., 2009).

This concludes a description of the major constructs of the modified environmental health paradigm upon which this dissertation was based. The last section of this chapter will return to the toxicokinetic and toxicodynamic stages for a review of the scientific literature pertaining to *in vivo* and *in vitro* mechanistic studies of binary chemical interactions and human studies related to exposure to all three chemicals.

Chemical Interaction Models

Despite what is known about the hazards of exposure to these specific environmental chemicals, the health effects from exposures to combinations and permutations of these environmental chemicals and their corresponding biologically-effective dose are relatively unknown. One would expect them to be more severe than those from exposure to a single specific chemical.

The U.S. Agency for Toxic Substances and Disease Registry (ATSDR) developed interaction models to evaluate chemicals with a common target site such as neurodevelopment or a single exposure source such as breast milk. To estimate the influence of binary interactions on toxicity, their meta-analyses assessed the "mechanistic" (chemical-chemical, toxicokinetic, toxicodynamic) understanding of the interaction, the toxicological significance of the interaction, the presence of any modifiers (i.e., route of exposure, exposure duration and sequence) and existing *in vivo* and *in vitro* data (Agency for Toxic Substances and Disease Registry, 2001; Mumtaz & Durkin, 1992). The endpoint of analysis was a binary weight-of-evidence (BINWOE) score as a weighted factor and an estimate of the direction of interaction (Appendix D: Assessing Chemical Interactions). Limitations of this modeling include

analyses of binary interactions only (Callahan & Sexton, 2007) and the general lack of data quality weighting factors (Monosson, 2005). Both of these limitations may underestimate the effects of these chemical interactions (Chen et al., 2001; Wilkinson et al., 2000). In general, there is a lack of data and understanding of the complex mechanisms of neurotoxicity by these chemicals.

ATSDR estimated the direction of interaction for neurotoxicity to be additive for lead on methylmercury and methylmercury on lead (Agency for Toxic Substances and Disease Registry, 2006) and greater-than-additive interactions for methylmercury on PCBs and PCBs on methylmercury (Agency for Toxic Substances and Disease Registry, 2004). To date, interactions of lead on PCBs and PCBs on lead have not been evaluated by ATSDR.

***In Vivo* and *In Vitro* Studies**. The following section begins with the scientific literature that served as the basis for these ATSDR prognostications. In general, these studies have focused either on the mechanisms of neurotoxicity or the clinical and/or subclinical manifestations of adverse development related to maternal exposures: death, malformation, growth retardation and/or functional defect (Wilson, 1959, 1973; Wilson & Fraser, 1977). These studies are summarized below.

Lead and Methylmercury. ATSDR (2006) characterized the direction of interaction for neurotoxicity of lead on methylmercury and methylmercury on lead as additive with moderate to moderately low uncertainty. No BINWOE score was provided (Agency for Toxic Substances and Disease Registry, 2006). This risk characterization (=III.C.) was based upon the evidence of two animal studies (Bellés, Albina, Sánchez, Corbella, & Domingo, 2002; Congiu et al., 1979).

Bellés, Albina, Sánchez, Corbella and Domingo (2002) exposed pregnant mice on gestation day 10 to lead nitrate (25 mg/kg) subcutaneously then, five minutes later, administered methylmercury chloride (12.5 mg/kg) by gavage. These mice were sacrificed on gestation day 18. Three fetuses from each dam were autopsied. Data were evaluated using one-way ANOVA, χ^2 analyses and independent sample *t*-tests. Binary chemical exposure was associated with a statistically significant increase ($p < 0.05$) in maternal deaths and a significant decrease in the number of litters than either chemical alone, indicating a synergistic effect for maternal toxicity. By comparison, no statistically significant differences were found in fetal deaths or physical anomalies. As a result, these researchers concluded lead and methylmercury to be additive for fetal toxicity at the doses tested.

In the study by Congiu et al. (1979), male rats were injected with lead nitrate (20.7 mg/kg), then given methylmercury chloride (0, 34.6, 39.6 or 44.6 mg/kg) by gavage 24 hours later. An equal number of rats were sacrificed prior to gavage and at 6 and 24 hours following gavage. Data were evaluated using ANOVA and Fisher's *t*-test. Pretreatment with lead nitrate potentiated methylmercury chloride in a dose-related response resulting in a higher mortality rate than among those given methylmercury chloride alone. Dose-dependent differences could account for these varying results (Meacham et al., 2005).

Since these studies were published, four *in vitro* and *in vivo* mechanistic studies on the neurotoxic interaction between methylmercury and lead were published in English from January, 2000 to December, 2009.

In the study by Chetty, Rajanna, Hall, Yallapragada and Rajanna (1996), rats received lead acetate (25 mg/kg) or methylmercury chloride (5 mg/kg) by intraperitoneal infusion for 3 or 24 hours. Another group of rats received lead acetate (25 mg/kg/day) or methylmercury chloride (2.5 mg/kg/day) for seven days. These researchers found lead and methylmercury each enhanced the binding affinity of two receptors to cerebellar intracellular membranes in a concentration-dependent manner when compared to controls ($p < 0.05$). Each chemical had a slightly different effect on each receptor. It is believed that these two receptors are integral to intracellular calcium regulation which in turn, influences neuronal activity.

In their 1997 *in vitro* study, Rajanna, Rajanna, Hall and Yallapragada examined the effect of methylmercury chloride or lead acetate on the binding affinity of a different receptor (NMDA) in neonatal (ten days old) and adult rat cerebral cortices. These researchers found significant dose-dependent inhibition of this binding affinity. These effects were more pronounced in the neonatal brain than in the adult brain.

The remaining two studies used *in vitro* toxicity assays to predict cellular level effects and identify toxic mechanisms of these chemicals on neural cells and cloned neural cells (Suñol, 2010).

Radio, Freudenrich, Robinette, Crofton and Mundy (2010) cultured cerebellar granule cells with astrocytes prepared from six-to-eight day-old rats. Separately, a PC12 cell clone, Neuroscreen-1™ was treated with nerve growth factor. Both cell cultures were optimized over eight days to ensure neurite outgrowth and cell viability. Then, each cell culture was exposed once to methylmercury chloride or lead chloride in concentrations from 1 μM - 100 mM. Total neurite length and cell viability were

examined 96 hours after exposure. Data were analyzed using two-way ANOVA, Student-Newman-Keuls' and Dunnett's tests. Lead and methylmercury each inhibited neurite outgrowth significantly ($p < 0.05$) in both culture types. Unlike lead chloride, methylmercury chloride affected neurite length at concentrations less than those that affected cell viability. Each chemical demonstrated preference for different cell types.

Hogberg, Kinsner-Ovaskainen, Coecke, Hartung and Bal-Price (2010) prepared primary cultures of neuronal and glial cells from 7-day-old rat cerebellar granule cells. They incubated these cultures for 24 hours then exposed them to lead chloride or methylmercury chloride for up to 12 days. Cells were evaluated at 1, 4, 8 and 12 days for neurite outgrowth and the later stages of morphological maturation by measuring gene expression of messenger ribonucleic acid (mRNA) for specific neuronal and glial markers using spectrophotometry. After logarithmic transformation of data, statistical analyses included one- and two-way ANOVA. These researchers found neuronal markers were more sensitive to methylmercury chloride while lead chloride affected glial markers.

PCBs and Methylmercury. ATSDR (2004) characterized the neurotoxic interaction effect of PCBs on methylmercury and methylmercury on PCBs as greater-than-additive with a BINWOE score of +0.20 (Agency for Toxic Substances and Disease Registry, 2004, pp. 90-93). This risk characterization (II.C.b.) was based upon the evidence of impaired neurodevelopment determined by one *in vitro* study (Bemis & Seegal, 1999) with a moderate degree of uncertainty due to one negative *in vivo* study (Tanimura, Ema, & Kihara, 1980).

In the Bemis and Seegal study (1999), dopamine concentrations were significantly decreased ($p \leq 0.001$) in adult rat brain (striata) when exposed to PCBs (1:1 mixture of Aroclor™ 1254/1260) and methylmercury as compared to either chemical alone. Aroclor™ is a commercially available PCB mixture. These observed values were lower (20-50%) than predicted values, suggesting a synergistic effect. To control for unequal cell size, data were analyzed using one-way ANOVA (*F* statistic) so that the varying number of observations could be weighted. Interactions were analyzed using two-way ANOVA with Bonferroni-corrected post hoc *t*-tests. Results were reported as a percentage of the average control value in order to reduce variance among the 14 individual experiments. These researchers attributed this synergy to a common site of action involving intracellular calcium regulation in neural cells.

By comparison, Tanimura, Ema, and Kihara's (1980) study results were inconsistent. While mortality was higher than controls among offspring of female mice exposed throughout gestation and lactation to Kanechlor™ , another commercially available PCB mixture at 500 ppm and methylmercury chloride at 0, 0.4, or 4 mg/kg in a dose-related response, there were no statistically significant differences in neurodevelopment and neurobehavioral tests among binary exposed groups versus singularly exposed groups; an additive effect.

In addition to these studies, 14 *in vitro* and *in vivo* mechanistic studies specific to neurotoxicity were published in English from January, 2000 to December, 2009. These studies demonstrated interaction between PCBs and methylmercury after preconceptual, gestational and/or lactational exposure (Coccini et al., 2007; Fischer, Fredriksson, & Eriksson, 2008). However, the characterization of this interaction

varied widely from antagonistic (Bemis & Seegal, 2000; Sitarek & Gralewicz, 2009; Vettori et al., 2006) to non-additive (Coccini et al., 2006; Widholm, Villareal, Seegal, & Schantz, 2004) to additive (Castoldi et al., 2006; Costa, Fattori, Giordano, & Vitalone, 2007; Roegge et al., 2004) to synergistic (Bemis & Seegal, 2000; Cheng et al., 2009). Goldoni et al. (2008) found asynchronous exposure produced antagonism when methylmercury preceded PCB 153 and additivity when PCB 153 preceded methylmercury. Gender differences in genetic expression were found among perinatally-exposed adult rat progeny (Padhi et al., 2008). These varying results may be due to differences among the mechanisms studied, outcomes evaluated and variability in tissue-, time- and dose-dependent bioaccumulation (Meacham et al., 2005).

PCBs and Lead. ATSDR did not characterize the interaction of lead and PCBs. A literature search of titles and abstracts was conducted in PubMed using keywords (PCBs and Pb and interaction). This search revealed no *in vitro* and *in vivo* mechanistic studies specific to neurotoxicity for PCBs on lead or lead on PCBs published in English from January, 2000 to December, 2009.

Human Studies. To date, few human studies have examined exposures to combinations of these environmental chemicals among women of childbearing age. A literature search was conducted in PubMed using keywords (methylmercury and Pb and PCBs and women) for studies published in English from January, 2000 through January, 2010. Studies of maternal exposures with neonatal outcomes were excluded. Search parameters were extended until two studies emerged. They are reviewed here.

Qin et al. (2010) found significantly higher PCB ($p < 0.05$), mercury ($p < 0.01$) and lead ($p < 0.01$) levels in the subcutaneous adipose abdominal tissue of ethnic Chinese women living in Hong Kong who were diagnosed with non-cancerous tumors of the uterus (uterine leiomyomas) versus those women who did not have this diagnosis. Statistically significant differences ($p < 0.01$) were found between these two groups for lead and mercury in visceral fat. These adipose tissue samples were obtained during elective abdominal surgery (24 cases) and liposuction (20 controls) performed at six hospitals and six cosmetic surgery clinics in Hong Kong. Questionnaires were administered by trained interviewers regarding age, weight, height, number of seafood meals per week, health status and medical history. Gravidity and lactation histories were not elicited. Any woman with a history of UL was excluded from the control group. Analyses of samples were conducted using cold vapor atomic fluorescence spectrometry for total mercury and inductively coupled plasma-optical emission spectrometry for lead, and gas chromatography-mass spectrometry for PCBs. Researchers did not document where these samples were analyzed or what quality control procedures were executed. Data were analyzed using Student's *t*-test, Duncan's multiple range tests and Pearson's correlation. Correlations of xenobiotic levels between chemical pairs were not calculated. Xenobiotic levels were strongly correlated with increased seafood consumption, body mass index, and age. Limitations of this study included small sample size.

In the cross-sectional study conducted by Denham et al. (2005), tribal members collected blood samples from 138 Akwesasne (Mohawk) Nation girls aged 10 to 16.9 who resided within ten miles of the Mohawk Nation's border. Tribal members who

collected the data had no prior knowledge of exposure status at time of data collection. Attainment of menses was self-reported as present or absent at time of blood sampling. Demographic data were obtained from the girls' mothers by trained Akwesasne interviewers. Those with fetal alcohol syndrome or other serious physical or mental condition as diagnosed by a physician were excluded. Analyses of samples were conducted using cold vapor atomic fluorescence spectrometry for total mercury, Zeeman-corrected graphite furnace atomic absorption spectrometry for lead, and gas chromatography with electron capture detection for lipid-adjusted PCBs. Heavy metals were analyzed by a different laboratory than that used to analyze congener-specific PCBs. No details were provided regarding laboratory quality control procedures. Values were logarithmically transformed prior to statistical analysis. The median age at menarche was 12.2 years, comparable to the distribution found in NHANES III (1997-1998). Binary logistic regression analyses were performed on single toxicants and total toxicants. Age and lower socioeconomic status were strongest predictors of menses onset. Body mass index (BMI) did not affect the model. The odds of having reached menarche decreased with blood lead levels above the geometric mean; this relationship was nonlinear. Similar results were found with levels of four estrogenic PCB congeners (52, 70, 101/90, 187). While a nonlinear effect of mercury was observed, it was marginally significant ($p = 0.08$) at the 95th percentile. Lead and PCBs were found to have a statistically significant interaction ($p < 0.05$). Limitations of this study included small sample size which affected the researchers' ability to test interactions.

Chapter Summary

Existing definitions and measurements of exposure in five disciplines central to environmental health were explored. The definition and measurement of exposure was aligned with current principles and practices in environmental health nursing. Six exposure-related concepts (environment, agent, human, dose, health and vulnerability) were identified and defined. After a transdisciplinary review, the conceptual framework chosen for this dissertation was Sexton, Olden and Johnson's modified environmental health paradigm (1993a) because it so aptly described exposure and the intersection of an agent's toxicity with the target's vulnerability. The historical and political context of this framework's development, stated purpose and goals, focus, scope and basic assumptions were outlined. A brief review of published research studies and critical analyses that tested the framework's concepts and operational constructs were provided. To date, no known tests for validity and reliability have been performed on this model.

Selection of the three chemicals of interest (lead, methylmercury and PCBs) was based upon evidence of their pervasiveness, persistence and co-occurrence in the environment; the existence of scientific evidence demonstrating that exposure to any one of these chemicals has neurobehavioral and/or neurodevelopmental consequences in animal models and human population studies; and existence of scientific evidence that these chemicals bioaccumulate in such a way that past and current maternal exposures have the potential for transgenerational consequences.

Despite what is known about the hazards of exposure to these specific environmental chemicals, the health effects from exposures to multiple environmental

chemicals and their corresponding biologically-effective dose are relatively unknown. *In vivo* and *in vitro* mechanistic studies of binary combinations of these chemicals are limited and their findings contradictory. These contradictions may be due to differences among the mechanisms studied, outcomes evaluated and variability in tissue-, time- and dose-dependent bioaccumulation. A search of the scientific literature identified only two human studies that evaluated health outcomes of exposures to each of these three chemicals among childbearing-aged women.

Once the concepts were defined and theoretical framework chosen, the data source was searched for congruent measures of the independent variables. These measurements as well as their validity and reliability are described in the next chapter. Other details in Chapter Three include detailed information on NHANES, data processing, analytic procedures and research ethics.

CHAPTER 3

METHODOLOGY

This chapter begins with a reiteration of this study's aim and research questions. Then, the choice of research design is discussed followed by a description of the data source that includes a brief summary of the origin of NHANES. Three major concerns involving the use of these existing data are addressed. A description of the dataset and study population are provided. Measurements of all dependent and independent variables are described and their validity and reliability are reviewed. Data processing and analytic procedures are detailed. Aspects of research ethics are discussed with regard to NHANES and this study.

Aim

The aim of this research was to examine childbearing-aged and pregnant childbearing-aged women's exposures to specific environmental chemicals known to have neurobehavioral and neurodevelopmental consequences in animal models and human population studies. This dissertation focused on exposures to each of these chemicals individually and in four different combinations and permutations. Additionally, this dissertation identified those population subgroups at highest risk for two or more xenobiotic (chemical-specific) blood levels at or above the geometric mean. This research used existing data from the National Health and Nutrition Examination Survey (NHANES), a national probability sample.

Research Questions. This study had three research questions:

1. What was the prevalence of childbearing-aged and pregnant childbearing-aged women's exposures to each of the following environmental chemicals: lead, methylmercury and polychlorinated biphenyls (PCBs) as measured by chemical-specific (xenobiotic) levels at or above geometric mean in blood or serum of these women who were living in the United States from 1999 through 2004?

2. What combinations and permutations of chemical exposures were most common among these childbearing-aged and pregnant childbearing-aged women as evidenced by xenobiotic blood levels at or above the geometric mean?

3. What, if any, subsets of childbearing-aged women were disproportionately exposed to two or more of these environmental chemicals based on susceptibility-related attributes (reproductive status, age, health and nutritional status), exposure-related attributes related to acculturation, proximity (residential characteristics and occupation), activity (diet and tap water supply) and behavior (alcohol consumption and tobacco use); socioeconomic factors (education, employment, income and marital status) and race-ethnicity?

Choice of Research Design

As little is known about exposures to combinations of these environmental chemicals among childbearing-aged and pregnant childbearing-aged women, this research was a descriptive and exploratory study. A cross-sectional study design was the best study design for determining the prevalence of exposure(s) among this population as a whole and within subgroups. A cross-sectional study design reveals patterns and connections among exposures and specific characteristics of vulnerability

based on existing scientific literature (Kleinbaum, Kupper, & Morgenstern, 1982). Such findings allow for the generation of new hypotheses that can be subsequently evaluated using more robust study designs including longitudinal, prospective cohort and case-control studies. Because a cross-sectional study is non-directional that is, all data are collected at a single point in time, this study design is particularly useful in describing exposures which have inherent individual variability and uncertainty in measurement. However, a very large number of study participants are required for such a study to detect differences among population subgroups. A study design that employs random probability sampling for selecting its study participants provides representative estimates of exposures. Such estimates are useful in future public health planning. Although all these study design attributes are desirable, a cross-sectional study in which a large amount of original data are collected and encoded is prohibitively time-consuming and expensive. As a result, a more practical and economical approach is to conduct a secondary analysis of existing cross-sectional data.

Description of Data Source

This study's research questions were addressed through secondary analysis of existing data from the National Health and Nutrition Examination Survey (NHANES), 1999 through 2004. NHANES is a continuous population-based survey from the Centers for Disease Control and Prevention (CDC) National Center for Health Statistics (NCHS). Data are publicly available online. NHANES provides a probability sample of baseline information on the health and nutritional status of the non-military, non-institutionalized adults and children living in the United States. As

part of this survey, biomonitoring data were collected for more than 116 environmental chemicals or their metabolites including all the chemicals of interest to this study (Centers for Disease Control and Prevention, National Center for Environmental Health, 2007).

Origin of NHANES

The need for scientific measurement of the health and well-being of the people living in the United States was recognized decades before the National Health Survey Act became law in 1956 (Khrisanopulo, 1963). By 1956, data from a previous national study were obsolete and data from regional studies were limited in scope. There was a growing demand for uniform and valid national morbidity and healthcare-related statistics, particularly as they related to chronic disease. Projected applications for these statistics included administrative planning, workforce availability, potential consumer markets, health education, provision of health services and medical research (Storck, 1966; U.S. National Committee on Vital and Health Statistics, Public Health Services, Division of Public Health Methods, 1957). The purpose of the U.S. National Health Survey Act (1956) was

> to provide for a continuing survey and special studies to secure on a non-compulsory basis accurate and current statistical information on the amount, distribution, and effects of illness and disability in the United States and the services received for or because of such conditions and for studying methods and survey techniques for securing such statistical information with a view toward their continuing improvement.

Plans for a continuing national health survey were crafted by consensus committees comprised of stakeholders from federal, state and city governments, healthcare, academia and insurance. Most notable was the careful consideration of conceptual definitions. For example, health was "a continuous scale of well-being" and morbidity

was “a general word to be used to designate illness (manifest and non-manifest), injuries, and impairments” (Khrisanopulo, 1964, p. 4). There was an acknowledgment that analysis would be at the individual level even though the study population was the general population or some segment of it (Linder, 1958).

As a result of the National Health Survey Act, the National Center for Health Statistics (NCHS) was created. The first National Health Examination Survey (NHES I) was conducted in 1960. National attention on the link between dietary habits and disease led to the addition of continuing nutrition surveillance to the survey in 1971 (Editor, 1969). By 1977, there was an increased awareness of the influence of environment on health and the need to collect environmental health statistics for conducting epidemiological studies (U.S. National Committee on Vital and Health Statistics, 1977). As a result, NHANES was broadened to include measures of environmental exposures (Appendix E: History of NHANES).

Secondary Data Analysis

There were three major concerns involving the use of these existing data: selection and feasibility criteria; theoretical and conceptual congruency between the original and new research questions; and internal and external validity and reliability issues in selection and recruitment of original survey participants, survey content, and data collection, encoding and analysis. As a result, a thorough review of NHANES was conducted so that real and potential biases within the original research could be identified. This review provided some anticipatory guidance in selecting specific variables and implementing statistical controls prior to analysis.

Selection and Feasibility Criteria. NHANES was selected because this dataset best answered this study's research questions in that it contained a plethora of information on environmentally-related exposures of interest. The NHANES dataset was compatible with available hardware and software and the data have been subjected to vigorous control standards. NHANES provided supporting documentation specifically, codebooks, notations on recoding, and information regarding the data collection process so that the quality of the data was able to be assessed properly (Centers for Disease Control and Prevention, National Center for Health Statistics, n.d.b). These data were obtained in an ethical manner with regard to informed consent and confidentiality and there were provisions for continued protection of participant identity (Centers for Disease Control and Prevention, National Center for Health Statistics, 2010c). These data were publicly and freely available online at http://www.cdc.gov/nchs/nhanes.htm.

Theoretical and Conceptual Congruency. NHANES was theoretically congruent with this research study. It was a semi-quantitative survey that was structured, cross-sectional and non-experimental. Since this study was a quantitatively-based study, preference was given to laboratory-based and/or objective measurements whenever possible and appropriate. Informed by Sexton, Olden and Johnson's modified environmental health paradigm (1993a) and the literature review, NHANES was scrutinized to identify measures which best represented the concepts. Both the level of analysis and unit of analysis were compatible with this study.

Recognition of real and potential biases within the original research provided some anticipatory guidance when specific variables were selected and statistical

controls were implemented (Kneipp & Yarandi, 2002). This study remained congruent with the sampling parameters of the original dataset. For example, even though all female participants aged 12 to 59 and menstruating females as young as eight were tested for pregnancy status, NHANES tested women aged 16 to 49, inclusively for the three chemicals of interest (Centers for Disease Control and Prevention, National Center for Health Statistics, 2009i). To remain congruent with the sampling parameters of the original dataset, female children ages 16 to 21 were included in this study and females younger than age 16 and older than 49 were excluded. This exclusion may have underestimated true pregnancy rates slightly.

Validity and Reliability.

Selection of Original Survey Participants. NHANES employed a four-stage, unequal probability and cluster sampling method to select study participants from the U.S. population. For each twelve-month period, NHANES selected twelve to fifteen counties (some contiguous) from across the United States and divided them into block segments using U.S. census data. Individual block segments were identified for sampling and subsequently, a cluster of households from each selected block segment was drawn at random. The probability-proportional-to-size (PPS) sampling technique for selecting counties and block segments ensured that the probability of selecting any one sampling unit was proportional to the U.S. population with the characteristic of interest such as geography and the proportion of minority populations (Centers for Disease Control and Prevention, National Center for Health Statistics, 2009h). Subsequentally, NHANES randomly selected from within these screening sub-domains (age, sex, and race-ethnicity) one or more residents from each household to

be study participants using quota sampling with replacement (Centers for Disease Control and Prevention, National Center for Health Statistics, 2010b). Using quota sampling with replacement does not insure that those who chose not to participate in the survey were identical to those who chose to participate in the survey. However, in their examination of the 2000 NHANES survey, Wendler et al. (2006) compared consent rates among Non-Hispanic Whites, Non-Hispanic Blacks and Hispanics for the interview and medical examination portions of the survey. Odds ratios (*OR*) were calculated. Non-Hispanic Blacks were less likely than Non-Hispanic Whites to participate in the initial interview (*OR* = 0.97) while Hispanics were slightly more likely to participate (*OR* = 1.63). Only those individuals who consented to be interviewed were invited to participate in the medical examination portion of the survey. Non-Hispanic Blacks and Hispanics were more likely than Non-Hispanic Whites to participate in the medical examination with odds ratios 1.04 and 1.56, respectively. However, these last findings were not statistically significant.

In NHANES, there was a purposeful over-sampling of select subgroups: adolescents, the elderly, African-Americans, Mexican-Americans and low-income Non-Hispanic White-Americans. Oversampling increased the reliability and precision of health status indicator estimates for these subgroups (Centers for Disease Control and Prevention, National Center for Health Statistics, 2009i). The tests administered for each individual were based on probability sampling. A sample weight was assigned to each person (Centers for Disease Control and Prevention, National Center for Health Statistics, 2009i). Sample weights accounted for specific data purposely not collected that is, unequal probability of selection. NHANES based these sample

weights on U.S. census data for gender, age and race-ethnicity with references to five racial and ethnic categories, that is, Non-Hispanic White; Non-Hispanic Black; Mexican American; Other Hispanic; and Asian, Pacific Islander, Native American, or Multi-Racial (Centers for Disease Control and Prevention, National Center for Health Statistics, 2010e). Demographic analysis of the 2000 U.S. census estimated a net undercount (-0.1%) which resulted from a combination of duplicate enumeration and undocumented migration (Mulry, 2006). This sampling error underestimated overall prevalence slightly (Table 1).

Recruitment of Original Survey Participants. NHANES sent communiqués regarding the survey to the media as well as state, county and local governments. Area households received a letter of introduction. Interviewers canvassed a sample cluster and asked at each house a set of questions to determine if anyone in the house was eligible to be in the sample. Interviewers included those who were bilingual (Centers for Disease Control and Prevention, National Center for Health Statistics, 2009i).

Original Survey Content. There were four components to the NHANES survey: demographics, data collected through household and mobile examination center interviews regarding alcohol consumption, tobacco use, medical, dietary and reproductive histories; physical examinations and laboratory tests (Centers for Disease Control and Prevention, National Center for Health Statistics, 2009i). Survey content was determined through a rigorous evaluation process in which components were added, modified, supplemented or dropped across survey years (Centers for Disease Control and Prevention, National Center for Health Statistics, 2004). "It can be

changed depending upon the complexity, reliability and validity of the health measure. … It still takes about two years of data collection to have stable national estimates" (Berman, Ostchega, Reed-Gillette, & Porter, 2003, p.713).

Original Survey Data Collection, Coding and Analysis. NHANES instituted a comprehensive and integrated quality assurance and quality control program encompassing a wide range of activities that occurred prior, during and after data collection to ensure high quality data and reduce systematic error (Berman et al., 2003). Bilingual interviewers who administered the structured in-home interview used a pen-touch handheld computer. Interview data were generated from self-reports. Within a few weeks of the home interview, the physical examination and laboratory testing occurred at a Medical Examination Center (MEC) centrally located within the survey area. It took approximately 3.5 hours per person, depending upon age. Select population subgroups or a given percentage of study participants may have been included or excluded from specific MEC components and were so noted. All questions were administered by trained interviewers except those of a "sensitive" nature (e.g., illicit drug use and sexual behaviors). These questions were self-administered using audio computer-assisted technology to minimize response bias. All MECs had identical environment and equipment. To minimize interviewer bias, all field staff (physicians, medical and health technicians, dietary and health interviewers) received comprehensive and annual refresher training. The contract medical personnel conducted the examination and laboratory phases of the survey using standardized procedures. From within the MEC, digital measuring equipment automatically transmitted data to central databases. Physicians entered physical

examination data directly into a computer. Interviewers used a pen-touch handheld computer to conduct additional interviewer-administered questionnaires. These measures decreased the occurrence of transcription or coding errors. Prior to release, data were checked for inconsistencies and "scrubbed" by identifying, replacing, modifying or deleting these errors. "Don't Know" and "Refused" answers were each coded differently. Incomplete data or incomplete survey components were coded as missing. Off-site contract laboratories analyzed some of the biological and environmental specimens. All laboratory specimen-related transport, storage and analytical procedures were standardized to maximize reliability and validity. As part of the overall quality assurance process, all collection materials and storage containers used for trace element assays were initially prescreened for environmental contaminants to minimize external contamination. External contaminants can limit accuracy especially at levels approximating detection limits. Analytical methods were selected in accordance with validated standards by the Clinical Laboratory Standards Institute and have been described elsewhere (Centers for Disease Control and Prevention, National Center for Health Statistics, n.d.b, 2009a, 2009b, 2010a).

In conclusion, NHANES contained good quality and useful data which were valid and reliable. It was theoretically and conceptually congruent with this study. Details regarding specific measures of validity and reliability for each dependent and independent variable are provided in the sections that follow.

Dataset Description

The dataset for this study was the NHANES population-based survey that collected data on the health and nutritional status of adults and children in the U.S.

from 1999 to 2004. NHANES was a semi-quantitative survey that was structured, cross-sectional and non-experimental. It was a nationally representative sample even though the study excluded non-civilian and institutionalized people. There was a purposeful over-sampling of select subgroups: adolescents, elderly, Non-Hispanic Blacks, Mexican-Americans and low-income Non-Hispanic Whites to increase the reliability and precision of health status indicator estimates for these groups. To overcome these selection biases, all data were weighted, thus allowing population estimates to be calculated. NHANES data provided a basis for estimating subpopulations at-risk, for monitoring trends in prevalence (particularly risk-related behaviors and environmental exposures) and for establishing and maintaining a national probability sample of baseline information on the U.S. population (Centers for Disease Control and Prevention, National Center for Health Statistics, n.d.b).

Study Population

The subjects of this study were childbearing-aged women (16 to 49 years, inclusively) of diverse races and ethnicities who were living in the United States from 1999 through 2004. Males were excluded because this study was interested in female and maternal exposures only. Additionally, not all males were tested for all chemicals of interest. From 1999 to 2004, there were 11,865 childbearing-aged female participants interviewed (Table 2) of whom 95.6% were examined (Table 3) and one-third were tested in accordance with survey design (Table 4). From this one-third cohort, approximately 15% were dropped from the original survey subsample for the purposes of this study (Table 5 and 6) because they did not meet this study's criteria that is, female, age between 16 and 49 inclusively, interviewed, examined, tested for

all chemicals of interest and deemed to have reliable dietary recall. The final cohort for this study consisted of 3,173 women (Table 7).

While there were a total of 1,304 females whose urine tested positive for pregnancy, not all these females were of childbearing-age. Pregnant women of childbearing-age were identified as a subset; 11.5% of those examined were pregnant (Table 8) and 34.25% of those women were tested (Table 9). Approximately 13% of the pregnant childbearing-aged women were dropped from the subsample because they did not meet this study's criteria (Tables 10 and 11). The final cohort included a subset of 391 pregnant women (Table 12). The sample size was adequate for this study's purposes.

To obtain weighted estimates for 1999 to 2004, a six-year weight variable was created by assigning two-thirds of the four-year weight provided by NHANES for 1999 to 2002 if the person was sampled in 1999 to 2002 and assigning one-third of the two-year weight for 2003 to 2004 if the person was sampled in 2003 to 2004. This is possible because the 2003 to 2004 weights were comparable on a population basis to the combined 1999 to 2002 four-year weights (Centers for Disease Control and Prevention, National Center for Health Statistics, 2006). There were 52,827 observations read from the data set. Using weighted data allowed for estimation of true variance and generalizability to the U.S. population (Centers for Disease Control and Prevention, National Center for Health Statistics, 2010e).

Measurement of Dependent Variables

Biomarkers. For this study, the outcome of interest was based on evidence of biological uptake of two or more of the following chemicals: lead, methylmercury and

the summed value of four lipid-adjusted polychlorinated biphenyl congeners (118, 138/158, 153 and 180). Exposures were measured by the presence of these xenobiotics in the blood or serum of these women. A biomarker of exposure reflects the relationship between external contaminant (i.e., amount available for contact from all potential sources) and body burden (i.e., internal dose). The presence of a xenobiotic does not by itself cause disease or suggest a causal pathway. Equal xenobiotic values across chemicals do not infer relative equality in toxicity (National Research Council, 2006). Since this study examined exposures and not outcomes of said exposures, toxic equivalency across chemicals was not considered. The large sample size compensated for intra-individual exposure variability associated with intermittent exposures (Needham et al., 2005c; Phillips et al., 1989). Differences in participation by season, time of day for data collection, fasting time or usual/unusual food consumption were not correlated ($p = 0.18$ to 0.63) with exposure (Table 13).

Biomarkers have been used in population studies to establish prevalence rates and reference ranges, track exposure trends over time and identify subpopulations that may be at-risk for health effects related to chemical exposure (Schmidt, 2006b). For those who are most vulnerable (fetuses, infants and children, pregnant women, elderly, and those who were otherwise ill), a safe level may be zero if the health effects of an exposure may not yet be fully known or realized.

Lead. For 1999 through 2002, NHANES measured blood lead by electrothermal atomic absorption spectrometry (ET-AAS) with Zeeman background correction (Centers for Disease Control and Prevention, National Center for Health Statistics, 2009b, 2010a). For 2003 to 2004, blood lead concentrations were determined by

inductively-coupled plasma dynamic reaction cell mass spectrometry (ICP/DRC-MS) (Centers for Disease Control and Prevention, National Center for Health Statistics, 2009i). Both methods have been validated (Dos Santos, Rodrigues, Silva, Nascimento, 2006; Miller, Paschal, Gunter, Stroud, & D'Angelo, 1987; Parsons & Slavin, 1993; Zhang, Shimbo, Ochi, Eguchi, Watanabe, Moon, & Ikeda, 1997). While changing analytic methods has the potential to introduce an instrumental bias, the results from analyses of whole blood reference materials showed a statistically significant correlation between these two methods (Zhang et al., 1997).

Methylmercury. Total blood mercury is comprised of organic and inorganic species (Cernichiari et al., 1995). Unlike methylmercury, ethyl-, phenyl- and methoxyethyl- mercury are convert rapidly to inorganic mercury (Clarkson & Magos, 2006). Prior research has assumed methylmercury and organic mercury levels in blood to be synonymous (Björnberg et al., 2003; Mahaffey, Clickner, & Jeffries, 2009). This study concurred with this assumption. As a result, true methylmercury values may be overestimated slightly.

For 1999 through 2002, NHANES measured total blood mercury by flow injection mass spectrometry cold vapor atomic absorption with online microwave digestion. Inorganic mercury was measured using stannous chloride as a reductant without utilizing the microwave digestion process (Centers for Disease Control and Prevention, National Center for Health Statistics, 2009b, 2010a). For 2003 to 2004, whole blood mercury concentrations were determined by inductively-coupled dynamic reaction cell plasma mass spectrometry (Centers for Disease Control and Prevention, National Center for Health Statistics, 2009j). The results from analyses of whole

blood reference materials showed a statistically significant correlation between these two methods (Chan et al., 2009). These methods have been validated (Chan et al., 2009; Chen, Paschal, Miller, & Morrow, 1998; Tiezheng & Baasner, 1993).

Polychlorinated Biphenyls. PCBs are comprised of 209 congeners (Appendix F: Polychlorinated Biphenyl Terminology). Most PCB congener concentrations are highly correlated with each other and with total PCBs (Gladen, Doucet, & Hansen, 2003). Four congeners were selected (118, 138/158, 153, 180) for this study because they are most consistently detected in biological samples among the general population and measured most reliably (Frame, 2001). "In most epidemiological studies, these selected congeners are adequate for estimating total PCB exposure …" (Schantz, Wildholm & Rice, 2003, p. 374). It has been found frequently that three ortho-substituted non-coplanar congeners (PCBs 138/158, 153 and 180) account for 50% on average of total reported PCB congeners (Hansen, 1998) with PCB 153 approximately 25% of total reported PCBs (Koopmans-Esseboom et al., 1994). If present in appreciable concentrations, congener 118 has been included (Korrick et al., 2000; Schantz, Widholm, & Rice, 2003) to provide an improved estimate of total PCBs (M. Longnecker, personal communication, February 4, 2010). PCB153 has been proposed as the sole indicator of total PCB exposure to facilitate comparison with literature data among studies (Hagmar et al., 1998). To approximate total PCB levels, one could multiply PCB 153 levels by four (1/0.25 = 4) (M. Longnecker, personal communication, February 4, 2010). Since NHANES did not provide total PCB levels, the uncertainty associated with using just PCB 153 may be as high as 50% (Longnecker, 2001). As a result, this study did not use PCB153 as a (sole) proxy

measure of total PCB exposure. Grandjean et al., (2001) multiplied the sum of PCB congeners 138/158, 153 and 180 by two (1.0/0.5 = 2). Their study is the only study to have approached estimation of total PCB exposures in this manner. This study has defined PCB exposure as the sum of four congeners (118, 138/158, 153, 180) in accordance with Needham et al. (2005). As a result, true total PCB exposure may be underestimated somewhat.

NHANES measured individual PCB congeners by high-resolution gas chromatography/isotope-dilution high-resolution mass spectrometry using a solid phase extraction electron capture detection method. This method has been validated (Barr et al., 2003; Bernert, Turner, Patterson, & Needham, 2007; DiPietro et al., 1997; Patterson et al., 1994; Van den Berg et al., 1998). NHANES calculated the method detection limit for each analyte by correcting for sample weight and recovery, with recovery of the internal quantitation standard greater than 90% (Centers for Disease Control and Prevention, National Center for Health Statistics, 2009j).

Known co-elutents include PCB 158, PCB 160, PCB 163, and PCB 164 for PCB 138; PCB 132 for PCB 153 (Centers for Disease Control and Prevention, National Center for Health Statistics, 2010a) and PCB 123 for PCB 118 (Van den Berg et al., 1995). These co-elutents are rarely found in human samples. As a result, potential interference from these co-elutents in the measurement of these specific PCB congeners was estimated to be minimal (Centers for Disease Control and Prevention, National Center for Health Statistics, 2010a).

Serum PCB levels correlate with serum lipid levels (Longnecker, 2001; Longnecker et al., 2003). As a result, each serum PCB was lipid-adjusted

(Schisterman, Whitcomb, Buck Louis, & Louis, 2005). In this study, lipid-adjusted PCB values were reported as whole-weight nanograms per gram lipid (ng/g lipid).

Limits of Detection. All specimens with a level at or above the upper limit of detection were diluted prior to reanalysis and recalculation (Centers for Disease Control and Prevention, National Center for Health Statistics, 2010a). As a result, readings above the upper limit of detection did not require any further adjustment (Taylor, 1987).

The lower limits of detection were 0.3 μg/dl for lead (1999-2004); 0.137 μg/dl (1999-2004), 0.1 μg/dl (2001-2002) and 0.14 μg/dl (2003-2004) for total mercury; and 0.446 μg/dl (1999-2000), 0.396 μg/dl (2001-2002) and 0.446 μg/dl (2003-2004) for inorganic mercury. Lower limits of detection for PCB congeners varied as each sample had its own limit. The larger an individual sample volume, the lower the detection limit. NHANES documented all PCB values below the limit of detection.

Imputation of Values. Based on the variance for the analysis of samples, the analytical lower limit of detection is defined as the lowest level at which a measurement had a 95% probability of being greater than zero (Taylor, 1987). NHANES' lower limits of detection were defined as three times the standard deviation of ten repeat measurements of a sample's lowest concentration or that measurement determined to be statistically different from a sample blank, depending upon the analytical methodology (Centers for Disease Control and Prevention, National Center for Health Statistics, n.d.a). Methodological uncertainty is high closest to these limits of detection (Taylor, 1987). This definition is associated with a one percent risk of reporting false negatives/type II error – how much analyte might be present but not

detectable (Greizerstein, Gigliotti, Vena, Freudenheim, & Kostyniak, 1997). To ignore non-detectables would overestimate the mean. To set non-detectables to zero would underestimate it. While there are a number of methods available to provide a more accurate estimation of the mean and standard deviation, each has limitations. Hald's method (1952) cannot be used when the limit of detection (i.e., point of truncation) is not a known constant or when more than 50% data are non-detectable. Additionally, this method has been deemed too cumbersome and complex to be practical (Hornung & Reed, 1990). The Nehls and Ackland method (1973) assigns all non-detectables to one-half the lower limit of detection (LoD/2) with the assumptions that the true concentration lies between zero and one. The data below the detection limit follow a uniform distribution in the shape of a rectangle. "But when the proportion of non-detectables is such that the limit of detection is not greater than the mode, the general shape of the left side of a lognormal distribution is better approximated by a right triangle" (Hornung & Reed 1990, p. 48). Their method divides the lower limit of detection by the square root of two (LoD/$\sqrt{2}$). The bias of estimating the geometric mean in this manner has been estimated to be 0.05% (Table 14).

For this study, lead, total mercury and inorganic mercury met the criteria for using the Hornung and Reed (1990) method to address sample values less than the lower detection limit. Each lower limit of detection was less than the mode and each geometric standard deviation was less than 3.0 (Table 15). Based on the percentage of non-detectables that is less than 10% for lead and total mercury, and greater than 60% for inorganic mercury, the Hornung and Reed method overestimated the true

geometric mean 0.12% - 2.9% for lead and total mercury, and underestimated it more than 6.1% for inorganic mercury (Table 14).

NHANES used the Hornung and Reed (1990) method (LoD/√2) to impute values less than the lower detection limit after correcting for sample weight and analyte recovery (Centers for Disease Control and Prevention, National Center for Health Statistics, 2009b, 2009j, 2010a). The Centers for Disease Control and Prevention, National Center for Environmental Health (2009) found these imputations made little difference in geometric mean estimates. This study's analyses of the data as described above concurred with this finding. As a result, this study made no additional adjustments for values less than the lower limit of detection.

Derivation of Methylmercury Values. Due to limits of existing analytic methodology, methylmercury could not be measured directly. As a result, each methylmercury (MeHg) level was derived by subtracting inorganic mercury (IHg) from total mercury (THg) (Cernichiari et al., 1995). Using this calculation, a negative value for methylmercury was observed in 18.4% cases among all NHANES participants and 15.1% cases among childbearing-aged female participants (Table 16). Mahaffey, Clickner and Bodurow (2004, p. 565). They attributed these negative values to differences in detection limits and recoded all methylmercury values less than zero equal to one-half inorganic mercury's detection limit. It was decided that this Mahaffey et al. method would be used in this study. This imputation may have underestimated total mercury and methymercury levels only slightly.

Logarithmic Transformation of Xenobiotic Values. Prior to data analysis, xenobiotic levels were transformed logarithmically to approximate normal

distribution. A geometric mean provides a better estimate of central tendency for these data which are distributed with a long tail at the upper end, a phenomenon found commonly among environmental chemical biomarkers (Centers for Disease Control and Prevention, National Center for Environmental Health, 2009). For this reason, logarithmic transformation is routinely performed in environmental health studies (Bellinger et al., 1991; Grandjean et al., 1992b; Park et al., 2007 and others). The geometric mean dampens the effect of higher values which would bias an arithmetic mean. The geometric mean represents a 50/50 distribution. Since values at +3 SD are of greatest concern to public health, all values were included in the analyses. Histograms of logarithmically-transformed detectable values demonstrated a normal (Gaussian) distribution (Figures 3 through 23). Since methylmercury was derived, values equal to zero presented a challenge to log transformation. As a result, a value of one was added to all methylmercury values prior to log transformation. Frequency distributions for lead, methylmercury and sum of PCBs prior to logarithmic transformation (Figures 24 through 26) were asymmetric (positively skewed). After logarithmic transformation, they approximated normal distributions (Figures 27 through 29).

Measurement of Independent Variables

Vulnerability was measured by susceptibility-related attributes, exposure-related attributes, socioeconomic factors and race-ethnicity (Table 17).

Susceptibility-Related Attributes.

Reproductive Status.

Pregnancy. A urine test for pregnancy was performed on all female participants aged 12 to 59 years and menstruating females aged 8 to 11 years. From 1999 to 2000, these data were released for females aged 18 to 59 only. As a result, the total number of pregnancies for these two survey years is underestimated for 16 and 17 year-olds. If a female did not report having regular periods in past 12 months, NHANES asked if she thought she was pregnant and, if yes, asked her the month of pregnancy (Centers for Disease Control and Prevention, National Center for Health Statistics, 2009c). For this study, these self-reports of pregnancy were compared to their respective pregnancy tests. All positive pregnancy tests were coded as pregnant and all negative pregnancy tests were coded as not pregnant. If the pregnancy test was missing and the trimester of pregnancy was reported as second or third, the response was recoded as pregnant. If the pregnancy test was missing and the trimester of pregnancy was reported as first, it was coded as missing as the pregnancy could not be confirmed.

Parity and Gravidity. Female participants were asked if they were ever pregnant: miscarriages, stillbirths, tubal pregnancies, abortions and live births. Initially for this study, all respondents who were currently pregnant were included in "ever pregnant." Since gravidity is subject to recall bias (Hassan, 2006), "ever pregnant" was deleted in

the final analysis in favor of keeping "ever" versus "never" live birth. A separate variable for current pregnancy was created.

Lactation. If females did not report having regular periods in past 12 months, NHANES asked if they were breastfeeding. Additionally, survey participants were asked if they ever breastfed any of their children for at least one month (Centers for Disease Control and Prevention, National Center for Health Statistics, 2009c). Currently, there is no consensus regarding the operational definition for breastfeeding (Thulier, 2010). For this study, "ever breastfed" was comprised of females 16 to 49 who breastfed one or more children for at least one month and those currently breastfeeding. Those who breastfed for less than one month were included in "never breastfed." This could have introduced a misclassification bias.

Age. Since NHANES oversampled 16 to 19 year-olds and restricted access to information about their alcohol consumption and tobacco use, females aged 16 years to 19 years (192 to 239 months) were considered one cohort while the other females were grouped by decade: 20 years to 29 years (240 months to 359 months), 30 years to 39 years (360 months to 479 months) and 40 years to 49 years (480 months to 599 months). Age in months was reported at time of examination.

Health Status. There are perceptual, biomedical, functional and adaptive aspects to health assessments (Sadana, Mathers, Lopez, Murray, & Iburg, 2001). For this study, health status was measured by perceived health status, the presence of co-morbidities, serum indicators of iron deficiency, and healthcare access and use.

Perceived Health Status. Self-rated health has been used globally to measure health perception (Gold, Franks, & Erickson, 1996). It has been shown to be a strong

predictor of mortality risk (McGee, Liao, Cao, & Cooper, 1999; Sadana, Mathers, Lopez, Murray, & Iburg, 2001). NHANES participants were asked "Would you say your health in general is (excellent, very good, good, fair or poor)?" Self-reported health perception is subject to responder bias (Reindl-Benjamins, Hummer, Eberstein, & Nam, 2004). Ethnic differences in response to this question have been demonstrated when the above five-point scale is used (Dunn, 2002; Kandula, Lauderdale, & Baker, 2007; Lee, 2000; Robine, Jagger, & Egidi, 2000). Therefore, to minimize this responder bias, a dichotomous response (excellent-very good-good or fair-poor) was used in this study.

Co-Morbidities. The Charleson Co-Morbidity Index (Charlson, Pompei, Ales, & MacKenzie, 1987) has been used widely in longitudinal clinical trials to predict 12-month life expectancy of an individual based on the relative risk of death for each disease. This index has demonstrated predictive validity for survival and treatment-related complications (Abdullah & Al-Salamah, 2009; Charlson et al., 2008; Charlson, Pompei, Ales, & MacKenzie, 1987; de Groot, Beckerman, Lankhorst, & Bouter, 2003; Wang et al., 2009). It is a weighted index on a continuous scale that accounts for the total number and relative seriousness of 19 medical conditions with an adjustment for each decade in age over 49 (Hall, Ramachndran, Narayan, Jani, & Vijayakumar, 2004). The Charlson Co-Morbidity Index provided a general description of each disease condition, developed from clinical definitions in Deyo, Cherkin and Ciol's (1992) adaptation for International Classification of Diseases diagnosis and procedure codes (Charlson et al., 2008).

All NHANES participants were asked to self-report on a broad range of diagnosed medical conditions: “Has a doctor or other health professional ever told you that you have (medical condition)?” “Do you still have (medical condition)?” and “During the past (specified time period), have you been on treatment for (medical condition)?” These general questions were linked with more disease-specific questions (Table 18). The specificity (99%) and sensitivity (78 to 90%) of self-reported disease prevalence has been validated (Oksanen et al., 2010). Disease burden may have been underestimated slightly in this study because these questions addressed only diagnosed - not undiagnosed - medical conditions.

NHANES did not address all medical conditions included in the Charlson Co-Morbidity Index. Specifically, NHANES excluded connective tissue diseases, hemiplegia, paraplegia, peripheral vascular disease and dementia. Questions relating to ulcer disease were asked of 1999 to 2000 participants only so 2001 to 2004 participants were coded as negative responses and which may have introduced a misclassification bias.

The Charlson Co-Morbidity Index defined tumor as “a solid tumor without documented metastases but initially treated in the prior five years” (Charlson et al., 1987, p. 383); this was differentiated from metastatic cancers. For this study, metastatic cancer was defined as reporting two or more cancers that were different from the primary site and more systemic in nature (e.g., nervous system or lungs). NHANES excluded individuals if they received chemotherapy within four weeks of the survey. This exclusion may have underestimated cancer and metastatic cancer rates somewhat.

The kidney questionnaire was asked only of participants 20 and older. The Charlson Co-Morbidity Index defined moderate renal insufficiency as serum creatinine greater than 3 mg/dl (Charlson et al, 1987, p. 382). Therefore, this laboratory cut-off value was used to identify or confirm moderate-severe renal disease in the absence of questions regarding diagnosis of kidney failure and use of kidney dialysis. Serum creatinine is determined by using the Jaffe Reaction method (Jaffe, 1886). This method has been validated (Chromý, Rozkosná, & Sedlák, 2008). Values less than 0.6 mg/dl were reported as less than 0.1 mg/dl. Values greater than 25 mg/dl were diluted prior to reanalysis (Centers for Disease Control and Prevention, National Center for Health Statistics, 2007a).

Acquired Immune Deficiency Syndrome (AIDS) has been defined as Human Immunodeficiency Virus (HIV) positive with a CD4 count less than or equal to 200 cells per cubic millimeter (Hanson, Chu, Farizo, & Ward, 1995; U.S. Department of Health and Human Services, n.d.). This laboratory-based definition was used to identify participants with AIDS-related complex in this study. Positive HIV status was confirmed if the enzyme immunoassay (EIA) was repeatedly positive and the Western Blot test was positive. If EIA was repeatedly negative, then the test for HIV was considered negative. If EIA was positive or indeterminate and the Western Blot test was indeterminate, then the HIV test was indeterminate. CD4 counts were performed for HIV-positive persons with available blood- and age-matched controls only. These tests are described elsewhere (Centers for Disease Control and Prevention, National Center for Health Statistics, 2009j).

To distinguish mild liver disease from moderate or severe liver disease, serum albumin and total bilirubin levels were compared to those classifications established in accordance with the Child-Turcotte-Pugh Score (Child & Turcotte, 1964; Pugh, Murray-Lyon, Dawson, Pietroni, & Williams, 1973). Similar to the Charlson Co-Morbidity Index, the Child-Turcotte-Pugh Score was used originally to predict one- and two-year mortality outcomes among hospitalized individuals with chronic liver disease based on key laboratory values (serum albumin, total bilirubin, prothrombin time) and the presence or severity of ascites and/or hepatic encephalopathy. In this dissertation, liver disease was determined by serum albumin and total bilirubin only. Since NHANES excluded individuals with hemophilia as well as those who were institutionalized (hospitalized), it is unlikely that survey participants were individuals with abnormally high prothrombin times and/or clinically apparent severe liver disease (ascites and/or hepatic encephalopathy). These exclusions may have underestimated Child-Turcotte-Pugh scores minimally.

Serum albumin was determined by a bichromatic digital endpoint method and total bilirubin by a timed-endpoint diazo method using a Beckman Synchron© LX-20. According to the Centers for Disease Control and Prevention, National Center for Health Statistics (2009j), linearity data verified reportable ranges for albumin (1.0 - 7.0 μg/dl) and total bilirubin (0.1 - 30.0 mg/dl). As a result, nondetectables were not relevant and a formal limit of detection study was unnecessary.

While the Charlson Co-Morbidity Index (CCMI) was developed to assess an individual's burden of concurrent chronic disease, the CCMI is not all-inclusive and its predictive value is limited within a generally healthy population (Ware, Brook,

Davies, & Lohr, 1981). However, where the prevalence of co-morbidity is low, grouping disease conditions in this manner is statistically advantageous through increasing individual cell size.

*Iron Deficiency***.** Iron deficiency was operationalized by two or more of the following abnormal serum values: mean cell volume less than 81 fL, transferin saturation less than 15% and serum ferritin less than 12 µg/L after adjusting for age and sex (Looker, Dallman, Carroll, Gunter, & Johnson, 1997; Mei, Parvanta, Cogswell, Gunter, & Grummer-Strawn, 2003). These three biochemical indicators for iron deficiency are more sensitive and specific than either hemoglobin or erythrocyte protoporphyrin alone (Mei et al., 2003; Ross, 2002). Iron deficiency decreases red blood cell size resulting in lower mean cell volume. All serum iron is bound to transferin. Transferin saturation is calculated as a percentage by dividing serum iron by serum total iron binding capacity. Serum ferritin reflects iron stores (Bryant, Hopkins, Arceo, & Leitman, 2009). Iron deficiency among pregnant women is diagnosed using these same indicators (Blackburn, 2007; Burst, 2003; Gabbe, Niebyl, & Simpson, 2007). Therefore, these parametrics for iron deficiency were applied equally to pregnant and non-pregnant female participants in this study.

Mean cell volume was determined by a National Committee for Clinical Laboratory Standards' procedure (National Committee for Clinical Laboratory Standards, 1985). Serum iron and serum total iron binding capacity (TIBC) were determined by a modified automated 25-colorimetric method for 1999 to 2002 samples (Ramsey, 1957; Giovaniello, Bendetto, Palmer, & Peters, 1968) and by a timed-endpoint method for 2003 to 2004 samples. According to the Centers for

Disease Control and Prevention, National Center for Health Statistics (2009j), linearity data verified reportable ranges (serum iron: 5 to 500 μg/dl and TIBC: 0 to 500 μg/dl). As a result, nondetectables were not relevant and a formal limit of detection study was unnecessary.

For 1999 through 2003, serum ferritin was determined by a single incubation two-site immunoradiometric assay based on the general principles of assays as described by Addison et al., (1972) and Miles (1977) and modified by Jeong, Blackmore, and Lewin (1981). For 2004, serum ferritin was determined by immunoturbidimetry. Both methods have been validated (Lipschitz, Skikne, & Thompson, 1981; Dupuy, 2009).

Anemia appears only when iron deficiency is chronic and severe (Morón & Viteri, 2009). NHANES asked "During the past three months, have you been on treatment for anemia, sometimes called tired blood or low blood?" Affirmative answers to this question were noted. For this study, a new variable was created that combine iron deficiency and anemia treatment (Table 19).

Healthcare Access. The ability to recover and/or maintain health is closely tied to affordability and continuity of healthcare and social services (Lee, 2000). "Absent or inadequate healthcare deters preventive healthcare practices" (Sampselle, 2007, p. 222). Affordability of healthcare, continuity of healthcare and adequate healthcare are intertwined (Lu, Samuels, & Wilson, 2004). Health insurance is a major determinant of access to healthcare (Heck & Parker, 2002). Regular healthcare has been shown to be a key factor in promoting positive health outcomes (Gorman & Braverman, 2008). A regular source of healthcare has been associated with improved health (Shi &

Stevens, 2005). However, episodic care provided by a hospital emergency room or outpatient department is an inadequate source of healthcare (Mayberry, Mili, & Ofili, 2000). To assess an individual's access to healthcare, four questions were selected: "Do you have health insurance?" "If yes, what type?" "Is there a place that you usually go to when you are sick or need advice about your health?" "What kind of place do you go to most often?" (Centers for Disease Control and Prevention, National Center for Health Statistics, n.d.b).

***Nutritional Status*.** Three variables have been used extensively to assess nutritional status: food insecurity and body mass index (Morón & Viteri, 2009). Other validated measures include changes in dietary intake of fat, protein and micronutrients (Detsky et al., 1987; Kondrup, Rasmussen, Hamberg, Stanga, & Ad Hoc ESPEN Working Group, 2003).

Household Food Security. To assess household food security, NHANES used the U.S. Food Security and Hunger Survey Module (U.S. FSSM) a/k/a Core Food Security Measure (CFSM) (Bickel, Nord, Price, Hamilton, & Cook, 2000). This 18-item questionnaire asked about food security conditions experienced by adults and children within a given household over the prior 30 days and included questions regarding the use of food stamps as well as participation in the federal Special Supplemental Nutrition Program for Women, Infants and Children (WIC) programs in the prior 12 months. Administered at the state level, WIC is a federal program that provides supplemental food, healthcare referrals and nutrition education for low-income pregnant, breastfeeding and non-breastfeeding postpartum women, infants and children (up to age five) who are found to be at nutritional risk. Based on the total

score of affirmative answers, households were classified into one of four categories: fully food secure, marginally food secure, food insecure without hunger and food insecure with hunger. The concept of food insecurity with hunger was based on the definition of hunger as part of a continuum of food insecurity (National Research Council, 2005). Validity and reliability of this module has been confirmed across family structures and ethnic groups (Derrickson, Fisher, & Anderson, 2000; Gulliford, Nunes, & Rocke, 2006). NHANES assumed those households with more than five times the federal poverty threshold level were food secure, so they did not administer the U.S. FSSM/CFSM to these households. Households that were fully food secure were over-represented in the sample with valid data at the household, adult and child level (Centers for Disease Control and Prevention, National Center for Health Statistics, 2006). In this study, all fully food secure households were coded as such and included in prevalence estimates.

*Body Mass Index***.** Body Mass Index (BMI) is a heuristic measure of body weight based on the proportion of weight-to-height (kg/m^2). It is significantly correlated with total body fat content. Classifications include: underweight (less than 18.5 kg/m^2), normal (18.5 to 24.9 kg/m^2), overweight (25.0 to 29.9 kg/m^2), obese I (30.0 to 34.9 kg/m^2), obese II (35.0 to 39.9 kg/m^2) and extremely obese III (40.0 kg/m^2 or greater). These classifications have been validated. There is a significant increase in mortality risk where BMI is greater than or equal to 30 kg/m^2 (U.S. Department of Health, Education & Welfare, National Institutes of Health, National Heart, Lung and Blood Institute, 1998). As a result, this study dichotomized these BMI data at less than 30 kg/m^2 versus 30 kg/m^2 or more.

Dietary Intake of Select Nutrients. Study participants were randomly assigned to interview method (in-person or telephone) and time of day (morning, afternoon or evening). This study included only those who were interviewed face-to-face, completed the dietary survey and rated reliable by CDC trained, bilingual dietary interviewers. Dietary interviewers were required to have a B.S. degree in food and nutrition or home economics with at least ten credit hours in food and nutrition. All interviewers completed an intensive two-week training course followed by a week of supervised, practice interviewing. Minimum criteria for reliability included providing food descriptions more than 75% of the time, food amounts more than 85% of the time and knowing at least one food item per meal (Centers for Disease Control and Prevention, National Center for Health Statistics, 2003; Centers for Disease Control and Prevention, National Center for Health Statistics, 2007b). NHANES used a four-pass method (quick list, time-occasion-place, food details and final review) in which survey participants were given four opportunities to think through what they ate and drank over the prior 24 hours. This method has been deemed the most reliable dietary assessment method (Nelson et al., 2009). NHANES calculated these nutrient intakes from the 24-hr dietary recall using the University of Texas Food Intake Analysis System (FIAS©) in conjunction with the U.S. Department of Agriculture Survey Nutrient Database (Centers for Disease Control and Prevention, National Center for Health Statistics, 2003).

In this study, fat and protein 24-hr intakes were converted from grams to calories then divided by total calories consumed. This value was compared to its acceptable macronutrient distribution range (AMDR), adjusted for sex, age, pregnancy and

lactation (Institute of Medicine, 2005). This study dichotomized the ratio of fat intake to AMDR as recommended or less (0.00 to 0.35) versus more than recommended (greater than 0.35) and the ratio of protein intake to AMDR as less than recommended (0.00 to less than 0.10) versus recommended or more (0.10 or greater) (Table 20).

In this study, 24-hr intakes of iron, calcium and selenium were each divided by their respective recommended daily allowance (RDA) which had been adjusted for sex, age, pregnancy and lactation (Freedman, Guenther, Dodd, Krebs-Smith, & Midthune, 2010; Institute of Medicine, 2005). This study dichotomized these values as less than 1.0 versus 1.0 or greater. Because intakes did not include iron, calcium and selenium obtained from other sources such as dietary supplements, antacids, medications, plain drinking water, salt and seasonings added to foods at the table, true total intakes of these micronutrients may have been underestimated somewhat.

Exposure-Related Attributes.

Acculturation. Language spoken at home has been found to be the strongest predictor of acculturation. Residency as percent of lifetime and generational status have high internal consistency and strong correlation among existing acculturation scales (Alegria, 2009). Other proxy variables for acculturation have included country of birth, age at immigration or generation from immigration, length of time in (new) country, and (new) language proficiency (Alegria, 2009; Anderson et al., 1993; Carter-Pokras & Bethune, 2009; Felix-Ortiz, Newcomb, & Myers, 1994; Lee, Nguyen, & Tsui, 2009; Marin, Sabogal, Marin, Otero-Sabogal, & Paerez-Stable, 1987; Thomson & Hoffman-Goetz, 2009). All NHANES participants were asked their country of birth, the length of time in U.S, citizenship status and language spoken at home.

Those participants reporting their ethnicity as Mexican American or Other Hispanic were asked five additional questions pertaining to language use preferences, each with five answer choices: only Spanish, Spanish better than English, both equally, English better than Spanish, and only English. This eight-item acculturation questionnaire has demonstrated high internal reliability (Carter-Pokras & Bethune, 2009). It was used initially in the Hispanic Health and Nutrition Examination Survey (HHANES), a national probability sample conducted from 1982 to 1984 of 16,000 Mexican American, Puerto Rican and Cuban-Americans (Centers for Disease Control and Prevention, National Center for Health Statistics, 2009f). For this study, birthplace was dichotomized as inside or outside the United States. The length of time in U.S. (less than five years versus five years or more) and language spoken at home (English versus non-English) were dichotomized as well. For Hispanic participants, answer choices for language spoken at home were recoded as English (only English, English more than Spanish, both equally) or non-English (only Spanish, Spanish more than English). Data were not coded in such a manner to allow residency as percent of lifetime or age at immigration to be calculated.

Dietary Consumption. Following the dietary 24-hr recall, a short questionnaire was administered whereby study participants estimated their fish and shellfish consumption during the past 30 days (Table 21) and intake of plain water during the previous 24-hr time period (Table 20).

Fish and Shellfish Consumption. Each study participant was asked, "Please look at this list of fish and shellfish." "During the past 30 days, did you eat any types of fish and/or shellfish listed on this card? Include any foods that had fish and/or

shellfish in them such as sandwiches, soups, or salads" (Centers for Disease Control and Prevention, National Center for Health Statistics, 2003).

Smith (1991) found that those experimental subjects who were provided with food group cues in the form of a list, reported significantly more food items than those with chronologically-based cues such as breakfast, lunch and dinner. As to the retention interval effect, omissions of food items were 50% after two weeks and 30% after four weeks and false recalls 30% after two weeks and 40% after four weeks when compared to food diaries (Smith, 1991). In a 24-hr dietary recall, Karvetti and Knuts (1985) found omissions of fish consumption were the lowest of all foods consumed (4%) with false recalls of fish consumption 7% when compared to observed food and nutrient intake. Additionally, these researchers found women to be somewhat more accurate than men. Generally, consistent food consumption patterns result in more frequent dietary recall. In the United States, approximately 9% of women consume fish at least once a week (Mahaffey et al., 2004).

Since this study did not examine exposure outcomes, actual amounts of fish and/or shellfish consumed were not considered. To minimize recall bias, separate variables for fish and shellfish consumption were each dichotomized as "ever" versus "never" eaten fish and/or shellfish in past 30 days. A composite variable for total seafood consumption was created as well.

Tap Water Consumption. Tap water was defined as plain and filtered tap water and water from a drinking fountain (Centers for Disease Control and Prevention, National Center for Health Statistics, 2003). Tap water potentially represents a fraction of total dietary water and moisture intake. NHANES asked study participants,

"In the prior 24-hr, how much of the plain water you drank was home tap water?" Tap water from residences represents a fraction of total tap water intake if an individual works, attends school, and/or eats outside the home (Shimokura, Savitz, & Symanski, 1998). It was decided to use 2,000 ml as the cut point for 24-hr tap water consumption as it represented 80% of RDA total water intake adjusted for gender and age (Institute of Medicine, 2005). Selection of this relatively high cutpoint may have underestimated exposures. Based on initial analyses of this study, use of residential water treatment systems may have overestimated exposures related to tap water intake as much as 11%.

Alcohol Consumption. Alcohol consumption was defined as a drink of one ounce (1 oz.) liquor such as whiskey or gin, twelve ounces (12 oz.) beer, and four ounces (4 oz.) wine, wine coolers or any other type of alcoholic beverage (Centers for Disease Control and Prevention, National Center for Health Statistics, 2003). Questions on alcohol consumption revolved around quantity and frequency within specified time intervals that is, lifetime, prior 12 months and prior 30 days. The general reliability, validity and utility of these measures have been supported (Del Boca & Darkes, 2003).

"Never" drinkers were defined as those who responded "no" to the question, "In your entire life, have you had at least twelve drinks of any type of alcoholic beverage?" Seldom drinkers were those who responded "yes" to this question but "no" to a second question, "In any one year, have you had at least twelve drinks of any type of alcoholic beverage?" Due to small cell size, "never" and "seldom" drinkers were grouped into one category in this study.

Women were categorized as drinkers if they responded "yes" to both of these questions and had at least one drink of alcohol on at least one day during the past 30 days. Those who responded "yes" to either of the following questions were considered "heavy" drinkers, "In the past 12 months, on how many days per week/month/year did you have five or more drinks of any alcoholic beverage?" and/or "Was there ever a time or times in your life when you drank five or more drinks of any kind of alcoholic beverage almost every day?" (Centers for Disease Control and Prevention, National Center for Health Statistics, 2003; Naimi et al., 2003).

For those women younger than 20, the alcohol consumption questionnaire was self-administered using audio computer-assisted technology to minimize response bias (Centers for Disease Control and Prevention, National Center for Health Statistics, 2003). Since these data were restricted, it was decided to code 16 to 19 year-old participants as "never or seldom" drinkers. This was in accordance with state laws that prohibit underage drinking. All laws were in effect prior to 1999. Although females tend to drink less often and less per occasion than their male counterparts (Zhong & Schwartz, 2010), this recoding may have underestimated true prevalence of alcohol consumption among these young women. Fryar, Merino, Hirsch, and Porter (2009) estimated as much as 18.5% females aged 16 to 17 are binge (heavy) drinkers.

Tobacco Use. Tobacco products included cigarettes, pipes, cigars, snuff, chaw and nicotine patches, gum or other nicotine products. Similar to alcohol consumption, questions on tobacco use revolved around quantity and frequency within specified time intervals that is, lifetime, per day and the prior 30 days.

For those women younger than 20, tobacco use questions were self-administered using audio computer-assisted technology to minimize expected response bias

(Centers for Disease Control and Prevention, National Center for Health Statistics, 2009i). Since these data were restricted from public access, it was decided to code participants aged 16 to 19 separately as "age-restricted."

"Never" tobacco users were defined as those study participants who responded "no" to a series of questions: "Have you smoked at least 100 cigarettes (20 pipes or 20 cigars or snuff 20 times or chaw 20 times) in your entire life?" (Bondy, Victor, & Diemert, 2009). "Former" tobacco users were those who responded "yes" to the preliminary questions but "no" to a second series of questions: "Do you now smoke cigarettes (pipes, cigars, snuff or chaw)?" Pack-years for former tobacco users could not be calculated from the data provided. While the literature supports the general utility of self-reported tobacco use, true prevalence may be underestimated by 6.2% as compared to serum cotinine levels, depending upon the population (Gorber, Schofield-Hurvitz, Hardt, Levasseur, & Tremblay, 2009).

Cotinine is a xenobiotic metabolite of nicotine. Serum cotinine reflects current use of tobacco products as well as environmental tobacco smoke exposure without differentiating exposure sources. Serum cotinine has a half-life of approximately 15 to 20 hours. Nicotine exposures prior to this time period are not captured by this biomarker. In general, current tobacco users tend to have blood cotinine levels 10 ng/ml or higher while "never" tobacco users exposed to no or very low levels of environmental tobacco smoke (ETS) typically have blood concentrations less than 1 ng/ml. Non-tobacco users with ETS exposures tend to have blood cotinine levels between these two values (U.S. Environmental Protection Agency, 2010b). Benowitz,

Bernert, Caraballo, Holiday, and Wang (2009) have recommended a cut point of 10 ng/ml.

NHANES analyzed serum for cotinine using isotope dilution-high performance liquid chromatography/atmospheric pressure chemical ionization tandem mass spectrometry (ID HPLC-APCI MS/MS) (Centers for Disease Control & Prevention, National Center for Health Statistics, 2009j). This analytical method has been validated (Bernert et al., 1997). Cotinine concentrations were derived from the ratio of native (98% laboratory grade cotinine) to labeled cotinine in the sample by comparisons to a standard curve. All specimens with a level at or above the upper limit of detection were diluted prior to reanalysis and recalculation. The lower limits of detection were 0.05 ng/ml for 1999 to 2001, and 0.15 ng/ml for 2002 to 2004. As explained previously, NHANES imputed values below these detection limits with values equal to the lower limit of detection divided by the square root of two (LoD/$\sqrt{2}$) that is, 0.035 ng/ml for 1999 to 2001 and 0.011 ng/ml for 2002 to 2004 (Centers for Disease Control and Prevention, National Center for Health Statistics, 2006). Using this method, the true geometric mean may be overestimated by 3.4 to 11.2% (Figures 30 to 32, Table 14). It should be noted that each lower limit of detection was equal to its mode and two geometric standard deviations were slightly larger than three (Table 22, Figures 33-34). If the Nehls and Ackland method (LoD/2) were used, the true geometric mean would have been underestimated by only 1.8% (Nehls & Ackland, 1973; Table 14).

In this study, self-reported tobacco use was correlated ($p < 0.0000$) to serum cotinine levels. However, information regarding those whose self-reported tobacco

use was age-restricted, 19.5% showed serum cotinine levels 10 ng/ml or higher. As a result, it was decided to use serum cotinine levels in lieu of self-reported tobacco use. Because there were questions about the validity of imputed values, it was decided that instead of using the geometric mean as a cut point for this variable, serum cotinine levels would be categorized as described above. Categorizing allowed for identification of tobacco users among females aged 16 to 19 as well as environmental tobacco smoke exposures among non-tobacco users. Important sources of nicotine exposure for non-tobacco users are the residence and the workplace. For current smokers, ETS may contribute as much as 23% of total nicotine exposure (Piccardo, Stella, & Valerio, 2010). NHANES asked study participants, "Does anyone who lives here smoke cigarettes, cigars, or pipes anywhere inside this home?" "At your current job or business, how many hours per day can you smell the smoke from other people's cigarettes, cigars, and/or pipes?" This study categorized ETS exposures as none, at home or at work, and both at home and at work; missing values were recoded as none. The exclusion of ETS exposures outside of home and work may have underestimated true exposure slightly.

Residential Characterisitcs. Variables pertaining to the built environment included specific housing conditions (e.g., tap water sources, residential water treatment, age and type of residence) and social factors (e.g., resident status, years at current residence and household size for crowding or population density).

Tap Water Sources. For this study, tap water sources were dichotomized into public or municipal versus private or wells with public sources as the referent category. Specific residential tap water treatment systems were dichotomized as well.

These water treatment systems included: Brita® or other pitcher water filters, a ceramic or charcoal filter, water softener, an aerator or reverse osmosis system. However, not all of these water treatment systems filter lead, methylmercury and PCBs. This may have represented a misclassification bias and underestimated exposure. For this variable, the referent category was no residential water treatment.

Age of Residence. In the absence of specific exposure data, residential age was used as a surrogate measure (Jacobs, Wilson, Dixon, Smith, & Evens, 2009; World Health Organization, European Centre for Environment and Health, 2006). In a study of children residing in Jefferson County, Kentucky, Kim, Staley, Curtis and Buchanan (2002) categorized residential age by decade of original construction. NHANES did not categorize age of residence in this manner (prior to 1940, 1940 to 1949, 1950 to 1959, 1960 to 1977, 1978 to 1989 and 1990 and newer). As a result, 1960 and 1978 were used as cut points in two separate variables. These cut points concurred most closely with promulgation of pertinent environmental regulations (Banned Hazardous Products, 1978; Banned Hazardous Substances, 1972, U.S. Environmental Protection Agency, 2009d, 2010a). There are limitations to using these dates. This researcher acknowledges the existence of a time lag between regulatory enactment and actual changes in the field. Additionally, these bans applied only to new housing construction and replacement of existing materials only when repairs or upgrades were made. Mitigation of existing structures is spurious but ongoing.

Type of Residence. For this study, attached and detached houses were grouped together. Since mobile or manufactured homes and trailers are located in distinctly different neighborhoods, they were grouped separately. All other types of residences

comprised the third category which included any missing data. Since NHANES identified individual block segments for sampling and subsequently drew at random a cluster of households from each selected block segment, it is unlikely that those who were homeless were included in the survey.

Resident Status. NHANES asked survey participants, "Is this residence owned, being bought, rented or occupied by some other arrangement by you or someone else in your family?" (Centers for Disease Control and Prevention, National Center for Health Statistics, n.d.b). This variable was categorized as owned (or being bought), rented, and other which included missing or unknown.

Years at Current Residence. Residency was dichotomized at five years in accordance with Dunn's study of self-rated health, mental health and household attributes (Dunn, 2002).

Household Size. The U.S. Census Bureau (2000) calculated household size by dividing the number of persons in households by the number of households. Dunn (2002) operationalized household size by the number of people per number of bedrooms while Pollack, von dem Knesebeck, and Siegrist (2004) relied on the total number of rooms. In this dissertation, it was not possible to create a similar composite variable due to small cell size. Instead, two separate variables were created. For the number of persons per household, there was a dichotomous variable with the cut point equal to the median (four). For the variable "total number of rooms in a residence" there were four categories: one to three, four to six, seven or more, and missing data.

Occupation. Once employment status was established, survey participants were asked about their current and longest-held jobs: "What kind of work were you doing last week?" "What kind of business or industry is this?" "Thinking of all the paid jobs

or businesses you ever had, what kind of work were you doing the longest?" "What kind of business or industry was that?" (Centers for Disease Control and Prevention, National Center for Health Statistics, 2008a). Trained coders grouped these industry and occupational data into 42 occupations and 45 industries using the 2000 U.S. Census Bureau Indexes of Industry and Occupations, North American Industrial Classification System or NAICS (U.S. Census Bureau, 2001a, 2001b, 2003a, 2003b). Due to small cell size, this study condensed these groups to two industry and two occupational categories (Tables 23 and 24). The decision to group sales positions with managerial and professional occupations and service positions with "heavy" industry-related occupations was based upon an assumed similarity in workplace chemical exposures. Data were not collected on those participants who held more than one job in different occupations or industries, thus introducing some opportunity for misclassification.

Total Hours Worked. The U.S. Department of Labor, Bureau of Labor Statistics (2008a) defined full-time work as 35 or more hours, so this value was used as the cut point for the variable "total hours worked in the prior week from all jobs and businesses." Time in current and longest employments was categorized as not working which included not applicable, less than five years, and five or more years. Although job duration may be somewhat age-related, the Bureau of Labor Statistics claims 65% of the baby-boomer generation experienced less than five years with the same employer with women spending 15% more time out of the workforce than men (U.S. Department of Labor, Bureau of Labor Statistics, 2008b).

Socioeconomic Factors. Socioeconomic factors included education, employment, income and marital status. While significant interrelationships exist among these variables, they are not redundant (Winkleby, Jatulis, Frank, & Fortmann, 1992).

***Education*.** Researchers have found that actual years of education do not reflect the potential socioeconomic effects of degree attainment. Not everyone who attends school graduates with a degree (Bauman & Graf, 2003; Frazis, Harrison-Ports, & Stewart, 1995; Kominski, & Siegel, 1995; Zajacova & Hummer, 2009). In NHANES, education was measured in years of schooling up to the twelfth year, then by degree attainment (i.e., high school diploma or equivalent, secondary and post-secondary degrees). Since the greatest disparities in health occur among those without a high school diploma or equivalent (Dube, Asman, Malarcher, & Carabollo, 2009; Kim, 2008; Lynch, 2003), this study chose attainment of high school diploma or equivalent as the cut point for this variable. For those survey participants aged 16 to 18 who were still attending school, years of schooling did not reflect future intent to graduate, thus introducing a potential age bias.

Employment. The U.S. Department of Labor, Bureau of Labor Statistics (2008a) defined employment as "working at least one hour of paid work; 15 hours or more paid work in family enterprise; employed but temporarily absent whether or not paid for the absence; and self-employment." Each person is counted only once, even if the person holds more than one job.

Those survey participants who responded "no" to current employment were asked, "What is the main reason you did not work last week?" (Centers for Disease

Control and Prevention, National Center for Health Statistics, 2008a). The U.S. Department of Labor, Bureau of Labor Statistics (2008a) defined unemployment as "persons available for work who had made specific efforts to find employment or those waiting to be recalled to a job following a layoff." For this study, unemployment was dichotomized as voluntary that is, those who are able to work but choose not to work, and involuntary that is, those who are unable to work for health reasons, cannot find work and/or lost their job. Voluntary unemployment included taking care of the house and/or family, going to school or retirement. Misclassification could have occurred among those survey participants who had stopped looking for a job that is, those who involuntarily retired or who are disabled and wanting to work but were not able to find suitable employment.

Lastly, a composite employment history variable was created: never employed, currently employed, employed in the past but not currently; and employed now and in the past. Missing data was included in "never employed."

Income. In accordance with the U.S. Census Bureau American Population Survey, NHANES defined family as "two or more people related to each other" (Centers for Disease Control and Prevention, National Center for Health Statistics, 2009c). It should be noted while every family is a household, not every household is a family. NHANES provided incremental incomes for households and families as well as a family poverty-to-income ratio. A family poverty-to-income ratio is equal to the family income divided by the federal poverty threshold. Each year, the U. S. Census Bureau establishes income thresholds that vary by family size and age to determine who lives in poverty. These calculations are based on Orshansky's concept of a

"market basket" where a standard budget was defined as "a list of goods and services that a family of a particular size and composition would require for a year to live at some specified level" (Assistant Secretary for Planning and Evaluation, U.S. Department of Health and Human Services, 2010; Fisher, 1997, p. 3). Poverty threshold levels are adjusted annually for inflation using the Consumer Price Index for All Urban Consumers (U.S. Department of Labor, Bureau of Labor Statistics, 2009). The federal poverty threshold has limitations. This threshold represents a level of "biological" subsistence based on minimal nutritional requirements with a fixed ratio for non-nutritional requirements. This threshold is relative to the 20th percentile of family income distribution and increases proportionately. The socially-defined minimum standard of living is not addressed (Hauver, Goodman, & Grainer, 1981; Smeeding, 2009). This federal poverty threshold level includes earned income before taxes but excludes capital gains and non-cash benefits such as food stamps, public housing and Medicaid. As a result, it underestimates total income and measures of total wealth. Additionally, these calculations ignore geographical differences which may overestimate disposable income. Despite their limitations, the federal poverty threshold and the poverty-to-income ratio are used widely as measures of income inequality (Hisnanick & Rogers, 2005) and so, subsequently, they were used in this study.

Other benchmarks of income inequality included relative poverty (60% of annual median income), low-income status (200% of the federal poverty level) and 50% of median income, the criterion used for housing assistance eligibility (U.S. Department of Housing and Urban Development, 2009). Median household and family incomes

are computed annually by the U.S. Census Bureau on the basis of a standard distribution of households and families including those with no income (U.S. Census Bureau, n.d.). For income calculations, this study referenced family median incomes from the U.S. Census Bureau (n.d.) for the first year of each two-year survey (1999 to 2000: $49,628; 2001 to 2002: $51,742; 2003 to 2004: $53,692). In this study, 6% to 11% chose not to report family or household income. It is not known how these refusals affected income-related data (Table 25).

Marital Status. In a study on marriage and women's health by Waldron, Hughes and Brooks (1996), marital status was measured as a dichotomous variable (i.e., married or living with partner versus never married, divorced, widowed or separated) because they found no differences in health among these subcategories of unmarried women aged 24 to 34. However, differences in health were found between never married and divorced or separated women at the five-year follow-up (Waldron, Weiss & Hughes, 1997). This suggested an age bias. For this study, it was decided to keep "never married" separate from "once married" (widowed, separated or divorced). Married or living with a partner were grouped together. Missing data were kept separately.

Race-Ethnicity. The Centers for Disease Control and Prevention, National Center for Health Statistics followed U.S. Office for Management and Budget standards for establishing the minimum number of categories for race and ethnicity (Centers for Disease Control and Prevention, National Center for Health Statistics, 2000). NHANES asked participants to self-identify into one category by ethnicity

(Hispanic versus Non-Hispanic) or race (Non-Hispanic White; Non-Hispanic Black; and Asian, Pacific Islander, Native American or Multi-Racial).

Approximately 58% of the U.S. Hispanic population is from Mexico or of Mexican descent (U.S. Census Bureau, 2000). Mexican Americans are purposely oversampled in NHANES. Mexican Americans were categorically differentiated from all Other Hispanics. For this study, "Mexican Americans" were merged with "Other Hispanics" into an "All Hispanics" category because of the relatively smaller cell size for "Other Hispanics".

The broadly-defined racial group "Other" may underestimate relative risk within its subgroups (Sarnquist, Moix Grieb, & Maldonado, 2009). On the other hand, this heterogeneous grouping of Asian, Pacific Islander, Native American and Multi-Racial individuals has been shown to be at increased relative risk for methylmercury exposure related to fish consumption (Hightower, O'Hare, & Hernandez, 2006).

For those participants who responded initially "don't know", NHANES asked them to choose the one category that *best* represented their ethnicity or race (Centers for Disease Control and Prevention, National Center for Health Statistics, 2000). Since race and ethnicity are social and not biological constructs, there is validity in self-identity. "People are who they say they are" (Kaufman, 1999, p. 103). Despite its imperfections, this categorical race-ethnicity variable was used in this study because it promoted statistical reliability and allowed for data comparability (Buescher, Gizlice, & Jones-Vessey, 2005).

Once all the dependent and independent variables were defined, data processing and analysis commenced.

Data Processing and Analytic Procedures

Phase One. Using SAS© and StatTransfer©, datafiles were downloaded from the NHANES website then organized and unified into one large database prior to identifying this study's population.

Organized Database. NHANES divided each two-year cycle into four sections: demographics, examination, laboratory tests and questionnaires. Each section contained many datafiles comprised of related variables. Documentation on each datafile was reviewed to identify which of them contained variables of interest. Each variable was examined in detail for relevance to this research application including population subset, skip pattern (if/then - go/to), description and range of values. Datafiles (109) for three contiguous two-year cycles (1999 to 2004) were downloaded from http://www.cdc.gov/nchs/nhanes in SAS© transport file format (.xpt) and imported into SAS© statistical software version 9.2 using StatTransfer©. The data files were merged into three separate datasets according to release years (1999 to 2000, 2001 to 2002 and 2003 to 2004), merging individual data by participant identification number (SEQN).

Codebooks were cross-checked for inconsistencies across years with regard to assigned variable names. Wherever the survey question, examination datum or laboratory test was identical across data sets but differed in variable name, the inconsistency was addressed by setting the variable name in the 1999 to 2000 and 2001 to 2002 data sets to that used in 2003 to 2004 data set. Additionally, codebooks were cross-checked for inconsistencies among answer codes. Where the question was the same but the answer codes differed across data sets, these inconsistencies were

corrected by recoding the answers to those used in the 2003 to 2004 dataset. Potential data truncation in variable length fields was avoided by using the longest of the three field lengths. Finally, these three datasets were concatenated into one large database.

Identified Study Population. Using SAS©, all gender- and age- eligible participants were identified for each of the three two-year surveys (1999 to 2004). Those participants who were interviewed, examined and tested were identified using a six-year laboratory subsample weight. Subsequently, age-eligible pregnant participants were identified. After reviewing frequencies by age, race-ethnicity and pregnancy status, it was decided that eligible participants would be required to have all seven blood tests (lead, total mercury, inorganic mercury and lipid-adjusted PCB 118, PCB 138, PCB 153 and PCB 180) and reliable dietary recall to be included in this study.

Phase Two. Using SAS©, dependent and independent variables were identified within the large unified database and prepared for data analyses.

Operationalized Dependent Variables. Lower limits of detection and imputed values were identified for each of the chemicals of interest. Initially, variables were created from detectable values only and transformed logarithmically. Subfiles containing only these values and their corresponding participant identification numbers were exported by StatTransfer© to SPSS© because the graphics interface in this software is more user-friendly and of better quality than SAS© or SUDAAN©. Histograms of log detectable values were created for each two-year survey period to check for normal distribution. In SAS©, descriptive statistics were performed. Results

were compared to Hornung and Reed (1990) criteria for imputing values below the lower level of detection.

Values for lipid-adjusted polychlorinated (PCB) congeners 118, 138/158, 153 and 180 were summed and transformed logarithmically to create a new variable, the sum of lipid-adjusted PCBs. Inorganic mercury (IHg) was subtracted from total mercury (THg) to create a new variable, methylmercury (MeHg). Negative methylmercury values were identified and imputed per Mahaffey et al. (2004). Since some of these derived values were equal to zero, a value of one was added to each data point prior to logarithmic transformation. Frequency distributions were examined before and after logarithmic transformation.

With an infinite number of possible values on the individual level, dichotomous variables were created for lead, methymercury and sum of PCBs with their respective geometric means as cutpoints. Exposure was defined as a xenobiotic blood level at or above the geometric mean. Two different exposure variables were created: one using four categories (0, 1, 2, or 3), the other using two categories (0 or 1 and 2 or more). Both of these operational definitions were conceptually congruent. There were no missing data.

Operationalized Independent Variables. Informed by Sexton, Olden and Johnson's modified environmental health paradigm (1993a) and the literature review, NHANES was scrutinized to identify measures which best represented the concepts. Independent variables of interest included specific susceptibility- and exposure-related attributes, socioeconomic factors and race-ethnicity. These measures were identified, relabeled and recoded as necessary. For example, "don't know" and "refused"

answers were recoded as "missing". Nominal categorical variables were created. Dichotomous variables were created whenever appropriate. Variable frequencies were checked (Table 26) and all 62 independent variables were assessed to assure adequate numbers met the NHANES guidelines for statistical reliability (Centers for Disease Control and Prevention, National Center for Health Statistics, 2006). As noted previously, some variables were dropped due to small cell size. Missing values were addressed on a variable-by-variable basis. Bivariate analyses were conducted on selected pairs of independent variables. Subsequently, some operational definitions were refined (Table 27).

Phase Three. Software instructions were constructed under SAS© and SAS-callable SUDAAN© for unweighted (sample population) and weighted (study population) statistical analyses, respectively. Both software programs are specifically designed for these types of survey data. To avoid biased estimates and overstated statistical significance levels, data were sorted by stratum and masked variance unit or primary sampling unit variables prior to analyses and estimates of sampling errors were calculated by the Taylor series linearization method with replacement per analytical guidelines (Centers for Disease Control and Prevention, National Center for Health Statistics, 2006).

To address this study's research questions, analyses included descriptive and univariate statistics, multivariate statistical and logistic regression modeling, and estimates of risk. Where an unweighted individual cell size was less than 30, the specific value was withheld and replaced with an asterisk per CDC NCHS

recommendations (Centers for Disease Control and Prevention, National Center for Health Statistics, 1993).

Research Questions One and Two. This study population's distribution of xenobiotic levels was analyzed using descriptive statistics including estimates of prevalence of exposure to each of the chemicals of interest. Univariate statistics for each of these xenobiotics were generated before (Tables 28 and 29) and after (Tables 30 and 31) logarithmic transformation. Crude prevalence estimates for each specific environmental chemical were derived by dividing the number of eligible women at or above the geometric mean by the total population of childbearing aged and pregnant women living in the U.S. (Tables 30 and 31). The age-adjusted prevalence was calculated by using standard adjustment techniques. During this phase of the analysis, exposures to combinations and permutations among childbearing-aged females (Table 32 and Figure 35) and pregnant childbearing-aged females (Table 33 and Figure 36) were identified. All estimates were weighted to be nationally representative using SUDAAN©. After examining these data it was decided to drop exposure as outcome in four categories (Tables 34 and 35, Figures 37 and 38) in favor of the two-category exposure variable (Tables 36 and 37, Figures 39 and 40). This dichotomous variable would assure adequate cell sizes and improve statistical reliability. All prevalence rates for childbearing-aged women and pregnant childbearing-aged women (unweighted and weighted) are summarized in Tables 38 through 41.

Research Question Three. Bivariate analyses of 54 independent variables on exposure as outcome with two categories were performed on unweighted and weighted data for childbearing-aged women (Table 42). Unadjusted (unweighted)

variables were examined primarily to determine association. All other analyses were conducted using adjusted (weighted) data. Thirty-three weighted (adjusted) independent variables with non-statistically significant *p* values ($p > 0.20$) were eliminated from further inclusion in this study (Table 43). Of the 21 remaining variables with statistically significant correlations ($p < 0.20$), four variables were dropped based on their low chi-square (χ^2) values (race-ethnicity with five categories, ever pregnant, trimester of pregnancy and seafood meals eaten in past 30 days) in favor of retaining very similar variables with higher chi-square (χ^2) values that is, race-ethnicity with four categories, live births, current pregnancy, fish and shellfish variables, respectively (Table 43).

Based on the results of the aforementioned bivariate analyses, a multivariate logistic regression exposure model was developed by creating a series of nested models and utilizing likelihood ratio testing per Hosmer and Lemeshow (2001). These stepwise regression analyses were not computer-generated. Stepwise logistic regression analysis of exposure as outcome with two categories is detailed in Table 44. The best-fit logistic regression exposure model had 13 variables (Table 45).

A variance inflation factor (VIF) test was performed to identify any collinearity beyond interaction among the independent variables using the best-fit logistic regression exposure model. No collinearity was found (Table 46).

Two-way interactions among the independent variables were assessed for inclusion by comparing nested models that is, the interaction model against another model without interaction using likelihood ratio testing per Hosmer and Lemeshow (2001). Overparameterization occurred after three sequential nested model operations

as the data were too sparse for the number of interactions. Rather than introduce prejudice to the model, efforts were redirected to identify all statistically relevant two-way interactions for future analyses (Table 47). Ten variable pairs could not be tested due to overparameterization. Nineteen pairs were not statistically significant ($p > 0.20$). For the remaining 48 pairs, 40% showed strong statistically significant interactions ($p < 0.001$). Finally, odds ratios (*OR*) were calculated with corresponding 95% confidence intervals (CI) as estimates of risk for each factor among childbearing-aged women using the best-fit exposure model with no interactions (Table 48).

In order to compare the exposure model to each of the models for lead, methylmercury and PCBs, additional data were generated. These data includes:

1. bivariate analyses of independent variables on exposure to each chemical of interest;
2. summaries of chi-square (χ^2) and *p* values;
3. stepwise logistic regression analyses;
4. variance inflation factor tests for collinearity;
5. statistical significance of interactions between independent variables and each chemical; and
6. odds ratios and confidence intervals for each best-fit logistic regression model with no interactions. (Tables 49 through 69.)

Further discussion regarding these data on specific chemicals is outside the scope of this dissertation and its research questions; these data are available for future study.

Ethical Research

All NHANES protocols were approved by the Centers for Disease Control and Prevention, National Center for Health Statistics Research Ethics Review Board and therefore assumed to be in compliance with federal regulations (Centers for Disease Control and Prevention, National Center for Health Statistics, 2010c; Protection of Human Subjects, 1991; U.S. Department of Health, Education & Welfare, National Institutes of Health, Office of Human Subjects Research, 1979, 2005).

Informed Consent of Original Survey Participants. Participants signed consent forms before the interview and the physical examination. If a child was capable, each assented to participation. Regardless of age, guardians consented to each child's participation. Participation was voluntary and an individual could withdraw at any time. Not all participants completed all survey components. Participants received compensation for their time and child or elder care if necessary as well as transportation to and from the Mobile Examination Center (Centers for Disease Control and Prevention, National Center for Health Statistics, 2010b).

Risks and Benefits to Participants in Original Survey.

Physical Risks and Benefits. A portion of survey participants received a thorough physical examination. The physical examinations posed no risk of harm or serious injury to the participants as this portion of the survey was similar to a routine physical examination.

During the laboratory examination, blood and urine samples were obtained from each participant for analysis. As a result, a venipuncture was required during which the participant would have experienced some brief pain and localized discomfort.

Blood draws posed a slight risk of harm (contusion) or injury (*in situ* infection) to the participants. To minimize these risks, venipunctures were performed by experienced healthcare personnel and universal precautions were followed per protocol.

A participant received the benefit of a thorough health assessment. This was beneficial, particularly for those who did not have routine health checkups or adequate healthcare insurance.

Psychological Risks and Benefits. The physical examination and laboratory testing took approximately 3.5 hours per person, depending upon age (details not provided). This may have fatigued participants, especially children. Conceivably, some participant responses may have been impacted by fatigue, introducing a recall bias. However, any recall bias would have minimal impact on study results due to large sample size.

Questions of a "sensitive" nature (e.g., illicit drug use and sexual behaviors) were self-administered in the privacy of participants' homes or in divided rooms within the Medical Examination Center (MEC). For results of tests considered "sensitive", participants were given a password, a toll-free number to call and the date to call. These provisions for privacy would have minimized risk of embarrassment or a reason for a participant to alter answers to questions to avoid embarrassment. Participants received a confidential medical report within 12 to 16 weeks. This equated to the benefit of an assurance of health or early diagnosis of a health problem (Centers for Disease Control and Prevention, National Center for Health Statistics, 2010c).

Financial Risks and Benefits. Participants would have incurred lost wages, if employed, for approximately 3.5 hours plus transportation time to and from MEC.

However, this loss was countered by the benefit of participants receiving compensation (amount not delineated) for their time and child / elder care if necessary as well as transportation to and from the mobile examination center (Centers for Disease Control and Prevention, National Center for Health Statistics, 2010b).

Protections Against Risk for Vulnerable Populations in Original Survey. All participants signed consent forms before the interview and the physical examination. If a child was capable, she assented to participation. Regardless of a child's capability, guardians consented to each child's participation. Participation was voluntary and an individual could withdraw at any time. The original study had the same direct benefit to children, pregnant women and their fetuses as any other study participant. The risk to the fetus and children was minimal. All NHANES protocols were approved by the CDC NCHS Research Ethics Review Board and therefore assumed to be in compliance with Subpart B (Protection of Human Subjects, 2001) and Subpart D (Protection of Human Subjects, 1983).

Confidentiality in Original Survey. Initial interviews were conducted with participants within the privacy of their own homes. Within the MEC, there were divided rooms to assure privacy. Participants received a confidential medical report within 12 to 16 weeks. For results of tests considered "sensitive", participants were given a password, a toll-free number to call and the date to call. Although NHANES assigned an identification number to each survey participant, to maintain anonymity, they did not publicly release details. For example, geography, genetic data and detailed age- or income-specific data which could have identified or assisted in the identification of individual survey participants were not released to the public. To

maintain confidentiality, NHANES used masked variance units, a collection of secondary sampling units aggregated into groups for the purpose of variance estimation (Centers for Disease Control and Prevention, National Center for Health Statistics, 2010b). NHANES de-identified all data to preserve confidentiality and protect its survey participants. For purposes of this study, HIV/AIDS-related information was incorporated into a more comprehensive variable (co-morbidities). Since the cell size was small, the specific number of cases was not released. This researcher did not add to the original dataset.

Dissertation Data Sharing and Release. This dissertation research used de-identified data with no access to personal identifiers. The database was already publicly and freely available. As a result, there was no need to request authorization for use and disclosure of protected health information (PHI). However, data were stored on a laptop computer as well as multiple 2-GB and one 16-GB flash memory drives. This researcher had continual data access. Faculty had access to the data if and when access to data was needed, particularly during data analysis. Files were shared electronically or hard copied. Results of the research will be released in a timely manner, completely and as accurately as possible following appropriate peer review. Raw data and analyses will be kept for at least three years.

Dissertation Research Results Sharing Plan. Sharing research results is essential to expanding the body of knowledge in environmental health, public health and nursing. The results of this research will be published as a dissertation in partial fulfillment of the requirements for a Doctorate in Philosophy (PhD) from the University of Rhode Island, College of Nursing and available through ProQuest©.

Subsequently, it is the intent of this researcher to submit for publication a number of articles (two or three minimally) related to this research and research findings to peer-review journals such as Journal of American Public Health Association (APHA), Environmental Health Perspectives (EHP), Journal of Epidemiology, Journal of Association of American Occupational Health Nurses (AAOHN) and/or Professional Safety (ASSE). Additionally, proposals (two minimally) will be submitted to present research findings and related topics at these associations' national, regional and/or state conferences.

Institutional Review Board. This study involved descriptive, univariate and multivariate analyses of existing retrospective data (1999 to 2004) from the National Health and Nutrition Examination Survey (NHANES). These data were publicly available and freely distributed online (http://www.cdc.gov/nchs/nhanes.htm). This researcher did not add to the dataset. The University of Rhode Island (URI) had an approved assurance of compliance on file with the Department of Health and Human Services which covered this research activity (FWA 00003132). Because this research was a secondary data analysis, the dissertation proposal was reviewed by the Chair of the Institutional Review Board and deemed it exempt ("not human subjects research") on December 30, 2009 (Appendix G: University of Rhode Island Institutional Review Board on Human Subjects IRB Action Report). The Rhode Island Department of Pubic Health signed an individual investigator agreement on January 12, 2010 (Appendix H: Rhode Island Department of Health Individual Investigator Agreement).

Chapter Summary

Every aspect involving the methodologies used in this dissertation have been described in detail: the operationalization of all dependent and independent variables, validity and reliability of these measures, analytic procedures and ethical protocols. The next chapter begins with a general description of the study population then addresses the results and limitations for each research question before concluding with a discussion. Additionally, comparisons between the best-fit logistical regression exposure model and each chemical's best-fit logistical regression model are drawn and discussed.

CHAPTER 4

FINDINGS

This chapter begins with a general description of the study population subset regardless of xenobiotic blood levels. These findings are made available for reference only and will not be discussed further (Tables 26, 27, 36 and 37). Subsequent to this general description, the findings for each research question are revealed. After a comparison between the best-fit logistic regression exposure model and the best-fit logistic regression model for each individual chemical, the discussion section concludes with reference to Sexton, Olden and Johnson's modified environmental health paradigm (1993a).

For ease of readability, this study's findings are reported in this chapter with references to weighted (adjusted) data only, rounded to the nearest whole number. Both weighted and unweighted data can be found in the tables referenced in this chapter. "Unweighted" data are raw data collected from NHANES participants. There were two sample (unweighted) populations: all male and female NHANES participants (1999 to 2004) and a subset consisting of 3,173 childbearing-aged female participants who were interviewed, examined, tested for all chemicals of interest and deemed to have reliable dietary recall. This subset of childbearing-aged females included 491 who were pregnant at time of their examination. "Weighted" data is raw data that has been adjusted to represent an entire population. There were two study (weighted) populations: all people living in the U.S. (1999 to 2004) and a subset

consisting of 134,502,033 childbearing-aged females living in the U.S. (1999 to 2004) of whom 4,842,189 were pregnant. Only this study population subset is reported in this dissertation unless otherwise specified.

The reader is reminded that dependent variables appear in tables in a consistent order: lead, methylmercury, then the summed value of four specific lipid-adjusted polychlorinated biphenyl congeners 118, 138/158, 153 and 180. Independent variables appear in tables in a consistent order that correlates with Sexton, Olden and Johnson's (1993a) modified environmental health paradigm (Figure 2).

General Description of the Study Population Subset

Fourteen percent of childbearing-aged women were 16 to 19 years old, 34% were ages 20 to 29, 27% were 30 to 39 years old, and 25% were ages 40 to 49. Seventy-three percent were Non-Hispanic White, 10% Non-Hispanic Black, 6% Mexican-American, 6% Other Hispanics (12% All Hispanics) and 5% were Asian, Pacific Islander, Native American or Multi-Racial (Table 7).

Of these childbearing-aged women, 4% were pregnant at the time of their examination. Eight percent of these pregnant women were 16 to 19 years old, 53% were ages 20 to 29, 35% were 30 to 39 years old, and 4% were ages 40 to 49. Sixty-three percent were Non-Hispanic White, 15% Non-Hispanic Black, 10% Mexican-American, 5% Other Hispanics (15% All Hispanics) and 7% were Asian, Pacific Islander, Native American or Multi-Racial (Table 12).

Susceptibility-Related Attributes.

Reproductive Status. Overall, half of the childbearing-aged women had given birth to one or more live children; 4% were currently pregnant. Thirty-two percent of

childbearing-aged women had breastfed one or more children for at least one month and/or was currently breastfeeding. Breastfeeding was correlated with age ($p < 0.000$).

Health Status. Overall, childbearing-aged women were healthy. Only 8% perceived their health to be fair or poor. Approximately 12% had one or more co-morbidities. Of those with more than one co-morbidity, 25% perceived their health to be fair or poor. Iron deficiency was found in 9% of these women. Approximately 8% of those iron deficient had not been diagnosed or received medical treatment for anemia in the prior three months. Sixteen percent had no health insurance. Fifty-one percent of those with private health insurance used the emergency room or hospital outpatient department as their regular source of healthcare. Of those who did not have health insurance, 40% used the emergency room or hospital outpatient department regularly for their healthcare.

Nutritional Status. Eleven percent of these women were identified as food insecure. Among those found to be food insecure, 18% were obese (i.e., body mass index of 30.0 or more). This percentage of obesity among food insecure women was slightly lower than that for overall obesity (26%). Forty percent of the study population subset exceeded daily fat intake requirements while 12% did not meet daily intake requirements for protein. The percentages of those who failed to meet minimum daily intake requirements for iron, calcium and selenium were 75%, 68% and 16%, respectively. Those women who met or exceeded selenium requirements were somewhat more likely to have eaten seafood (85%) than not (76%) in the previous 30 days.

Exposure-Related Attributes.

Acculturation. Eleven percent of childbearing-aged women were born outside the United States. Of these women, 8% had lived in the U.S. for more than five years and 2% lived in the U.S. for less than five years. Of the three percent of childbearing-aged women who spoke a language other than English at home, 65% of these women had lived in the U.S. five yearsor more and 28% had lived in the U.S. less than five years. There was a statistically significant correlation between language spoken at home and U.S. citizenship ($p < 0.000$).

Dietary Consumption. Seventeen percent of childbearing-aged women did not eat any fish or shellfish meals within the prior 30 days while 43% ate both fish and shellfish in this same time period. There were 25% more fish eaters than shellfish eaters. Only 12% of these women drank 2,000 ml or more of residential tap water in the previous 24 hr. Thirty percent of childbearing-aged women reported no water intake from this source.

Alcohol Consumption. One-third reported drinking at least twelve alcoholic drinks in any one year and at least one drink in the prior 30 days. Another 27% reported drinking five or more drinks of any alcoholic beverage in any one day and/or almost every day within the span of the previous twelve months. Drinking correlated with serum cotinine levels ($p < 0.024$).

Tobacco Use. The majority (74%) of childbearing-aged women had serum cotinine levels lower than 1 ng/dl while a much smaller percentage (22%) had levels greater than 10 ng/dl. Serum cotinine levels correlated significantly with both self-reported tobacco use and reported environmental tobacco smoke exposure ($p < 0.000$).

Twenty percent of women aged 16 to 19 for whom self-reported tobacco use was withheld from public release had serum cotinine levels higher than 10 ng/ml. At this level, they were most likely current smokers.

Residential Characteristics. Residential water treatment systems were more prevalent when sources of tap water were public (83%) than private (16%). Only eleven percent drew their tap water from private sources. Of the women who consumed 2,000 ml or more of tap water, an equal percentage (12%) drew their water from public or private sources. Two-thirds of childbearing-aged women lived in detached or attached housing of which 90% were owned. Only 7% resided in mobile homes or trailers. Thirty-six percent of renters and 41% of those residing in alternate living arrangements did not know the age of their residence. Among those women who knew the age of their residence, 24% of residences were built before 1960 and another 17% were constructed between 1960 and 1978. Renters were more likely to have lived in their current residence less than five years (89%) than home owners (50%). Household size (i.e., the total number of family members) correlated with the number of rooms in the residence ($p < 0.000$). Only 7% of households with four or more persons lived in less than four rooms.

Occupation. Approximately half of working childbearing-aged women held management, professional or sales-related occupations. These women were four times more likely to have held a job for more than five years than those women in the services- and goods-related occupations. Of those who worked longest in the services- and goods-related occupations, 39% had worked more than five years. Women working in this occupational grouping were four times more likely to live at or below

the U.S. poverty threshold level than those who worked in the management, professional and sales-related occupations. Among those who were not working (31%), twice as many lived above the U.S. poverty threshold level (65%) than at or below this level (28%).

Socioeconomic Factors.

Education. One fifth of childbearing-aged women had less than a high school education. The percentage of women with a high school diploma, GED or higher was 86 to 89% within each age cohort except for those aged 16 to 19, some of whom had not yet completed their education. While age was correlated with educational level ($p < 0.000$), there was no statistically significant interaction between education and age in terms of exposure risk. Those women who had less than a high school education were twice more likely to live at or below U.S. poverty threshold level.

Employment. Employment rate was 69%. Forty-one percent worked 35 hours or more per week. Among those who were unemployed, 21% did so voluntarily; 6% had never worked. Women aged 20 and older who had a high school diploma, GED or higher were three times more likely to be employed than unemployed. In contrast, women of the same age who did not have a high school diploma or GED were equally as likely to be employed (56%) as unemployed (44%). Sixty-seven percent of married women were working.

Income. Of the 17% who lived at or below the U.S. poverty threshold level, 43% were aged 20 to 29. Twenty-one percent of 16 to 19 year olds lived at or below this level. Slightly more than half of the childbearing-aged women who lived at or below the U.S. poverty threshold level either had never married or were widowed, divorced

or separated. Eighty-six percent of women who were married or living with a partner lived above this level.

Marital Status. Forty-six percent of childbearing-aged women were married or living with a partner (46%); 40% had never married and 11% were widowed, divorced or separated. Marital status was significantly correlated with age ($p < 0.000$). Twenty-five percent of Non-Hispanic Black women were married or living with a partner as compared to 50% of Non-Hispanic Whites and 40% of Hispanics.

Race-Ethnicity. Non-Hispanic Blacks and Hispanics were 50% less likely to have a high school diploma or GED. Twice as many Non-Hispanic Blacks and more than 7% of Hispanics lived at or below the U.S. poverty threshold than above it. Of those who were unemployed, Non-Hispanic Black women were twice as likely to be involuntarily unemployed as Non-Hispanic White or Hispanic women. Thirty-six percent of Hispanic women were unemployed voluntarily versus 20% Non-Hispanic Whites and 16% Non-Hispanic Blacks. Age was correlated with race-ethnicity ($p < 0.04$). There were no statistically significant differences among racial and ethnic groups with regard to the type of residence but there was a statistically significant difference ($p < 0.006$) among these groups as to whether they owned or rented their home. Missing data on age of residence precluded further analysis.

Findings

Research Question One. What was the prevalence of childbearing-aged and pregnant childbearing-aged women's exposures to each of the following environmental chemicals: lead, methylmercury and polychlorinated biphenyls (PCBs) as measured by chemical-specific (xenobiotic) levels at or above geometric mean in

blood or serum of these women who were living in the United States from 1999 through 2004?

Among childbearing-aged females, the prevalence rates for xenobiotic blood or serum levels at or above the geometric mean were 49 per 100 for lead, 48 per 100 for methymercury, and 67 per 100for PCBs (Table 30). The number of childbearing-aged females above the 99th percentile was approximately 3 million, 2 million and 1.8 million for lead, methylmercury and PCBs, respectively (Table 29). Among those who were pregnant, prevalence rates were 25 per 100, 33 per 100, and 50 per 100 for lead, methylmercury and PCBs, respectively (Table 30). The number of pregnant females above the 99th percentile was approximately 433,000 for lead, 190,000 for methylmercury, and 21,000 for PCBs (Table 29).

Research Question Two. What combinations and permutations of chemical exposures were most common among these childbearing-aged and pregnant childbearing-aged women as evidenced by xenobiotic blood or serum levels at or above the geometric mean?

Approximately 20% – one fifth – of childbearing-aged females had xenobiotic blood levels at or above the geometric mean for all three chemicals. For the 38% of these women who had two xenobiotic blood levels at or above the geometric mean, it was equally likely to be methylmercury and PCBs (44%) or PCBs and lead (43%) as it was lead and methylmercury (14%). Among the 26% of childbearing-aged females having one xenobiotic blood level at or above the geometric mean, it was twice more likely to be PCBs (51%) than either lead (28%) or methylmercury (21%). Sixteen percent of childbearing-aged females had no xenobiotic blood levels at or above the

geometric mean (Table 32, Figure 35.) There was only a 4% difference between those childbearing-aged females with no xenobiotic blood level at or above the geometric mean and those with three xenobiotic blood levels at or above the geometric mean.

Only 6% of pregnant childbearing-aged females had xenobiotic blood levels at or above the geometric mean for all three chemicals. For the 36% of those who had two, it was highly likely that it was methylmercury and PCBs (74%) than either PCBs and lead (18%) or lead and methylmercury (8%). Among the 25% of these women who had one, it was more likely that it was PCBs (42%) or lead (39%) than methylmercury (19%). Thirty-three percent of pregnant childbearing-aged females had none (Table 33, Figure 36.) There was a 27% difference between those pregnant childbearing-aged females with no xenobiotic blood level at or above the geometric mean and those with three xenobiotic blood levels at or above the geometric mean.

Table 32
Combinations and Permutations of Exposures: Number of Childbearing-Aged Females[1] with Xenobiotic Blood Levels At or Above the Geometric Mean (unweighted and weighted data 1999-2004)

	0	1	2	3
Total Number of Chemicals At or Above Geometric Mean				
Frequency $_{unweighted}$	702.00	971.00	1,005.00	495.00
Row Pct.	(22.12%)	(30.60%)	(31.67%)	(15.60%)
Frequency $_{weighted}$	20,889,388.72	35,175,071.94	51,205,786.00	27,231,786.77
Row Pct.	(15.53%)	(26.15%)	(38.07%)	(20.25%)
Total Number of Chemicals At or Above Geometric Mean				
Frequency $_{unweighted}$				
Col. Pct.				
Frequency $_{weighted}$				
Col. Pct.				
	702.00			
	(100.00%)			
All Xenobiotic Blood Levels	20,889,388.72			
Below Geometric Mean	(100.00%)			
		371.00		
		(38.21%)		
Lead Only		9,875,692.09		
At or Above Geometric Mean		(28.08%)		
		229.00		
		(23.58%)		
Methylmercury Only		7,218,046.91		
At or Above Geometric Mean		(20.52%)		
		371.00		
		(38.21%)		
Sum of PCBs Only		18,081,332.94		
At or Above Geometric Mean		(51.40%)		
			191.00	
			(19.00%)	
Lead and Methylmercury			7,027,018.85	
At or Above Geometric Mean			(13.72%)	
			347.00	
			(34.53%)	
Methylmercury and Sum of PCBs			22,339,886.83	
At or Above Geometric Mean			(43.63%)	
			467.00	
			(46.47%)	
Sum of PCBs and Lead			21,838,880.31	
At or Above Geometric Mean			(42.65%)	
				495.00
				(100.00%)
All Xenobiotic Blood Levels				27,231,786.77
At or Above Geometric Mean				(100.00%)

[1] included pregnant childbearing-aged females

[2]where the third xenobiotic is below geometric mean

Figure 35. Number of Childbearing-Aged Females in U.S. with Xenobiotic Blood Levels At or Above the Geometric Mean (weighted data 1999-2004)

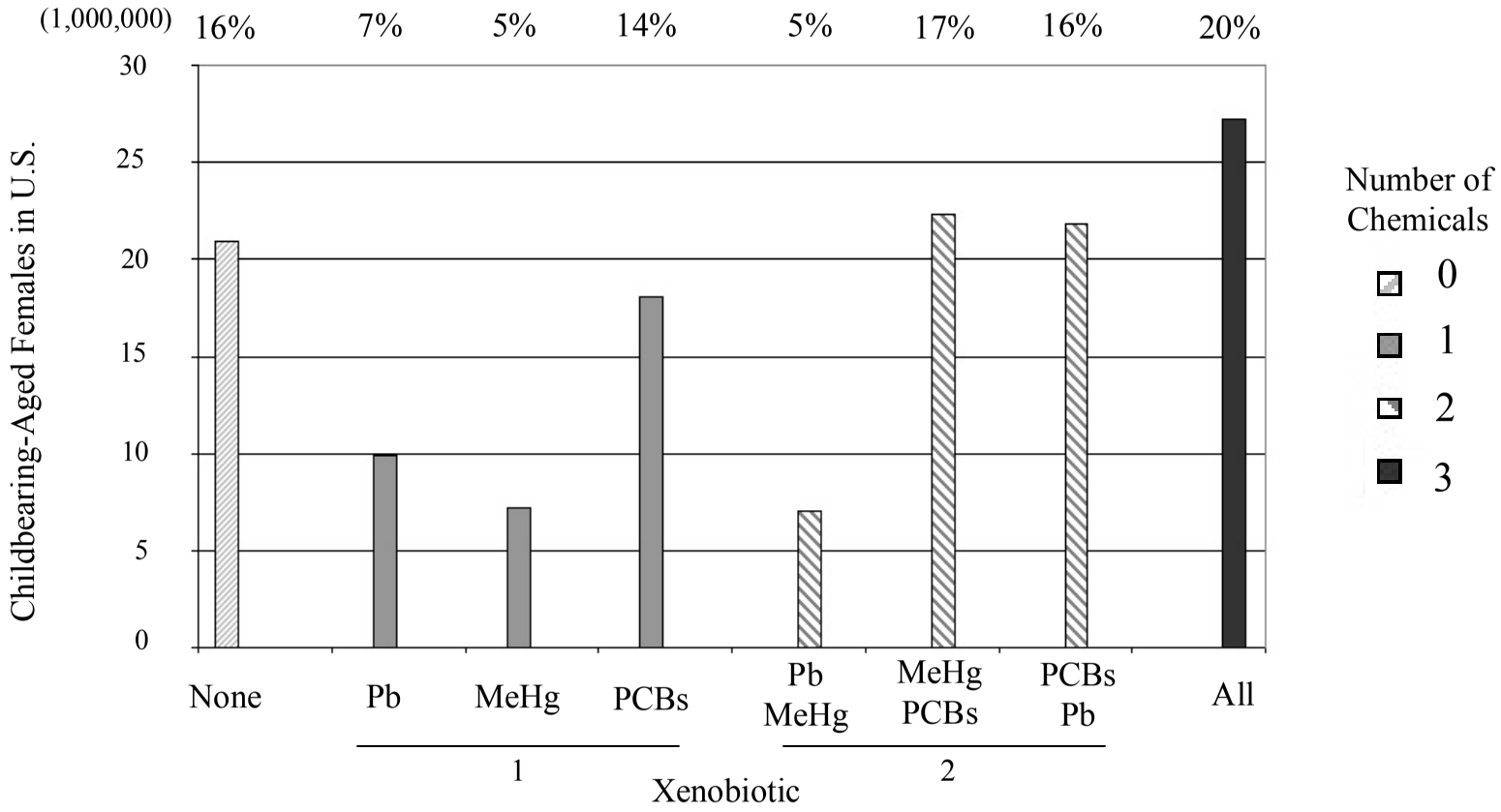
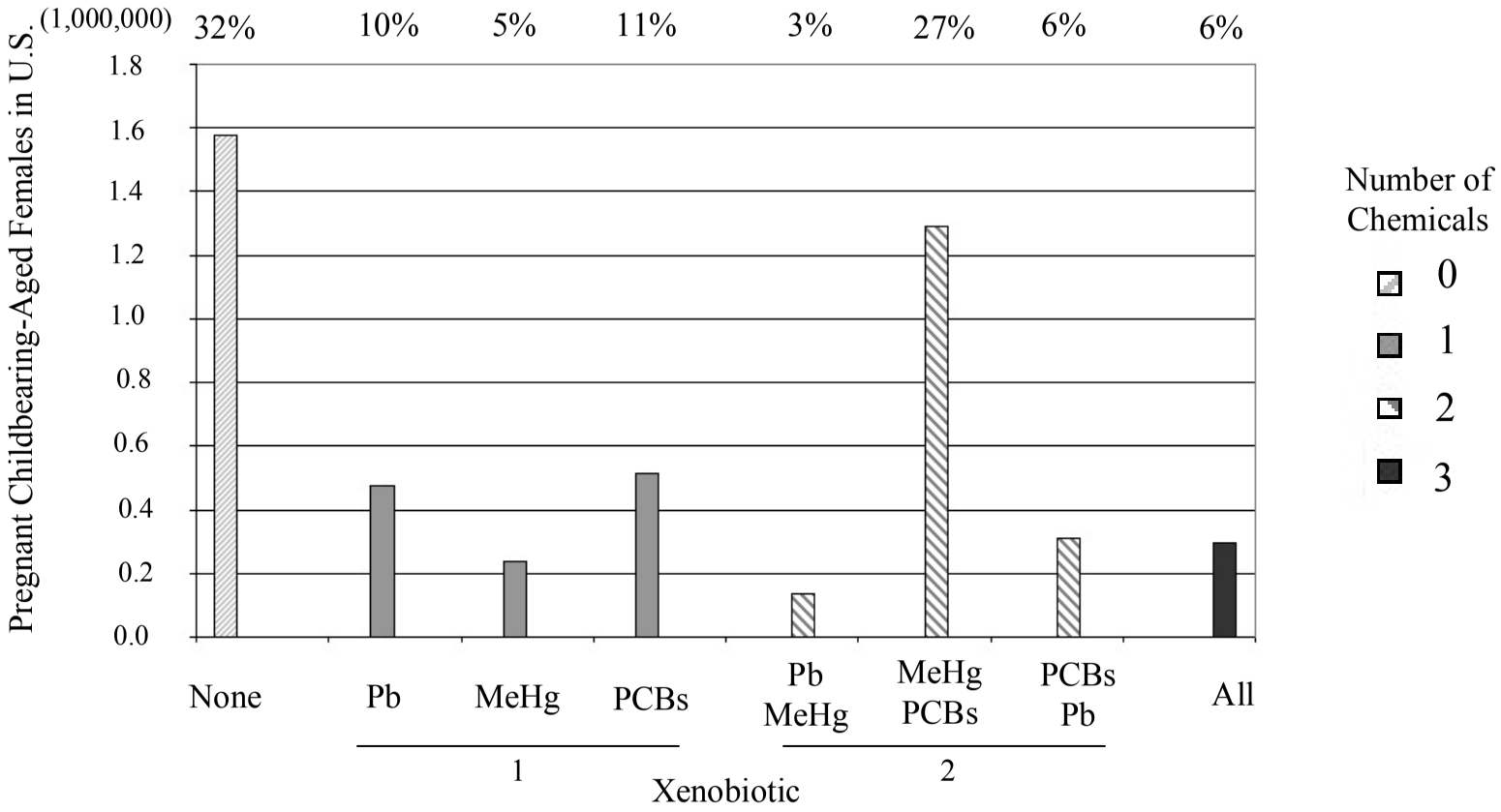


Table 33
Combinations and Permutations of Exposures: Number of Pregnant Childbearing-Aged Females with Xenobiotic Blood Levels At or Above the Geometric Mean (unweighted and weighted data 1999-2004)

	0	1	2	3
Total Number of Chemicals At or Above Geometric Mean				
Frequency $_{unweighted}$	137.00	118.00	110.00	*
Row Pct.	(35.64%)	(3.18%)	(28.13%)	(0.00%)
Frequency $_{weighted}$	1,575513.12	1,231,170.80	1,738,373.16	297,132,00
Row Pct.	(32.54%)	(25.43%)	(35.90%)	(6.13%)
Total Number of Chemicals At or Above Geometric Mean				
Sample Frequency $_{unweighted}$				
Col. Pct.				
Population Estimated Frequency $_{weighted}$				
Col. Pct.				
All Xenobiotic Blood Levels Below Geometric Mean	137.00 (100.00%) 1,575,513.12 (100.00%)			
Lead Only At or Above Geometric Mean		51.00 (43.22%) 477,587.57 (38.79%)		
Methylmercury Only At or Above Geometric Mean		33.00 (27.97%) 239,178.70 (19.43%)		
Sum of PCBs Only At or Above Geometric Mean		34.00 (28.81%) 514,404.53 (41.78%)		
Lead and Methylmercury At or Above Geometric Mean[1]			* (0.00%) 135,932.33 (7.82%)	
Methylmercury and Sum of PCBs At or Above Geometric Mean[1]			56.00 (50.91%) 1,292,921.09 (74.38%)	
Sum of PCBs and Lead At or Above Geometric Mean[1]			40.00 (36.36%) 309,519.74 (17.80%)	
All Xenobiotic Blood Levels At or Above Geometric Mean				* (100.00%) 297,132.00 (100.00%)

[2]where the third xenobiotic is below geometric mean

Figure 36. Number of Pregnant Childbearing-Aged Females in U.S. with Xenobiotic Blood Levels At or Above the Geometric Mean (weighted data 1999-2004)



Research Question Three. What, if any, subsets of childbearing-aged women were disproportionately exposed to two or more of these environmental chemicals based on susceptibility-related attributes (reproductive status, age, health and nutritional status), exposure-related attributes related to acculturation, proximity (residential characteristics and occupation), activity (diet and tap water supply) and behavior (alcohol consumption and tobacco use); socioeconomic factors (education, employment, income and marital status) and race-ethnicity?

The best fit logistic regression exposure model without interactions included 13 independent variables (in order of ascending p values and descending χ^2 values): any fish consumption in the past 30 days, age, food security, ever breastfed, highest education level attained, any shellfish consumption in the past 30 days, marital status, selenium intake/RDA, time in longest employment, alcohol consumption, household size, serum cotinine and race-ethnicity with all Hispanic grouping (Table 45).

There were three notable findings regarding relative risk for exposure with fish consumption, age and breastfeeding (Table 48).

1. Any fish consumption in the past 30 days tripled the odds of two or more xenobiotic blood levels at or above the geometric mean 95% CI [1.9, 4.9] when compared to no fish consumption during this same time period (Figure 41).

2. The odds of having two or more xenobiotic blood levels at or above the geometric mean rose non-linearly with age. From *OR* = 1.0 for ages 16 to19, to *OR* = 3.5, 95% CI [1.6, 7.9] for ages 20 to 29; *OR* = 8.5, 95% CI [3.2, 22.7] for ages 30 to 39; and finally, *OR* = 30.2, 95% CI [8.4, 109.2] for ages 40 to 49 (Figure 42).

3. Those women who were currently breastfeeding or had ever breastfed a child for more than one month were 44% less likely 95% CI [0.34, 0.93] to have two or more xenobiotic levels at or above the geometric mean than those who had never breastfed (Figure 43).

The odds of having two or more xenobiotic levels at or above the geometric mean was slightly higher for Non-Hispanic Blacks, *OR* = 1.08, 95% CI [0.56, 2.11] but slightly less for Hispanics, *OR* = 0.67, 95% CI [0.39, 1.15] and Asian, Pacific Islander, Native American or Multi-Racial, *OR* = 0.59, 95% CI [0.15, 2.32] as compared to Non-Hispanic Whites. The relative risk for exposures to multiple environmental chemicals among Hispanics and Asian, Pacific Islander, Native American or Multi-Racial was most likely underestimated due to small cell size.

It should be noted that 53% of women with two or more xenobiotic levels at or above the geometric mean worked in management, professional or sales-related occupations, 17% services- and goods-related occupations and 30% did not work at all. Data regarding occupationally-related exposures were inadequate to sufficiently assess for workplace chemical exposures.

Missing data factored into the odds ratios for marital status and food security. The significance of this missing data on results would infer that those who declined to answer these questions were somehow different than those who did answer these questions; no further explanation is offered at this time.

Comparison of Risk Factors Across Models. In an effort to better understand the origins of the risk factors that comprised the exposure model, the best-fit logistic regression exposure model with no interactions was compared to each of the best-fit

models with no interactions for lead, methylmercury and PCBs (Table 70). Variable by variable, there appeared to be no discernable pattern(s) across models. There were two notable findings regarding education and current pregnancy.

1. Education was found to be a risk factor in the exposure model but not in each chemical model. The odds of having two or more xenobiotic blood levels at or above the geometric mean were twice more likely if a childbearing-aged woman did not have a high school diploma or GED, *OR* = 1.96; CI [0.98, 3.93].

2. Conversely, current pregnancy was strongly protective in the lead, *OR* = 0.30; 95% CI [0.1, 0.7], methylmercury, *OR* = 0.65; 95% CI [0.4, 1.1] and PCBs, *OR* = 0.60, 95% CI [0.3, 1.3] best-fit logistic regression models (Table 71, Figure 44).

Modified Environmental Health Paradigm. This study tested the modified environmental health paradigm (Sexton et al., 1993b, p. 714) by exploring the interrelationships between exposure as outcome in two categories and 54 measures of vulnerability (susceptibility- and exposure-related attributes, socioeconomic factors and race-ethnicity). The goodness-of-fit for the final exposure model with no interactions was "fair" (r^2 = 0.27). It was about the same for lead (r^2 = 0.26), slightly less for methylmercury (r^2 = 0.23) but slightly higher for PCBs (r^2 = 0.35). (Table 70). This study was unable to fully test this paradigm with these models.

Discussion

Research Question One. Exposures to lead, methylmercury and PCBs were widespread among childbearing-aged and pregnant childbearing-aged women. While PCBs was the most prevalent xenobiotic overall, a relatively smaller number of

women had levels above the 99th percentile. In contrast, there were much larger numbers of women above the 99th percentile for lead and methylmercury. These findings would suggest a more widespread environmental exposure to PCBs among childbearing-aged and pregnant childbearing-aged women but disproportionately higher exposures to lead and methylmercury among among subgroups of the study population.

Other than NHANES, there are few population-based studies among women of childbearing-age in the U.S. to which comparisons can be made. In 2004, data compiled from 37 states participating in CDC's Adult Blood Lead Epidemiology and Surveillances (ABLES) Program indicated a prevalence rate of 60 per 100 (with blood lead levels ≥ 40 μg/dl among women aged 16 to 44 years (Calvert, 2007). Their prevalence rate is 11% higher than the prevelance rate calculated for this study. This difference is most likely due to an inherent selection bias in the ABLES program that is, the women tested had either lead-related occupations and/or clinically-suspected non-occupationally-related lead exposures. There were no comparable state monitoring programs identified for methylmercury or PCBs.

The National Center for Environmental Health publishes a report biennially on human exposures to environmental chemicals in the U.S. based on NHANES data (Centers for Disease Control and Prevention, National Center for Environmental Health, 2010). However, for each two-year dataset, geometric means and percentiles for specific xenobiotics are published in separate tables for gender, broad age cohorts (i.e., 12 to 19; 20 and older), and three racial-ethnic groups (i.e., Non-Hispanic

Whites, Non-Hispanic Blacks and Mexican-Americans). As a result, comparisons to these data were not possible.

Research Question Two. The magnitude of exposures to these environmental chemicals is reflected in this study's finding that 58% of childbearing-aged women (Figure 39) and 42% of pregnant women (Figure 40) had two or more xenobiotic blood levels at or above the geometric mean. Unfortunately, lead, methylmercury and PCBs represent only a fraction of all environmental chemicals to which these women were exposed. Across chemical classes such as pesticides and phthalates, Woodruff, Zota and Schwartz (2011) have documented detection of 4 to 50 chemical analytes in blood samples of pregnant and non-pregnant women.

PCBs and Lead. The binary chemical combination of PCBs and lead was identified in 17% of childbearing-aged women who had two xenobiotic levels at or above the geometric mean. In their study of adolescent girls, Denham et al. (2005) found a statistically significant interaction between these two chemicals ($p < 0.05$). Neither ATSDR nor others has assessed this chemical pair for toxicological interactions.

Methylmercury, PCBs and Fish Consumption. Another binary chemical combination, methylmercury and PCBs was identified in 27% of those who were pregnant. As discussed in Chapter Two, ATSDR (2004) predicted a greater-than-additive interaction between these two chemicals. Domestic and imported seafood and freshwater fish are primary sources of methylmercury and PCBs for adults. In this study, the strongest risk factor for two or more xenobiotic blood levels at or above the geometric mean was any fish consumption within the prior 30 days. In 2003, a

concerted effort was initiated by federal and state agencies to educate people, and in particular pregnant women, about avoiding predatory species of fish in which the biomagnification of methylmercury and PCBs was greatest. In 2009, three states (ME, RI, WA) participating in CDC's Pregnancy Risk Assessment Monitoring System (PRAMS) asked pregnant women routinely about whether their healthcare professionals counsel them about fish consumption, mercury exposure and the potential for adverse fetal outcomes (Centers for Disease Control and Prevention, 2004). In 2009, there was a 73.6% (weighted) positive response (T. Stancil, personal communication, January 26, 2011).

Research Question Three.

Age. The odds of having two or more xenobiotic blood levels at or above the geometric mean rose non-linearly with age. This study confirmed previously reported findings of a strong correlation between age with PCBs (Axelrad, Goodman, & Woodruff, 2009) and age with lead (Mushak, 1998). Bioaccumulation of these xenobiotics could explain this non-linear rise. While this study was able to confirm a statistical correlation of age with methylmercury (Caldwell, Mortensen, Jones, Caudill, & Osterloh, 2009), the relationship remained essentially unchanged with advancing age (Table 72). This may indicate a relatively larger influence of more recent exposures than those associated with long-term bioaccumulation of methylmercury.

The oldest cohort of women (aged 40 to 49) had an exponential risk, $OR = 30.2$, 95% CI [8.4, 109.2] for two or more xenobiotic blood levels at or above the geometric mean. This cohort was born from 1950 to 1963 during a time when occupational and

environmental contamination went unabated (Table 73). If historical emissions are a valid explanation, women older than 49 may have equally high or higher xenobiotic levels, though differences may be due to longer bioaccumulation. NHANES tests women aged 49 to 69 for some but not all of these xenobiotics and for some but not all the years involved in this study. While five studies (Western New York, Mount Sinai, Yale, Campaign Against Cancer and Stroke and the Nurses' Health Study) have examined blood for lipid-adjusted levels of PCB congeners 118, 138, 153, 180 in women as old as 90 (Laden et al., 2001), data correlating xenobiotic levels with age by decade were not available for comparison. A search of the scientific literature (PUBMED) have not identified any women's health studies involving methylmercury or lead among older women. To date, there are no known plans to incorporate biomarkers for these chemicals in the National Women's Health Initative (Z. Chen, personal communication, December 6, 2010).

Conversely, if historical emissions are a valid explanation, with the advent of occupational and environmental regulation and remediation in the 1970s, one would expect to find ever decreasing xenobiotic levels among successive cohorts of childbearing-aged women in subsequent survey years. The National Children's Study is a prospective longitudinal study begun in late 2009 that will examine the effects of environmental influences on the health and development of more than 100,000 U.S. children beginning preconceptually or prenatally through age 21 (Children's Health Act of 2000). It is unknown whether maternal postnatal exposures will be addressed.

Breastfeeding. In contrast to age and fish consumption, breastfeeding appeared to be protective for childbearing-aged women, *OR* = 0.56, 95% CI [0.34, 0.93]. These

odds applied to women who were currently breastfeeding as well as those who had ever breastfed at least one child for one month or more. Since all three chemicals have been measured in breast milk (Agency for Toxic Substances and Disease Registry, 2004; Bjornberg et al., 2005; Dórea, 2004), transfer of these chemicals from mother to infant-child via lactation is a likely explanation. There was no statistically significant interaction found between ever breastfed and age with the best-fit exposure model.

Current Pregnancy. Overall, women who were pregnant had lower prevalence rates than those of childbearing-aged women. This study confirms findings of Woodruff, Zota and Schwartz (2011) who found xenobiotic levels to be lower among pregnant women than non-pregnant women in their analyses of NHANES 2003 to 2004. The percentage of pregnant women with all three xenobiotic levels at or above the geometric mean was significantly lower (6%) than that of childbearing-aged women (20%). There are four possible explanations.

1. Those who were pregnant modified their lifestyles to decrease their exposures to these environmental chemicals. For example, pregnant women are advised routinely by their obstetricians to stop smoking. Based on data from 26 states, CDC estimated 13% of women reported smoking during the last three months of pregnancy (Centers for Disease Control and Prevention, 2004). This is compared to 22% of all childbearing-aged women in this study (as assessed by cotinine > 10.0 ng/ml). In this study, cotinine was associated with higher lead levels. Additionally, cotinine was one of the significant risk factors for having two or more xenobiotic blood levels at or above the geometric mean. Interaction between current pregnancy and cotinine was not statistically significant when tested with the lead model. This

interaction could not be tested in the exposure model because current pregnancy was dropped during the logistic regression analysis. Woodruff, Zota and Schwartz (2011) found relatively higher blood levels of lead and cotinine in non-pregnant women than pregnant women. In this study, while methylmercury ranked third most prevalent among childbearing-aged females, it ranked second among those who were pregnant. This rise in rank may have reflected more accurately the relatively lower level of lead among pregnant women who stop smoking.

2. These chemicals transferred from the women to their fetuses via the placenta and umbilical cord. In a study by Butler et al. (2006), maternal blood methylmercury levels were significantly lower than those in umbilical cord blood ($p < 0.0001$). Similar findings have been reported in other studies and reviewed elsewhere (Hamada, Arimura, & Osame, 1997). While it has been found to be generally true that maternal blood lead levels are significantly higher ($p < 0.0001$) than umbilical cord levels (Butler Walker et al., 2006), other studies have reported higher umbilical cord blood lead levels than maternal lead levels in approximately 25% to 28% of subjects. A study by Harville et al. (2005) attributed this phenomenon to maternal alcohol consumption. As stated in Chapter Two, alcohol has been shown to potentiate blood lead levels in animal studies (Gupta & Gill, 2000). This study could not confirm findings from Harville et al. (2005) as the interaction of alcohol consumption and current pregnancy experienced overparameterizaton when tested with the lead model. While the low lipid content of cord blood prevents similar comparisons with PCBs levels, some studies (Bergonzi et al., 2009; Wang, C. Y., et al.

2009; Wang, R., Jain, Wolkin, Rubin, & Needham, 2009) have reported higher lipid-adjusted PCB levels in maternal serum than those levels found in the placenta.

3. Age acted as an effect modifier for current pregnancy. As previously discussed in Chapter Two, studies have found maternal age to be correlated with umbilical and placental xenobiotic blood levels. Axelrad and Cohen (2010) contended that equal weighting for all age cohorts may produce a biased estimate of exposure. In other words, younger women have lower xenobiotic blood levels than older women but younger women are more likely to get pregnant than older women. In this study, only 4% of those who were pregnant were 40 to 49 years old. Age was found to be a significant predictor of two or more xenobiotic blood levels at or above the geometric mean while current pregnancy was not. While this study documented overall lower prevalence rates of each chemical among pregnant women as compared to childbearing-aged females, it could neither test for interaction between age and pregnancy with any of the regression models due to overparameterization nor confirm age as an effect modifier due to small cell size.

4. Plasma volume expansion (hemodilution) during pregnancy may have underestimated xenobiotic levels (Faupel-Badger, Chung-Cheng, Troisi, Lagiou, & Potischman, 2007). Serum albumin has been used as a surrogate for measuring plasma volume expansion by Woodruff, Zota and Schwartz (2011). When adjusted for serum albumin, the geometric means of some persistent chemicals increased while others did not change (chemicals not specified).

Comparison of Risk Factors Across Models. As stated previously, there were no discernable patterns when the best-fit logistic regression exposure model with no

interactions was compared to each of the best-fit models with no interactions for lead, methylmercury and PCBs (Table 70). There were two anomalies regarding the "appearance" of education and the "disappearance" of current pregnancy across models. One possible explanation may be differences in the operational definitions of the dependent variable across models. One xenobiotic level at or above the geometric mean was defined in the individual chemical models as "exposed" but defined as "not exposed" in the multiple chemical exposure model. One xenobiotic level at or above the geometric mean represented 26% of the data. Twice as many pregnant women had no xenobiotic blood level at or above the geometric mean than childbearing-aged women. The percentages of those with one or two chemicals at or above the geometric mean were about the same. Without further analysis, it is unknown to what extent this classification could account for the other differences seen across these models. If one chemical is the defining difference between models, it underscores the importance of studying the phenomenon of multiple environmental chemical exposures.

Modifed Environmental Health Paradigm. This study was unable to fully test the paradigm with these models. Overparameterization prevented interactions from being included in the models. There were numerous statistically significant ($p < 0.001$) interactions identified with the exposure (26), lead (29), methylmercury (23) and PCB (32) models. The overall number of interactions understate the complexity of their interrelationships. Not all known (genetic predisposition, developmental stage, activity patterns, location) and unknown contributing factors were measured while other factors not in the model (acculturation, reproductive status

and marital status) were included. Ultimately, issues of data accessibility and availability will limit the extent to which this paradigm can be tested.

Models created for examining single chemical exposures may be inappropriate for evaluating multiple chemical exposures. There may be limits to which the modified environmental health paradigm can explain the phenomenon of exposure to multiple environmental chemicals. Best-fit logistic regression models for binary chemical combinations may yield discernable patterns with respect to the multiple chemical exposure model in this study. If they do not, it would imply a complexity that seems to increase exponentially with the addition of one more chemical. In the end, this paradigm may or may not have to be modified further or discarded all together.

Now that this study's findings have been revealed and discussed, the next chapter will summarize the study, draw its conclusions and outline its limitations before outlining its implications for theory development, research, education, policy and practice.

CHAPTER 5

SUMMARY, CONCLUSIONS, LIMITATIONS AND IMPLICATIONS

In this chapter, the study is summarized, conclusions are drawn and limitations are outlined. This is followed by implications of the study for theory development, research, education, practice and policy.

Summary

Lead, methylmercury and polychlorinated biphenyls (PCBs) are pervasive, persistent and co-occur in the environment. Each of these environmental chemicals are known to have neurobehavioral and neurodevelopmental consequences in animal models and human population studies. Since these neurotoxins bioaccumulate, the body burden from past exposures as well as maternal exposures during gestation transfer from mother to fetus via the placenta and to an infant and young child through lactation. Despite what is known about the hazards of exposures to these specific environmental chemicals, little is known about exposures to combinations of these chemicals. The purpose of this study was to address this research gap. This secondary analysis established the prevalence of four combinations and permutations of these chemicals in a large national probability sample of childbearing-aged women living in the United States 1999to 2004.

Exposure was defined as "the contact between an agent and a target with contact taking place at an exposure surface over an exposure period by an exposure route" (International Programme on Chemical Safety, 2000, p. 21). Six exposure-related

concepts were identified and defined: agent, environment, target (human), dose, health and vulnerability. The conceptual framework was Sexton, Olden and Johnson's modified environmental health paradigm (1993a).

This descriptive and exploratory study, existing data were analyzed from the National Health and Nutrition Examination Survey (NHANES), a national probability sample. The outcome of interest was based on evidence of biological uptake of two or more of the following: lead, methylmercury and the summed value of four lipid-adjusted polychlorinated biphenyl congeners (118, 138/158, 153 and 180) as measured by the presence of these xenobiotics at or above the geometric mean in the blood or serum of childbearing-aged females aged 16 to 49 of diverse races and ethnicities who were living in the U.S. from 1999 to 2004. The final cohort for this study consisted of 3,173 women including a subset of 391 who were pregnant. When adjusted (weighted) to the U.S. population, these participants represented 134.5 million childbearing-aged females of whom 4.8 million were pregnant. There were 62 measures of vulnerability (susceptibility- and exposure-related attributes, socioeconomic factors and race-ethnicity).

Data analysis encompassed concatenating and organizing the dataset, operationalizing dependent and independent variables and constructing software instructions. Descriptive and univariate statistics were used to estimate prevalence of exposure to each of the environmental chemicals of interest. Bivariate analyses (χ^2) identified the most common combinations and permutations of exposures. Best-fit logistic regression models were developed and analyzed. Tests for collinearity among independent variables were negative. All statistically-significant two-way interactions

among the independent variables were identified by comparing nested models using likelihood ratio testing. The data analysis concluded with calculating estimates of risk. The three research questions and major findings related to each one are summarized below.

1. What was the prevalence of childbearing-aged and pregnant childbearing-aged women's exposures to each of the following environmental chemicals: lead, methylmercury and polychlorinated biphenyls (PCBs) as measured by chemical-specific (xenobiotic) levels at or above geometric mean in blood or serum of these women who were living in the United States from 1999 through 2004?

Among childbearing-aged females, the prevalence rates for xenobiotic blood or serum levels at or above the geometric mean were 49 per 100 for lead, 48 per 100 for methymercury, and 67 per 100for PCBs. The number of childbearing-aged females above the 99th percentile was approximately 3 million, 2 million and 1.8 million for lead, methylmercury and PCBs, respectively. Women who were pregnant had lower prevalence rates.

2. What combinations and permutations of chemical exposures were most common among these childbearing-aged and pregnant childbearing-aged women as evidenced by xenobiotic blood levels at or above the geometric mean?

One fifth of childbearing-aged females had xenobiotic blood levels at or above the geometric mean for all three chemicals. The binary chemical combination of PCBs and lead was identified in 17% of childbearing-aged women who had two xenobiotic levels at or above the geometric mean while methylmercury and PCBs was identified in 27% of those who were pregnant.

3. What, if any, subsets of childbearing-aged women were disproportionately exposed to two or more of these environmental chemicals based on susceptibility-related attributes (reproductive status, age, health and nutritional status), exposure-related attributes related to acculturation, proximity (residential characteristics and occupation), activity (diet and tap water supply) and behavior (alcohol consumption and tobacco use); socioeconomic factors (education, employment, income and marital status) and race-ethnicity?

The best fit logistic regression exposure model included 13 independent variables: any fish consumption in the past 30 days, age, food security, ever breastfed, highest education level attained, any shellfish consumption in the past 30 days, marital status, selenium intake/RDA, time in longest employment, alcohol consumption, household size, serum cotinine and race-ethnicity.

Conclusions

Exposures to lead, methylmercury and PCBs were widespread among childbearing-aged and pregnant childbearing-aged women. Prevalence rates and distributions above the 99th percentile suggested a more widespread environmental exposure to PCBs among childbearing-aged and pregnant childbearing-aged women but disproportionately higher exposures to lead and methylmercury among subgroups of the study population. Women who were pregnant had lower prevalence rates. Lead and PCBs and PCBs and methylmercury were identified as the most common binary combinations among childbearing-aged women and pregnant women, respectively.

There were three notable findings regarding risk factors for multiple chemical exposures. Any fish consumption in the past 30 days tripled the odds of two or more

xenobiotic blood levels at or above the geometric mean. The odds of having two or more xenobiotic blood levels at or above the geometric mean rose non-linearly with age; exponentially among those aged 40 to 49. Those women who were currently breastfeeding or had ever breastfed a child for more than one month were 44% less likely to have two or more xenobiotic levels at or above the geometric mean than those who had never breastfed.

There were no discernable patterns across models. There were two anomalies noted regarding the appearance of education and the omission of current pregnancy in the best-fit exposure model as compared to those best-fit models for each chemical.

Limitations

There were six major limitations to this study. Since all data were collected at a single point in time only associations could be made about the relationships between dependent and independent variables. This study examined three chemicals which represent only a fraction of all chemicals detectable in the environment. While independent variables were selected based on the theoretical framework and a review of the scientific literature, not all variables could be operationalized given the data that were available in NHANES. This study was unable to fully test Sexton, Olden and Johnson's modified environmental health paradigm (1993a). Not all known (genetic predisposition, developmental stage, activity patterns, and location) and unknown contributing factors were measured while new factors were included (acculturation, reproductive status and marital status). The best-fit logistic regression models did not include interactions because the data were too sparse to test for all interactions. While this study's findings can be generalized to the population of childbearing-aged women

who lived in the United States 1999 to 2004, no inferences should be made regarding individual exposures or exposures among other populations inside or outside the United States.

Implications for Theory Development

In this study, a newly-identified concept (exposure) was introduced to nursing within the client domain. A concept analysis (Thompson, 2006) revealed that exposure had not been defined explicitly in the nursing literature even though the term was used frequently and characterized in many different ways. A transdisciplinary perspective was used to define exposure as "the contact between an agent and a target with contact taking place at an exposure surface over an exposure period by an exposure route" (International Programme on Chemical Safety, 2000, p. 21). As it is currently defined here, use of this concept should be incorporated into other nursing-related research, regardless of specialty. In this way, its usefullness to nursing can be evaluated.

Additionally, exposure could be considered a central concept in the environmental domain. Exposure as currently defined is conceptually congruent with Kim's definition of environment: "a separate entity that exists external to a person or to humanity, conceived … as that containing many distinct elements" that is, spatial, temporal and qualitative (socio-cultural) (Kim ,2000, p. 166). All nursing specialties support the relevancy and inclusion of the environment in research, practice and policy.

Alignment of this concept with existing theories was critically examined. The original environmental health paradigm explained exposure adequately but did not

address vulnerability. The modified environmental health paradigm was particularly helpful in understanding the interrelationships of exposure and vulnerability. In turn, this research supported its use empirically. Therefore, further use of this modified paradigm is endorsed for other related research.

In this study, new variables (acculturation, marital status and reproductive status) were introduced to the paradigm. It is unclear whether acculturation should continue to be subsumed as an exposure-related attribute as it was in this study or integrated with race-ethnicity. The role of marital status as a socioeconomic factor remains unclear due to missing data. Reproductive status was particularly important with this study population. All of these variables should be explored further to determine whether they should remain in the model.

Implications for Research

There were a number of useful avenues identified for progressing research in environmental health regarding multiple chemical exposures, including other population subgroups, improving specific variable measurement and expanding public access to NHANES data.

The widespread prevalence of exposures to these chemicals clearly indicate that further research on exposures to multiple environmental chemicals is needed. In general, mechanistic studies on interactions of multiple chemical combinations using *in vitro* toxicity assays would improve understanding of toxicokinetics and toxicodynamics at the cellular level. The need for mechanistic studies of PCBs on lead and lead on PCBs is underscored by this study's finding that this particular binary chemical combination was prevalent in 17% of childbearing-aged women who had

two xenobiotic levels at or above the geometric mean. Whether these chemicals have the same or similar toxic action may be irrelevant if there is a cumulative impact on health. In an effort to further understand the origins of the risk factors that comprised the best-fit logistic regression exposure model, best-fit logistic regression models for binary combinations of these chemicals should be formulated using these same datasets. Then, variables could be examined for discernable patterns across all models. These comparisons could assist in illuminating whether binary chemical models are appropriate for evaluating multiple chemical exposures and/or confirm whether single chemical models used currently to evaluate binary chemical exposures are still appropriate. In anticipation of improving the models' goodness-of-fit, interactions among independent variables could be more fully described by adding to the dataset from NHANES survey years (2005 to 2010). Evaluating the impact of bioaccumulation from multiple environmental chemical exposures on health will require longitudinal prospective studies. Transgenerational consequences of exposures will require prospective studies spanning more than two generations. In the meantime, childbearing-aged and pregnant childbearing-aged women will continue to be exposed to these neurotoxins. The magnitude of harm is larger than once thought. The severity of harm for those most vulnerable is irreversible (Barker, 2004; Barker et al., 2002).

With this study's finding of an exponential relative risk for exposure among women aged 40 to 49, establishing the prevalence of exposures to these environmental chemicals among females older than 49 should be strongly considered. With this additional information, it may be possible to determine whether this risk is related to

historical emissions, bioaccumulation or both. There may be opportunities for collaboration with existing studies. For example, from 1999 to 2004, NHANES analyzed blood samples of women aged 49 to 69 for lead and PCBs but not total and inorganic mercury. These blood samples are still available. Supplemental research proposals could be submitted to existing studies such as the National Women's Health Initiative and others (Western New York, Mount Sinai, Yale, Campaign Against Cancer and Stroke and/or the Nurses' Health Study).

Some methodological challenges were encountered during operationalization of independent variables. In general, validation is needed in the use of the Charleson Comorbidity Index (Charlson et al., 1987) and Child-Turcotte-Pugh Score (Child & Turcotte, 1964; Pugh et al., 1973) as measures of co-morbidity in a non-institutionalized population. In addition, the following recommendations address the NHANES dataset specifically:

1. Release population density-related data for all survey participants with regard to U.S. Census Bureau designations for urban, suburban or rural residence;
2. Obtain more detailed occupationally-related hazards data;
3. Change measurement of physical activity in accordance with established national standards (Ainsworth et al., 2000; Haskell et al., 2007);
4. Calculate packyears, pipeyears, etc. for former as well as current tobacco users;
5. Reinstate the question regarding history of peptic ulcers and add a question regarding gastric esophageal reflux disease (GERD);

6. Unrestrict data on alcohol consumption and tobacco use among 16 to 19 year olds.

7. Since CDC's *Healthy People 2020* objective EH-20.12 designated PCB 153 and PCB 126 as representatives of the non-dioxin-like and dioxin-like PCBs, respectively (Centers for Disease Control and Prevention, 2010), PCB 126 should be included in future studies. A petition to NHANES would be required to obtain data for this PCB congener for 1999 to 2004.

Implications for Education

The findings of this study should be used to inform healthcare practitioners and occupational and environmental health and safety (OEHS) professionals of the widespread prevalence of childbearing-aged and pregnant childbearing-aged women's exposures to lead, methylmercury and PCBs from 1999 to 2004. Emphasis should be placed on the transgenerational consequences of bioaccumulation and maternal exposures during gestation and lactation. Of equal importance are the interrelationships of exposure and vulnerability and risk factors for multiple environmental chemical exposures illuminated by this study.

In general, one outcome of this study is recognition of the need for continuing education with regard to these and related subjects, particularly, but not exclusively, among those practitioners in the maternal and child health-related specialties. Another outcome would be a more formal integration of these subjects into undergraduate and graduate curricula. It is through these educational efforts that healthcare practitioners and OEHS professionals will begin to integrate this new knowledge into their clinical practice.

Implications for Practice

In this study, a multitude of factors that increased or decreased prevalence of exposures were evaluated. By doing so, this study has facilitated practice aimed at ameliorating and preventing adverse health outcomes. Efforts at the state and local levels should concentrate on identifying, mitigating and eradicating existing anthropogenic sources of lead, methylmercury and PCBs. *Think global, act local* (Dubos, 1977). These interventions should emphasize continuous improvement and incorporate management of change with the application of the ALARA principle (As Low As Reasonably Achievable) and the use of best available technology.

Risk-based communication should ensure that the results of this research are readily available and understandable especially to demographic groups who were disproportionately represented in at-risk categories in this study. These communications must be appropriate to the audience and adequately address four areas of concern:

1. What is known with what accuracy and with what confidence?
2. What is not known and why is there uncertainty?
3. What could be known if there were more time, money and talent?
4. What should be known in order to act in the face of uncertainty?

(National Research Council, 2006, p. 209).

Opportunities to conduct participatory-based research should be encouraged. The effectiveness of fish consumption advisories for childbearing-aged women and in particular those who are pregnant should be evaluated. As one of the public's most trusted disciplines, nursing is in a unique position to be most effective in addressing the public's concerns about their environmentally-related health.

In order to protect the public's health, it becomes clear that risk-based efforts are not enough. Precaution and proaction should be advocated. Precautionary-level interventions should be designed and implemented to prevent exposures to these chemicals. Over time, these efforts will most likely result in lower exposure levels with a goal of zero harm through a cleaner environment, safer workplaces and healthier homes.

Currently, the routine use of xenobiotic biomonitoring in clinical practice is discouraged. Traditionally, occupational and environmental regulations have relied on noninvasive area and/or personal monitoring to quantify chemical exposures with subsequent invasive biomonitoring only if the regulatory action level is exceeded. While the elegance of xenobiotic biomonitoring is that it confirms exposure to a specific chemical, the ugliness of it is that it does not identify exposure sources. Transgenerational consequences of maternal exposures and bioaccumulation compound this problem. Interpreting biomonitoring results represents the biggest challenge to practitioners: *What does it mean in terms of an individual's health?* This question is not addressed in population-based research such as this study. However, public health policy is being formulated around it.

For example, one of CDC's *Healthy People 2020* objectives is a 30% reduction in these xenobiotic blood levels(Centers for Disease Control and Prevention, 2010):

Lead: 2.94 µg/dl
Mercury: 1.26 µg/l (ages 1-5) and 3.22 µg/l (childbearing-aged women)
PCB 126: 48.09 pg/g of lipid (ages 12 and older)
PCB 153: 67.97 ng/g of lipid (ages 12 and older)

Implications for Policy

Public health policy has a regulatory and fiduciary obligation to address two questions: *Is it safe? Is it safe enough?*

Is it safe? In this study, one fifth of childbearing-aged women were exposed to lead, methylmercury and PCBs as evidenced by blood levels concurrently at or above the geometric mean. Current environmental health policy has not addressed adequately the potential health effects of exposures to multiple environmental chemicals. The demand for strong empirical justification has led to a regulatory process that responds only when a high certainty of severe harm exists, a casualty of the 1980 U.S. Supreme Court benzene decision (Appendix I. International Union, AFL-CIO, et al. v. Petroleum Institute 448 U.S. 607, 1980). As discussed previously, strong empirical evidence will take decades to assemble. Proposed legislation that is currently before the U.S. Senate Committee on Environment and Public Works would strengthen the effectiveness of U.S. EPA's 1976 Toxic Substances Control Act (TSCA). However, given the recent changes in House majority and opposition from business, the future of this pending legislation is uncertain. In the meantime, another generation is exposed. The proposed *Safe Chemicals Act* is based on six principles established by the U.S. Environmental Protection Agency (2010f):

1. Chemicals should be reviewed against safety standards that are based on sound science and reflect risk-based criteria protective of human health and the environment.
2. Manufacturers should provide EPA with the necessary information to conclude that new and existing chemicals are safe and do not endanger public health or the environment.
3. Risk management decisions should take into account sensitive subpopulations, cost, availability of substitutes and other relevant considerations.

4. Manufacturers and EPA should assess and act on priority chemicals, both existing and new in a timely manner.
5. Green chemistry should be encouraged and provisions assuring transparency and public access to information should be strengthened.
6. EPA should be given a sustained source of funding for implementation.

These principles bellweather two changes to current policy. One is the shift in the burden of proof from "innocent until proven guilty" to "guilty until proven innocent." The other requires risk assessments to address "sensitive subpopulations." It is uncertain whether this reference encompasses fetuses.

Is it safe enough? Should protecting the next generation by regulating environmental exposures of the current generation be addressed in public and environmental health policy? Should pregnant and lactating women be exclusively protected? These are complex issues with implications for healthcare practice and far-reaching impacts on corporate social responsibility and society as a whole. There are no guidelines for multiple chemical exposures among pregnant and lactating women. Until just recently, there were no guidelines for single chemical exposures among these women either. Clearly, this study suggests that guidelines are needed for both. Though this study did not focus on single chemical exposures, these recent guidelines do herald first steps into the quagmire of controversy. The possibilities for unintended consequences of its implementation are discussed here.

Recently, the National Center for Environmental Health (NCEH) published *Guidelines for the Identification and Management of Lead Exposure in Pregnant and Lactating Women* (2010). "These recommendations … should not significantly impact many individuals or clinical practices" (National Center for Environmental Health, 2010, p. iv). These guidelines do not recommend routine prenatal testing of

all women in the U.S. because it is estimated only one percent of the population would be impacted. This study estimated three million childbearing-aged and 433,000 pregnant childbearing-aged women had blood lead levels above the 99th percentile. These guidelines recommend client education as well as environmental, nutritional, and behavioral interventions beginning when a woman's prenatal blood lead level is 5 μg/dl and higher. Secondly, these NCEH guidelines encourage mothers in the U.S. to breastfeed as long as their blood lead levels are less than 40 μg/dl. It encourages those women with higher blood lead levels to pump and discard their breast milk until their blood lead levels are below 40 μg/dl. The reader is reminded that for every 10 μg/dl increase in blood lead level there is a corresponding five point decrease in the mean IQ scores in children (Needleman, 1989). This study has reported prevalence rates were lower for pregnant women than those of childbearing-aged women. Four possible explanations were offered as to why current pregnancy was protective for these women. As discussed previously, interpretation of biomonitoring results will be complicated by the physiological changes that occur during pregnancy and bioaccumulation from past exposures. Given this study's findings and the litigious society in which healthcare practices, the most prudent recommendation may be for clinical practitioners to routinely conduct lead biomonitoring with initial preconceptual or prenatal care which is followed by postnatal testing of lactating women. Limited access to healthcare for women at highest risk for exposure and the availability of qualified analytical laboratories and the cost will be significant barriers to implementation.

These guidelines recommend removing pregnant women from occupational lead exposures at blood lead levels 10 μg/dl and higher. Currently, medical surveillance guidelines in the U.S. Department of Labor, Occupational Safety and Health Administration's (OSHA) lead standard (29 Code of Federal Regulations §1910.1025, §1926.62) requires employers to implement a biomonitoring program only when the environmental lead level exceeds an eight-hour time-weighted average of 30μg/m^3 for more than 30 days per year. Subsequently, any worker with a blood lead level 40 μg/dl and higher is removed from the work area while being afforded job protection. OSHA allows an employee to return to work when two consecutive blood lead levels are below this threshold. The NCEH guidelines neither indicate at what blood lead level a pregnant woman should return to work nor clarify whether a blood lead level of 10 μg/dl and higher is de facto worker compensable. (It should be noted that this study found non-U.S. citizenship, older residential age and tobacco use were three of the statistically significant risk factors for a blood lead level at or above the geometric mean). Employers have a statutory obligation to provide a safe workplace for all workers. While a fetal protection policy is neither acceptable nor legal (Appendix J. International Union, United Automobile, Aerospace and Agricultural Implement Workers of American, UAW, et al. v. Johnson Controls, Inc. 499 U.S. 187, 1991), would employers be held liable for fetal injury? Clearly, these incongruences require further clarification. *So, why are we waiting nine months to find out about maternal and fetal exposures to environmental chemicals?* "The Precautionary Principle urges precaution when the magnitude of the potential adverse event is large

or the adverse outcome is severe, even if its probability is small" (Ricci et al., 2003, p. 3).

Exposures to environmental chemicals in the United States impact society as a whole. In a 1989 commentary on lead poisoning, Dr. Herbert L. Needleman discussed a study which found a difference in mean IQ scores between children exposed to lead and those who were not exposed. He wrote:

> This four-to-seven point difference in means has been taken by some as a small effect. This is deceptive. The cumulative frequency distribution for IQ, typical for many distributions is sigmoid. When cumulative distributions between groups are plotted and compared, a shift in the curve resulting in a difference in medians of six points results in a four-fold increase in the rate of severe deficit (IQ < 80). This same shift in distribution truncates the upper end of the curve, where superior function is displaced by 16 points. This means that five percent of lead-exposed children are prevented from achieving truly superior function (IQ > 125). The costs of this effect at the high end of the distribution have received no attention; they may be extraordinarily important to our society (p. 643).

Just think how smart we all could have been.

APPENDIX A

ACRONYMS

AAOHN	Association of American Occupational Health Nurses
ABLES	Adult Blood Lead Epidemiology and Surveillance
ALARA	as low as reasonably achievable
AMDR	acceptable macronutrient distribution range
ANOVA	analysis of variance
APHA	American Public Health Association
ASSE	American Society of Safety Engineers
ATSDR	Agency for Toxic Substances and Disease Registry
BINWOE	binary weight of evidence
BMI	body mass index
χ^2	chi -square
CCMI	Charlson Co-Morbidity Index
CD4	cluster of differentiation 4 cells
CDC	Centers for Disease Control and Prevention
CFSM	Core Food Security Measure
CI	confidence intervals
CINAHL	Cumulative Index to Nursing and Allied Health Literature
DNA	deoxyribonucleic acid
EHS	environmental health and safety
EIA	enzyme immunoassay
EPA	Environmental Protection Agency
ET-AAS	electrothermal atomic absorption spectrometry
ETS	environmental tobacco smoke
fL	femtoliters (a measurement of volume)
FSSM	Food Security and Hunger Survey Module
FWA	Federal Wide Assurance
GAO	Governmental Accounting Office
GB	gigabyte
GED	general educational development
Hg^0	elemental mercury
HHANES	Hispanic Health and Nutrition Examination Survey
HIV	Human Immunodeficiency Virus
HRGC/HRMS	high resolution gas chromatography/high resolution mass spectrometry
ICP/DRC-MS	inductively-coupled plasma dynamic reaction cell-mass spectrometry
ICP-MS	inductively-coupled plasma-mass spectrometry

ID HPLC-APCI MS/MS	isotope dilution high performance liquid chromatography – atmospheric pressure chemical ionization tandem mass spectrometry
IHg	inorganic mercury
IPCS	International Programme on Chemical Safety
IQ	Intelligence Quotient
IRB	Institutional Review Board
IUPAC	International Union of Pure and Applied Chemistry
kg/m^2	kilograms per squared meter
LoD	level of detection
µg/dl	micrograms per deciliter
µg/L	micrograms per liter
mg/dl	milligrams per deciliter
mg/kg	milligrams per kilogram
mg/m^3	milligrams per cubic meter
MEC	Medical Examination Center
MeHg	methylmercury
mRNA	messenger ribonucleic acid
ng/dl	nanograms per deciliter
ng/g	nanograms per gram
ng/ml	nanograms per millilter
NCEH	National Center for Environmental Health
NCHS	National Center for Health Statistics
NHANES	National Health and Nutrition Examination Survey
NHES	National Health Examination Survey
NIEHS	National Institute of Environmental Health Sciences
NMDA	n-methyl-D-aspartic acid
OEHS	Occupational and Environmental Health and Safety
OR	odds ratio
OSHA	Occupational Safety and Health Administration
oz.	ounce
pg/g	picograms per gram
p value	probability of rejecting the null hypothesis when it is actually true
Pb	lead
PC12	a cell line derived from a pheochromocytoma cell of rat adrenal medulla
PCBs	polychlorinated biphenyls
PHI	protected health information
ppm	parts per million
PRAMS	Pregnancy Risk Assessment Monitoring System
PUBMED	Publication Service of U.S. National Library of Medicine
r^2	goodness-of-fit statistic
RDA	recommended dietary allowance
RNA	ribonucleic acid

RR	relative risk
SD	standard deviation
SELDI-TOF MS	surface-enhanced laser desorption/ionization-time-of-flight mass spectrometry
TIBC	total iron binding capacity
TSCA	Toxic Substances Control Act
TSDF	transfer, storage and disposal facility
VIF	variance inflation factor
WIC	Special Supplemental Nutrition Program for Women, Infants and Children

APPENDIX B

HAZARD CATEGORIES

Chemical Hazards
- Health-Related Chemical Hazards (corrosive, irritant, sensitizer, toxic, carcinogen, reproductive, asphyxiates)
- Physical-Related Chemical Hazards (flammable, combustible, reactive, oxidizer, compressed gas, cryogen)
- Nanotechnology

Physical Hazards
- Energy/Electro-Magnetic Fields/Electricity
- Ionizing Radiation (x-rays, gamma rays)
- Non-Ionizing Radiation (ultraviolet, laser, infrared, radio frequencies, sound/noise)
- Temperature (heat/cold)
- Pressure
- Climatological

Mechanical Hazards
- Mechanical Energy
- Vibration
- Ergonomically-Related Hazards (posture, position, pressure, repetitive motion)
- Manual Materials Handling
- Impact (slip, trip and fall hazards, trauma)
- Confined Spaces

Biological Hazards
- Allergens, bacteria, endotoxins, envenomations, fungi/mold, malignant cells, microbacteria, parasites, recombinants, rickettsiae, viruses, wood dust

Psychosocial Hazards
- Stress and Strain (emotional strain, interpersonal strain, lateral violence, work-family conflict)
- Fatigue (shift work)
- Organizational Culture
- Violence/Terrorism

Appendix B. Hazard Categories. Adapted from "Scientific Foundations of Occupational and Environmental Health Nursing Practice" by J. Agnew, 2001. In M. K. Salazar (Ed.), *Core Curriculum for Occupational & Environmental Health Nursing* (2nd ed., pp. 111-145). Copyright 2001 by W. B. Saunders Company.

APPENDIX C

CONCEPTUAL DEFINITIONS

Environment

A separate entity that exists external to a person or to humanity … as that containing many distinct elements: spatial, temporal and qualitative (Kim, 2000, p. 167)

Exposure Pathway. The course an agent takes from its source to the target (International Programme on Chemical Safety, 2000, p. 24; Zartarian, Ott, & Duan, 2007, p. 58)

Transport. Carrier medium for an agent: air, water, soil, dust, food, product or item (International Programme on Chemical Safety, 2000, p. 22)

Medium. Material surrounding or containing an agent: air, water, soil, food, product or item (Zartarian, Bahadori, & McKone, 2005, p. 4)

Micro-Environment. Surroundings that can be treated as homogeneous or well-characterized in regard to the concentration of an agent (Zartarian, Bahadori, & McKone, 2005, p. 4)

Accumulation in Environment. Refers to agents with extended biogeochemical cycles; persistence; environmental factors that facilitate accumulation: air and sea temperatures, wind speed, variation in precipitation (Lindberg et al., 2007) or soil acidification (Navratil, Skrivan, Vach, Dobesova, & Langrova, 2004)

Transformation. Conversion of agent into one or more resultant products by biotic or abiotic processes such as hydrolysis, oxidation-reduction; dependent upon physical and chemical properties of agent and medium (Yong, 2001, p. 208)

Agent

A chemical, biological or physical entity that contacts a target (Zartarian, Ott, & Duan, 2007, p. 58); a/k/a hazard, stressor

Hazard. Agent or environment capable of causing harm. There are five general types of hazards: chemical, physical, mechanical, biological and psychosocial (Agnew, 2001, p. 111); a threat comprised of perturbations, stress/stressors and the consequences they produce (Turner et al., 2003a, p. 8074)

Stressor. Any entity, stimulus or condition that modulates normal functions of an organism (target) or induces an adverse response (Zartarian, Ott, & Duan, 2007, p. 59); a source of continuous or slowly increasing stress; commonly found within the range of normal variability (Turner et al., 2003a, p. 8074)

Perturbation. Beyond the normal range of variability in which the system operates (Turner et al., 2003a, p. 8074)

Source a/k/a Emission Source. The origin of an agent: anthropogenic (man-made) or non-anthropogenic (exists in nature); area or point; stationary or mobile; indoor or outdoor; occupational or residential or community; site- or source-specific; geographic in scope (i.e., local, regional, national, international, global) (International Programme on Chemical Safety, 2000, p. 23)

Concentration (of agent). The amount of matter-form agent per unit volume for example, mg/kg (food), mg/liter (water), $\mu g/cm^2$ (surface), % by weight, $\mu g/m^3$ (air),

fibers/ m^3 (air), parts per million or ppm (air); a/k/a media concentration (International Programme on Chemical Safety, 2000, p. 22; Zartarian, Ott, & Duan, 2007, p. 57)

Properties. Physical state and chemical behavior of agent that directly affects rate and extent of dose and distribution in the environment and target: energy or matter; gas or vapor; molecular size; pH (acidity or alkalinity); hydro- or lipid-solubility; (Zartarian, Ott, & Duan, 2007, p. 39)

Intake Fraction. Incremental intake of a pollutant summed over all exposed individuals and occurring over a given exposure time released from a specified source or source class, per unit of pollutant emitted (Bennett et al., 2002, p. 2)

Mixture. Any combination of two or more agents regardless of source or of spatial or temporal proximity (Agency for Toxic Substances and Disease Registry, 2001, p. 3)

Similar Mixture. Agents with comparable properties that is, chemical structure, toxicological mechanism or common mode of toxicity (Sexton et al. 1995c; Sexton & Hattis, 2007, p. 825)

Defined Mixture. Agents possessing reasonably defined composition but not necessarily possess similar properties when emitted at a given time and place (Sexton et al., 1995c; Sexton & Hattis, 2007, p. 825)

Coincidental Mixture. Agents present at a common time or place of interest but not necessarily possess similar properties or composition (Sexton et al., 1995c; Sexton & Hattis, 2007, p. 825)

Exposure

Contact between an agent and a target with contact taking place at an exposure surface over an exposure period by an exposure route (International Programme on Chemical Safety, 2000, p. 21)

Exposure Surface. A surface on a target where an agent is present a/k/a contact boundary or contact surface (Zartarian, Ott, & Duan, 2007, p. 57)

Exposure Period. The time of continuous contact between an agent and a target (Zartarian, Ott, & Duan, 2007, p. 58)

Exposure Frequency. The number of exposure events in an exposure duration (Zartarian, Ott, & Duan, 2007, p. 58)

Exposure Duration. Cumulative length of time over which continuous or intermittent contact occurs between an agent and a target (International Programme on Chemical Safety, 2000, p. 22; Zartarian, Ott, & Duan, 2007, p. 58)

Exposure Route. The way an agent enters a target after contact: inhalation, ingestion, dermal absorption, injection; a/k/a/ route of entry (International Programme on Chemical Safety, 2000, p. 22; Zartarian, Bahadori, & McKone, 2005, p. 3)

Exposure Concentration. Concentration of an agent at the point of contact with the outer boundary of the target (International Programme on Chemical Safety, 2000, p. 26)

Absorption Barrier. A contact boundary or exposure surface that allow differential diffusion of an agent into a target: skin, respiratory tract lining, gastrointestinal tract wall (International Programme on Chemical Safety, 2001b, p. 3; Zartarian, Bahadori, & McKone, 2005, p. 2)

Exposure-Related Terms

Target. A biological entity, physical or ecological object exposed (or potentially exposed) to an agent that is, human or non-human, population, subpopulation, organ system, subsystem or system component (Zartarian, Bahadori, & McKone, 2005, p. 4); an entity capable of compensatory response and adaptation (Dubos, 1980, p. 22); a/k/a organism

Dose. The amount of agent that enters a target in a specified time duration after crossing an exposure surface or absorption barrier a/k/a internal dose, absorbed dose (International Programme on Chemical Safety, 2000, p. 27)

Intake. The process by which an agent crosses an outer exposure surface of a target without passing an absorption barrier (Zartarian, Ott, & Duan, 2007, p. 58)

Intake Dose. Dose resulting when an agent crosses an outer exposure surface of a target without passing an absorption barrier (Zartarian, Ott, & Duan, 2007, p. 58)

Uptake. The process by which an agent crosses an absorption barrier (Zartarian, Ott, & Duan, 2007, p. 59)

Uptake Dose. Dose that results from an agent crossing an absorption barrier (Zartarian, Ott, & Duan, 2007, p. 58)

Bioavailability. The rate and extent to which an agent can be absorbed by a target and is available for metabolism or interaction with biologically significant receptors; a/k/a internal dose (International Programme on Chemical Safety, 2000, p.26)

Toxicokinetics. Modeling and mathematical description of the time course of disposition of xenobiotics in the whole organism (Medinsky & Valentine, 2003, p. 98). "What does the target do to the agent?" (Rozman, Doull, & Hayes, 2001, p. 3)

Distribution. Distribution of agent among target's anatomical or physiologic compartments via systemic circulation (blood and/or lymph) resulting in different concentrations in various compartments (tissues and/or organs) over time; distribution rate depends initially upon absorption rate of agent by target (Gregus & Klaassen, 2003, p. 23)

Mechanisms Facilitating Distribution. Porosity of the capillary endothelium, specialized transportation across the plasma membrane, accumulation in cell organelles, reversible intracellular binding (Gregus & Klaassen, 2003, p. 24)

Mechanisms Opposing Distribution. Binding to plasma proteins, specialized barriers, distribution to storage sites, association with intracellular binding proteins, exportation from cells (Gregus & Klaassen, 2003, p. 24)

Bioaccumulation. Accumulation of agent and/or its metabolites in the target via storage and/or re-absorption (Eaton, 2005, p. 98)

Storage. Accumulation of agent in one or more of the target's tissues (Dix, 2001, p. 568)

Re-absorption. Agent diffuses back across cellular membrane and re-enters distribution system. Increases half-life. Dependent upon lipid solubility of agent. Inversely related to degree of ionization (Gregus & Klaassen, 2003, p. 25)

Elimination. Chemical and physical mechanisms by which a target first detoxifies then excretes an agent (Gregus & Klaassen, 2003, p. 24)

Biotransformation. Biochemical mechanism employed by target to breakdown the agent; enzymatic pathways. Metabolic activation or detoxification reactions that increase hydrophilicity and promote excretion by changing an agent into its metabolite which may be more or less toxic to target (Eaton, 2005, p. 99)

Excretion. Physical mechanism by which target returns agent to the environment: urine, bile/feces, exhaled breath, sweat, hair (Gregus & Klaassen, 2003, p. 24)

Toxicodynamics. Modeling and mathematical description of the time course of disposition of xenobiotics in the whole organism (Medinsky & Valentine, 2003, p. 98). "What does the agent do to the target?" (Rozman, Doull, & Hayes, 2001, p. 3)

Biologically-Effective Dose. That portion of the dose that reaches the target site of (toxic) action; a/k/a delivered dose (International Programme on Chemical Safety, 2000, p. 27)

Target Site. Endogenous molecule, cell, tissue, organ and/or whole organism for which an agent has an affinity based on its chemical reactivity properties (Gregus & Klaassen, 2003, p. 27); site at which an agent alters cell function, regulation and/or maintenance

Biological Effect. A measurable response to dose in a molecule, cell or tissue; a functional compensatory change in morphology, physiology, growth, development and/or life span of the target as a result of stressor or other environmental influences (International Programme on Chemical Safety, 2000, p. 27)

Body Burden. The amount of agent in the body at a given instant in time (Zartarian, Ott, & Duan, 2007, p. 57)

Half-Life. Amount of time required for a given chemical concentration in target's blood or plasma to decrease by 50% (Medinsky & Valentine, 2003, p. 101)

Steady-State. A dynamic equilibrium or concentration constant when dose and exposure frequencies remain constant (adapted from Dix, 2001, p. 570)

Dose-Response Relationship. A relationship in which a change in the amount, intensity or duration of exposure is associated with a change (increase or decrease) in risk of a specified outcome (adapted from International Programme on Chemical Safety, 2001b, p. 15); cause-and-effect relationship that is precise and measurable (Eaton & Klaassen, 2003, p. 16)

Hormesis. Phenomenon where a modest stimulation of response occurs at low doses and an inhibition of response occurs at high ones; graphically depicted as an inverted *u*-shaped or *j*-shaped dose response curve; the shape difference being dependent upon the endpoint measured that is, growth or survival versus disease incidence, respectively (Calabrese & Baldwin, 2003)

Dose Threshold. A minimally-effective dose of an agent below which the probability of a target's response is zero (Rozman, Doull, & Hayes, 2001, p. 10)

Independence. Dose or effect is unaffected by the presence of another component dose or effect (Sexton & Hattis, 2007, p. 828)

Antagonism. Dose or effect is less-than-additive than the sum of individual component doses or effects (Sexton et al., 1995c, p. 436)

Additivity. Dose or effect is equivalent to the sum of individual component doses or effects, respectively (Sexton et al., 1995c, p. 436)

Inhibition. One component decreases the effect of another without having an effect itself (Agency for Toxic Substances and Disease Registry, 2001, p. 4)

Potentiation. One component increases the effect of another without having an effect itself (Agency for Toxic Substances and Disease Registry, 2001, p. 4)

Synergism. Dose or effect is greater than the sum of individual component doses or effects (Sexton et al., 1995c, p. 436)

Health

Functional compensatory capacity for stress/stressors and other environmental influences (adapted from the definition of adverse biological effect, International Programme on Chemical Safety, 2000, p. 27); an expression of the success experienced by the organism in its effort to respond adaptively to environmental challenges (Dubos, 1980, p. xvii); on a continuous scale of well-being (Linder, 1958, p. 1276)

Morbidity. Illness (manifest and non-manifest), injuries and impairments (Linder, 1958, p. 1276)

Disease. Expression of failure in an effort to respond adaptively to environmental challenges (Dubos, 1980, p. xvii)

Toxicity. The accumulation of injury over short or long periods of time which renders an organism incapable of functioning within the limits of adaptation or other forms of recovery (Rozman, Doull, & Hayes, 2001, p. 1)

Biomarker

Biochemical, molecular, genetic, immunologic or physiologic indicator of a recent or previous event in biological systems (National Research Council, 2006, p. 21)

Biomarker of Exposure. An agent, its metabolite or product of an interaction between an agent and a target molecule or cell that is measured in a compartment in a target (National Research Council, 2006, p. 21)

Metabolite. Chemical alteration of the agent produced by target's body tissue (National Research Council, 2006, p. 15)

Xenobiotic. An agent's parent compound(s) measured as a concentration per specific target medium (Wallace, 2007, p. 395)

Biomarker of Effect. A biochemical, molecular, genetic, immunologic or physiologic indicator of effect from exposure/dose (National Research Council, 2006, p. 21)

Biomarker of Susceptibility. Used to identify either individuals or populations who might have a different risk based upon differences that are inherent or acquired; this inherent category includes genetic polymorphisms (National Research Council, 2006, p. 22)

Genetic Polymorphism. Presence of a genetic abnormality, specifically two or more alleles in the DNA sequence of a particular gene, with a frequency of occurrence greater than or equal to one percent (National Research Council, 2000a, p. 90)

Vulnerability

Susceptibility to harm; the degree to which a system, subsystem or system component is likely to experience harm due to exposure to a hazard (Turner et al., 2003a, p. 8074)

Susceptibility. The combination of intrinsic and acquired attributes that alter biological response to environmental insult (Sexton, 1997, p. 264)

Health Disparity. Higher relative risk; increased comparative morbidity, premature mortality and/or diminished quality of life (adapted from Flaskerud & Winslow, 1998, p. 69)

Risk

The probability that an event will occur over some period of time (Johnson, 2007, p. 416)

Risk Assessment. Hazard identification, dose-response assessment, exposure assessment, risk characterization (U.S. Environmental Protection Agency, 2010c)

***Hazard Identification*.** The process of identifying and determining agent, exposure and outcome. "Does the agent cause the adverse effect?" (U.S. Environmental Protection Agency, 2010c)

***Dose-Response Assessment*.** Describes the quantitative relationship among agent, exposure and outcome. "What is the relationship between dose and incidence in humans?" (U.S. Environmental Protection Agency, 2010c)

***Exposure Assessment*.** The estimation or measurement of the magnitude, duration, timing and route of exposure. "What exposures are currently experienced or

anticipated under different conditions?" (U.S. Environmental Protection Agency, 2010c)

Risk Characterization. Combines information from the other elements to estimate the level of response for the identified outcome at the specific level of exposure to the agent in a defined population. "What is the estimated incidence of the adverse effect in a given population?" (U.S. Environmental Protection Agency, 2010c)

Environmental Justice. The fair treatment and meaningful involvement of all people regardless of race, color, national origin or income with respect to the development, implementation and enforcement of environmental laws, regulations and policies (U.S. Environmental Protection Agency, 2010d)

Fair Treatment. No group of people should bear a disproportionate share of the negative environmental consequences resulting from industrial, municipal and commercial operations or the execution of federal, state, local, and tribal environmental programs and policies (U.S. Environmental Protection Agency, 2010d)

Meaningful Involvement. Potentially affected community residents have an appropriate opportunity to participate in decisions about a proposed activity that will affect their environment and/or health. There are three outcomes to a meaningful involvement: 1. the public's contribution can influence the regulatory agency's decision; 2. the concerns of all participants involved will be considered in the decision-making process; and 3. the decision-makers seek out and facilitate the involvement of those potentially affected (U.S. Environmental Protection Agency, 2010d)

Precautionary Principle

"When an activity raises threats of harm to human health or the environment, precautionary measures should be taken even if some cause and effect relationships are not fully established scientifically. The Principle includes taking action in the face of uncertainty; shifting burdens of proof to those who create risks; analysis of alternatives to potentially harmful activities; and participatory decision-making methods. The precautionary principle takes the life cycle of products or chemicals into account and adds the proactive step of pre-market analysis of environmental harm" (American Nurses Association, 2003).

Major Concepts of the Precautionary Principle.

Sustainability. Conduct environmental health work in such as way that it allows future generations to meet their health needs as well;

Healthfulness. The health of humans and the environment needs to be restored, balanced, and harmonized;

Ecological Health. Field of inquiry and action to reconcile the care and health of ecosystems, populations, communities and individuals;

Interconnection. Environmental health actions have far-reaching consequences;

Respect for All Life. Environmental health work should be conducted with respect for both human and non-human life;

Global Equity. Everyone is entitled to just and equal access to the basic resources needed for an adequate and healthy life;

Respectful Participation. Respect the considered and responsible choices of stakeholders, whether individuals or organizations; and

Realistic Understanding. Environmental health ethics should be founded on a realistic understanding of the health sciences and the risks and benefits of proposed activities and investments.(Adapted from Jameton, 2005)

APPENDIX D

ASSESSING CHEMICAL INTERACTIONS

Classification for Understanding the Mechanisms of Interaction		**Weighting**
I.	well characterized mechanisms and unambiguous interpretation of the direction of interaction	1.00
II.	structure/activity relationships infer likely mechanisms and direction of interaction	0.79
III.	information on mechanisms inadequate or ambiguous with direction of interaction unclear	0.32

Classification of Toxicological Significance of the Interaction		**Weighting**
A.	directly demonstrated	1.00
B.	inferred or demonstrated in related compounds	0.79
C.	unclear	0.32

Modifiers		**Weighting**
1.	anticipated exposure duration and sequence	1.00
2.	different exposure duration and sequence	0.79
a.	*in vivo* data	1.00
b.	*in vitro* data	0.79
i.	anticipated route of exposure	1.00
ii.	different route of exposure	0.79

Weighting Factor = Product of Weighting Scores	**0.05 - 1.00**

Direction of Interaction		Direction
>	Greater-Than-Additive	+1
=	Additive	0
<	Less-Than-Additive	-1
?	Indeterminate	0

BINWOE = (Weighting Factor)(Direction Factor) = -1 through 0 to +1

Appendix D. Assessing Chemical Interactions. Adapted from *Guidance Manual for the Assessment of Joint Toxic Action of Chemical Mixtures* by the Agency for Toxic Substances and Disease Registry, 2001, p. B-4. Copyright 2001 Author.

APPENDIX E

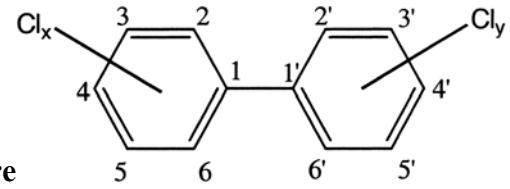
HISTORY OF NHANES

YEAR	DESCRIPTION
1949	U.S. National Committee on Vital and Health Statistics
1951	Subcommittee on National Morbidity Surveys Report
1953	Proposal for Collection of Data on Illness and Impairment
1956	National Health Survey Act (Public Law 652)
	National Center for Health Statistics (NCHS)
1956-1960	Public Health Services Report to Surgeon General
	Public Health Conference on Records and Statistics
1960-1962	First National Health Examination Survey NHES I
1963-1965	NHES II
1966-1970	NHES III
1969	White House Task Force Report on Nutrition
1971-1975	National Health and Nutrition Examination Survey
	NHANES I
1976-1980	NHANES II
1982-1984	Hispanic HANES
1988-1994	NHANES III
1999-present	Continuous NHANES

Appendix E. History of NHANES. Adapted from *Survey Overview and History* by the Centers for Disease Control and Prevention, National Center for Health Statistics, 2009h. Copyright 2009 Author.

APPENDIX F

POLYCHLORINATED BIPHENYL TERMINOLOGY



Basic PCB Molecular Structure

Congener. 209 different configurations of the basic PCB molecular structure having one to ten chlorine atoms attached to the biphenyl molecule

Homolog. PCB molecules having the same number of chlorine atoms

Isomers. Homologs with different chlorine substitution patterns

Substitution. Chlorine atom replaces hydrogen atom in the biphenyl molecule

Ortho-Substituted Positions: 2, 2′, 6, 6′

Meta-Substituted Positions: 3, 3′, 5, 5′

Para-Substituted Positions: 4, 4′

Planar or Coplanar. Two benzene rings lie in the same plane

Non-Planar or Non-Coplanar. Two benzene rings lie perpendicular to each other; degree of planarity determined by number of *ortho*-substitutions

IUPAC Number. Nomenclature assigned to a specific PCB congener for ease of reference

Dioxin-Like PCBs.

PCB 118: 2,3′,4,4′5-Pentachlorobiphenyl (mono-*ortho*-substituted planar)

Non-Dioxin PCBs.

PCB 138: 2,2′,3,4,4′,5′-Hexachlorobiphenyl (*ortho*-substituted non-planar)

PCB 153: 2,2′,4,4′,5,5′-Hexachlorobiphenyl (*ortho*-substituted non-planar)

PCB 180: 2,2′,3,4,4′,5,5′-Heptachlorobiphenyl (di-*ortho*-substituted planar)

Appendix F. Polychlorinated Biphenyl Terminology. Adapted from "Effects of polychlorinated biphenyls on the nervous system," by O. Faroon, D. Jones, and C. de Rosa, 2000, *Toxicology and Industrial Health, 16*, p. 308. Copyright Arnold, 2000.

APPENDIX G

UNIVERSITY OF RHODE ISLAND

INSTITUTIONAL REVIEW BOARD ON HUMAN SUBJECTS

IRB ACTION REPORT

The University of Rhode Island
INSTITUTIONAL REVIEW BOARD ON HUMAN SUBJECTS (IRB)
IRB ACTION REPORT

The activity indicated below has been reviewed by the University of Rhode Island Institutional Review Board (IRB) in accordance with the requirements of Title 45, Part 46 of the Code of Federal Regulations (Protection of Human Subjects), or other federal regulations as required such as 21CFR 50. The University has an approved assurance of compliance on file with the Department of Health and Human Services which covers this activity. Our assurance number is FWA 00003132. Any changes which may alter the investigational situation must be reported promptly to the IRB. Any questions concerning this action can be directed to the Office of Research Compliance at:

The Office of Research Compliance, 70 Lower College Road
University of Rhode Island, Kingston, RI 02881
telephone: (401) 874-4328
robind@uri.edu

Mailed to: IRB ID No. HU0910-084

Faculty Investigator or Sponsor:	Student Investigator or Co-PI:
Donna Schwartz Barcott Nursing White Hall	Marcella Remer Thompson 355 Grandview Road East Greenwich, RI 02818

Project Title: "Exposures to Multiple Environmental Chemicals (Lead, Methylmercury, Polychlorinated Biphensls) Among Childbearing-Aged Women in the U.S."

Date of Initial IRB Review:	Initial Review:	Date of Action:	Action:	Monitoring Interval:
12/30/2009	Category: Exempt not human subject	12/30/2009	Reviewed	duration of project
Date of InitialApproval :		*Once approved, you may not use more than ? subjects		

Waiver of elements of Informed Consent : FDA regulated study?

The IRB chair has deemed this to be not human subjects research.

IRB Chair (or Designated Member) 1/2/10 Date

Doreen B. Lawson Director of Compliance 1/5/10 Date

APPENDIX H

RHODE ISLAND DEPARTMENT OF HEALTH

INDIVIDUAL INVESTIGATOR AGREEMENT

Version Date: 1/6/2005

(9) The Investigator acknowledges and agrees to cooperate in the IRB/IEC's responsibility for initial and continuing review, record keeping, reporting, and certification for the research referenced above. The Investigator will provide all information requested by the IRB/IEC in a timely fashion.

(10) The Investigator will not enroll subjects in research under this Agreement prior to its review and approval by the IRB/IEC.

(11) Emergency medical care may be delivered without IRB/IEC review and approval to the extent permitted under applicable federal regulations and state law.

(12) This Agreement does not preclude the Investigator from taking part in research not covered by this Agreement.

(13) The Investigator acknowledges that he/she is primarily responsible for safeguarding the rights and welfare of each research subject, and that the subject's rights and welfare must take precedence over the goals and requirements of the research.

Co-Investigator Signature: _____ Date: 11/11/09

Name: Vanderslice (*Last*) Robert (*First*) R. (*Middle Initial*) Degree(s): PhD

Address: RI Department of Health 3 Capitol Hill Phone #: 401-222-7766

Providence (*City*) RI (*State/Province*) 02908-5097 (*Zip/Country*)

FWA Institutional Official (or Designee): _____ Date: 11/17/09

Name: FULTON (*Last*) JOHN (*First*) P. (*Middle Initial*) Institutional Title: IRB CHAIR

Address: RHODE ISLAND DEPARTMENT OF HEALTH. 3 CAPITOL HILL Phone #: 401-641-8806

PROVIDENCE (*City*) RI (*State/Province*) 02908-5097 (*Zip/Country*)

APPENDIX I

INDUSTRIAL UNION DEPT. v. AMERICAN PETROL. INST., 448 U.S. 607 (1980)

Industrial Union Department, AFL-CIO v. American Petroleum Institute, et al. Certiorari to the United States Court of Appeals for the Fifth Circuit No. 78-911. Argued October 10, 1979. Decided July 2, 1980. Together with No. 78-1036, Marshall, Secretary of Labor v. American Petroleum Institute et al., also on certiorari to the same court.

The Occupational Safety and Health Act of 1970 (Act) delegates broad authority to the Secretary of Labor (Secretary) to promulgate standards to ensure safe and healthful working conditions for the Nation's workers (the Occupational Safety and Health Admissions (OSHA) being the agency responsible for carrying out this authority). Section 3(8) of the Act defines an "occupational safety and health standard" as a standard that is "reasonably necessary or appropriate to provide safe or healthful employment." Where toxic materials or harmful physical agents are concerned, a standard must also comply with 6(b)(5), which directs the Secretary to "set the standard which most adequately assures, to the extent feasible, on the basis of the best available evidence, that no employee will suffer material impairment of health or functional capacity." When the toxic material or harmful physical agent to be regulated is a carcinogen, the Secretary has taken the position that no safe exposure level can be determined and that 6(b)(5) requires him to set an exposure limit at the lowest technologically feasible level that will not impair the viability of the industries

regulated. In this case, after having determined that there is a causal connection between benzene (a toxic substance used in manufacturing such products as motor fuels, solvents, detergents, and pesticides) and leukemia (a cancer of the white blood cells), the Secretary promulgated a standard reducing the permissible exposure limit on airborne concentrations of benzene from the consensus standard of 10 parts benzene per million parts of air (10 ppm) to 1 part benzene per million parts of air (1 ppm), and prohibiting dermal contact with solutions containing benzene. On pre-enforcement review, the Court of Appeals held the standard invalid because it was based on findings unsupported by the administrative record. The court concluded that OSHA had exceeded its standard-setting authority because it had not been shown that the 1 ppm exposure limit was "reasonably necessary or appropriate to provide safe and healthful employment" as required by 3(8), and that [448 U.S. 607, 608] 6(b)(5) did not give OSHA the unbridled discretion to adopt standards designed to create absolutely risk-free workplaces regardless of cost.

APPENDIX J

AUTOMOBILE WORKERS v. JOHNSON CONTROLS, INC.,

499 U.S. 187 (1991)

International Union, United Automobile, Aerospace and Agricultural Implement Workers of American, UAW, et al. v. Johnson Controls, Inc. Certiorari to the United States Court of Appeals for the Seventh Circuit No. 89-1215. Argued October 10, 1990. Decided March 20, 1991.

A primary ingredient in respondent's battery manufacturing process is lead, occupational exposure to which entails health risks, including the risk of harm to any fetus carried by a female employee. After eight of its employees became pregnant while maintaining blood lead levels exceeding that noted by the Occupational Safety and Health Administration (OSHA) as critical for a worker planning to have a family, respondent announced a policy barring all women, except those whose infertility was medically documented, from jobs involving actual or potential lead exposure exceeding the OSHA standard. Petitioners, a group including employees affected by respondent's fetal-protection policy, filed a class action in the District Court, claiming that the policy constituted sex discrimination violative of Title VII of the Civil Rights Act of 1964, as amended. The court granted summary judgment for respondent, and the Court of Appeals affirmed. The latter court held that the proper standard for evaluating the policy was the business necessity inquiry applied by other Circuits; that respondent was entitled to summary judgment because petitioners had failed to satisfy

their burden of persuasion as to each of the elements of the business necessity defense under Wards Cove Packing Co. v. Atonio, 490 U.S. 642; and that, even if the proper evaluative standard was bona fide occupational qualification (BFOQ) analysis, respondent still was entitled to summary judgment because its fetal-protection policy is reasonably necessary to further the industrial safety concern that is part of the essence of respondent's business.

Held: Title VII, as amended by the Pregnancy Discrimination Act (PDA), forbids sex-specific fetal-protection policies (pp. 197-211).

(a) By excluding women with childbearing capacity from lead-exposed jobs, respondent's policy creates a facial classification based on gender and explicitly discriminates against women on the basis of their sex under 703(a) of Title VII. Moreover, in using the words "capable of bearing children" as the criterion for exclusion, the policy explicitly classifies on the basis of potential for pregnancy, which classification must be 499 U.S. 187, 188 regarded, under the PDA, in the same light as explicit sex discrimination. The Court of Appeals erred in assuming that the policy was facially neutral because it had only a discriminatory effect on women's employment opportunities, and because its asserted purpose, protecting women's unconceived offspring, was ostensibly benign. The policy is not neutral, because it does not apply to male employees in the same way as it applies to females, despite evidence about the debilitating effect of lead exposure on the male reproductive system. Also, the absence of a malevolent motive does not convert a facially discriminatory policy into a neutral policy with a discriminatory effect. Cf. Phillips v.

Martin Marietta Corp., 400 U.S. 542. Because respondent's policy involves disparate treatment through explicit facial discrimination, the business necessity defense and its burden-shifting under Wards Cove are inapplicable here. Rather, as indicated by the Equal Employment Opportunity Commission's enforcement policy, respondent's policy may be defended only as a BFOQ, a more stringent standard than business necessity (pp. 197-200).

(b) The language of both the BFOQ provision set forth in 703(e)(1) of Title VII – which allows an employer to discriminate on the basis of sex "in those certain instances where ... sex ... is a [BFOQ] reasonably necessary to the normal operation of [the] particular business" – and the PDA provision that amended Title VII – which specifies that, unless pregnant employees differ from others "in their ability or inability to work," they must be "treated the same" as other employees "for all employment-related purposes" - as well as these provisions' legislative history and the case law, prohibit an employer from discriminating against a woman because of her capacity to become pregnant unless her reproductive potential prevents her from performing the duties of her job. The so-called safety exception to the BFOQ is limited to instances in which sex or pregnancy actually interferes with the employee's ability to perform, and the employer must direct its concerns in this regard to those aspects of the woman's job-related activities that fall within the "essence" of the particular business. Dothard v. Rawlinson, 433 U.S. 321, 333, 335; Western Air Lines, Inc. v. Criswell, 472 U.S. 400, 413. The unconceived fetuses of respondent's female employees are neither customers nor third parties whose safety is essential to the business of battery manufacturing (pp. 200-206).

(c) Respondent cannot establish a BFOQ. Fertile women, as far as appears in the record, participate in the manufacture of batteries as efficiently as anyone else. Moreover, respondent's professed concerns about the welfare of the next generation do not suffice to establish a BFOQ of female sterility. Title VII, as amended by the PDA, mandates that decisions about the welfare of future children be left to the parents 499 U.S. 187, 189 who conceive, bear, support, and raise them, rather than to the employers who hire those parents or the courts (pp. 206-207).

(d) An employer's tort liability for potential fetal injuries and its increased costs due to fertile women in the workplace do not require a different result. If, under general tort principles, Title VII bans sex-specific fetal-protection policies, the employer fully informs the woman of the risk, and the employer has not acted negligently, the basis for holding an employer liable seems remote, at best. Moreover, the incremental cost of employing members of one sex cannot justify a discriminatory refusal to hire members of that gender. See Los Angeles Dept. of Water & Power v. Manhart, 435 U.S. 702, 716-718, and n.32 (pp. 208-211).

886 F.2d 871 (CA7 1989), reversed and remanded.

Table 1
Estimated 2000 U.S. Census Coverage Error on U.S. Population (1999-2004)

Total	Population	Net		
		-0.1%		
Females Ages 16 - 49	134,502,033	134,502		
	Age			
Race-Ethnicity/Hispanic Grouping (race4cat)	20-29	Net	30-49	Net
Non-Black Females				
uncorrected	5,825,898	-1.94%	6,524,398	-1.01%
corrected	5,938,920	+113,022	6,590,294	+65,896
Black Females				
uncorrected	3,746,816	-0.66%	7,286,247	+1.28%
corrected	3,771,545	+24,729	7,192,983	-93,264

Note . Adapted from *Summary of Accuracy and Coverage Evaluation for Census 2000* by M. Mulry, 2006. Washington, D.C.: Statistical Research Division, U.S. Census Bureau.

Table 2
Childbearing-Aged Female Participants Interviewed by Age
(1999-2004)

Frequency Row Pct. Col. Pct.	1999-2000	2001-2002	2003-2004	Total
16y - 19y 192m - 239m	1,168 (27.71%) (35.50%)	1,499 (35.56%) (33.48%)	1,548 (36.73%) (37.77%)	4,215 (35.52%)
20y - 29y 240m - 359m	794 (28.04%) (21.70%)	1,164 (38.29%) (26.00%)	1,082 (35.59%) (26.40%)	3,040 (25.62%)
30y - 39y 360m - 479m	714 (28.05%) (24.14%)	1,033 (40.57%) (23.07%)	799 (31.38%) (19.50%)	2,546 (21.46%)
40y - 49y 480m - 599m	614 (29.75%) (18.66%)	781 (37.84%) (17.44%)	669 (32.41%) (16.33%)	2,064 (17.40%)
Total	3,290 (27.73%)	4,477 (37.73%)	4,098 (34.54%)	11,865 (100.00%)

Table 3
Childbearing-Aged Female Participants Examined by Age
(1999-2004)

Frequency Row Pct. Col. Pct.	1999-2000	2001-2002	2003-2004	Total
16y - 19y 192m - 239m	1,090 (26.97%) (35.56%)	1,450 (35.87%) (33.54%)	1,502 (37.16%) (37.97%)	4,042 (35.63%)
20y - 29y 240m - 359m	745 (25.40%) (24.31%)	1,121 (38.22%) (25.93%)	1,067 (36.38%) (26.97%)	2,933 (25.86%)
30y - 39y 360m - 479m	659 (27.38%) (21.50%)	1,004 (41.71%) (23.07%)	744 (30.91%) (18.81%)	2,407 (21.22%)
40y - 49y 480m - 599m	571 (29.10%) (18.63%)	748 (38.12%) (17.30%)	643 (32.77%) (16.25%)	1,962 (17.30%)
Total	3,065 (27.02%)	4,323 (38.11%)	3,956 (34.87%)	11,344 (100.00%)

95.61% interviewed were examined

Table 4
Childbearing-Aged Female Participants in Laboratory Subsample by Age
(1999-2004)

Frequency Row Pct. Col. Pct.	1999-2000	2001-2002	2003-2004	Total
16y - 19y 192m - 239m	288 (21.80%) (33.26%)	468 (35.43%) (32.01%)	565 (42.77%) (40.94%)	1,321 (35.62%)
20y - 29y 240m - 359m	235 (23.64%) (27.14%)	395 (39.74%) (27.02%)	364 (36.62%) (26.38%)	994 (26.81%)
30y - 39y 360m - 479m	207 (25.31%) (23.90%)	361 (44.13%) (24.69%)	250 (30.56%) (18.12%)	818 (22.06%)
40y - 49y 480m - 599m	136 (23.65%) (15.70%)	238 (41.39%) (16.28%)	201 (34.96%) (14.57%)	575 (15.51%)
Total	866 (23.35%)	1,462 (39.43%)	1,380 (37.22%)	3,708 (100.00%)

32.69% examined were sampled

Table 5
Childbearing-Aged Female Participants Sampled for Lead, Any Mercury and Any PCBs of Interest[1] by Age and Race-Ethnicity (1999-2004)

Laboratory Tests	Lead	Mercury 1 THg or IHg	Mercury 2 THg + IHg	PCBs 1 - 3	PCBs 4
16y - 19y 192m - 239m	1,282	10	1,245	13	1,173
20y - 29y 240m - 359m	977	11	966	6	923
30y - 39y 360m - 479m	776	12	761	7	743
40y - 49y 480m - 599m	563	7	556	1	534
Non-Hispanic White	1,678	23	1,644	7	1,571
Non-Hispanic Black	733	12	715	2	682
Mexican-	855	4	839	14	798
Other Hispanic	189	0	188	2	183
Other Racial	143	1	142	2	139
Total	3,598	40 (1.08%)	3,528	25 (0.67%)	3,373
All Hispanic	1,044	4	1,027	16	981

Table 6
Childbearing-Aged Female Participants Sampled for All Chemicals of Interest[1] and Reliable Dietary Recall[2] by Age and Race-Ethnicity (1999-2004)

Study Criteria	Incomplete Sample	Complete Sample	Unreliable Dietary Recall	Reliable Dietary Recall	Chemicals and Reliable Dietary Recall[3]
16y - 19y 192m - 239m	185	1,136	60	1,261	1,085
20y - 29y 240m - 359m	82	912	37	957	884
30y - 39y 360m - 479m	91	727	33	785	702
40y - 49y 480m - 599m	46	529	28	547	502
Non-Hispanic White	200	1,536	55	1,681	1,493
Non-Hispanic Black	95	666	51	710	623
Mexican-	93	782	44	831	745
Other Hispanic	10	182	4	188	178
Other Racial	6	138	4	140	134
Total	404 (11.98%)	3,304	158 (4.26%)	3,550	3,173
All Hispanic	103 (10.68%)	964	48 (4.71%)	1,019	923

[1]Chemicals of Interest = Lead (Pb), Total Mercury (THg), Inorganic Mercury (IHg), Polychlorinated Biphenyl (PCB) Congeners 118, 138, 153 and 180

[2]NHANES Interviewers' Rating: Reliable and Meets Minimum Criteria for Dietary Recall (DRDDRSTS = 1)

[3]Total loss to sample: 535 (14.43%)

Table 7
Childbearing-Aged Females with All Chemical Tests[1] and Reliable Dietary Recall[2] by Age and Race-Ethnicity (unweighted and weighted data 1999-2004)

	Sample (unweighted)	U.S. Population (weighted)	Percent (weighted)
16y - 19y 192m - 239m	1,085	18,510,469	14%
20y - 29y 240m - 359m	884	45,347,515	34%
30y - 39y 360m - 479m	702	36,357,837	27%
40y - 49y 480m - 599m	502	34,286,213	25%
Non-Hispanic White	1,493	97,887,544	73%
Non-Hispanic Black	623	12,747,178	9%
Mexican-American	745	8,670,576	6%
Other Hispanic	178	7,525,992	6%
Other Racial	134	7,670,743	6%
Total	3,173	134,502,033	100%
All Hispanic (race4cat)	923	16,196,568	12%

[1]Chemicals of Interest = Lead (Pb), Total Mercury (THg), Inorganic Mercury (IHg), Polychlorinated Biphenyl (PCB) Congeners 118, 138, 153 and 180

[2]NHANES Interviewers' Rating: Reliable and Meets Minimum Criteria for Dietary Recall (DRDDRSTS = 1)

Table 8
Pregnant Childbearing-Aged Participants Examined by Age (1999-2004)

Frequency Row Pct. Col. Pct.	1999-2000	2001-2002	2003-2004	Total
16y - 19y 192m - 239m	55 (28.20%) (14.75%)	65 (33.34%) (12.10%)	75 (38.46%) (18.84%)	195 (14.91%)
20y - 29y 240m - 359m	193 (28.98%) (51.74%)	285 (42.79%) (53.07%)	188 (28.23%) (47.24%)	666 (50.91%)
30y - 39y 360m - 479m	120 (27.39%) (32.17%)	186 (42.47%) (34.64%)	132 (30.14%) (33.17%)	438 (33.49%)
40y - 49y 480m - 599m	5 (55.55%) (1.34%)	1 (11.11%) (0.19%)	3 (33.34%) (0.75%)	9 (0.69%)
Total	373 (28.52%)	537 (41.05%)	398 (30.43%)	1,308 (100.00%)

11.53% examined were pregnant

Table 9
Pregnant Childbearing-Aged Participants in Laboratory Subsample by Age (1999-2004)

Frequency Row Pct. Col. Pct.	1999-2000	2001-2002	2003-2004	Total
16y - 19y 192m - 239m	17 (23.29%) (15.04%)	23 (31.51%) (11.33%)	33 (45.20%) (25.00%)	73 (16.29%)
20y - 29y 240m - 359m	63 (28.25%) (55.75%)	108 (48.43%) (53.20%)	52 (23.32%) (39.39%)	223 (49.78%)
30y - 39y 360m - 479m	33 (22.00%) (29.20%)	71 (47.33%) (34.98%)	46 (30.67%) (34.85%)	150 (33.48%)
40y - 49y 480m - 599m	0 (0.00%) (0.00%)	1 (50.00%) (0.49%)	1 (50.00%) (0.76%)	2 (0.45%)
Total	113 (25.22%)	203 (45.31%)	132 (29.47%)	448 (100.00%)

34.25% examined and pregnant were sampled

Table 10
Pregnant Childbearing-Aged Participants Sampled for Lead, Any Mercury and Any PCBs of Interest[1] by Age and Race-Ethnicity (1999-2004)

Laboratory Tests	Lead	Mercury 0 or 1 THg or IHg	Mercury 2 THg + IHg	PCBs 0 - 3	PCBs 4
16y - 19y 192m - 239m	70	3	70	12	61
20y - 29y 240m - 359m	219	4	219	19	204
30y - 39y 360m - 479m	139	11	139	16	134
40y - 49y 480m - 599m	2	0	2	0	2
Non-Hispanic White	212	11	212	23	200
Non-Hispanic Black	60	2	60	5	57
Mexican-	116	5	116	17	104
Other Hispanic	23	0	23	0	23
Other Racial	19	0	19	2	17
Total	430	18 (4.02%)	430	47 (11.72%)	401
All Hispanic	139	5	139	2	127

Table 11
Pregnant Childbearing-Aged Participants Sampled for All Chemicals of Interest[1] and Reliable Dietary Recall[2] by Age and Race-Ethnicity (1999-2004)

Study Criteria	Incomplete Laboratory Sample	Complete Laboratory Sample	Unreliable Dietary Recall	Reliable Dietary Sample	Chemicals and Reliable Dietary Recall[3]
16y - 19y 192m - 239m	12	61	1	72	61
20y - 29y 240m - 359m	19	204	8	215	198
30y - 39y 360m - 479m	16	134	9	141	130
40y - 49y 480m - 599m	0	2	0	2	2
Non-Hispanic White	23	200	7	216	198
Non-Hispanic Black	5	57	2	60	55
Mexican-	17	104	7	114	100
Other Hispanic	0	23	2	21	21
Other Racial	2	17	0	19	17
Total	47 (10.49%)	401	18 (4.02%)	430	391
All Hispanic	2 (4.76%)	40	9 (6.25%)	135	121

[1]Chemicals of Interest = Lead (Pb), Total Mercury (THg), Inorganic Mercury (IHg), Polychlorinated Biphenyl (PCB) Congeners 118, 138, 153 and 180

[2]NHANES Interviewers' Rating: Reliable and Meets Minimum Criteria for Dietary Recall (DRDDRSTS = 1)

[3]Total loss to sample: 57 (12.72%)

Table 12
Pregnant Childbearing-Aged Females with All Chemical Tests[1] and Reliable Dietary Recall[2] by Age and Race-Ethnicity
(unweighted and weighted data 1999-2004)

	Survey Sample (unweighted)	U.S. Population (weighted)	Percent (weighted)
16y - 19y 192m - 239m	61	404,786	8%
20y - 29y 240m - 359m	198	2,562,931	53%
30y - 39y 360m - 479m	130	1,687,711	35%
40y - 49y 480m - 599m	2	186,761	4%
Non-Hispanic White	198	3,035,932	63%
Non-Hispanic Black	55	713,663	15%
Mexican-American	100	487,086	10%
Other Hispanic	21	263,559	5%
Other Racial	17	341,950	7%
Total	391	4,842,189	100%
All Hispanic	121	750,645	15%

[1]Chemicals of Interest = Lead (Pb), Total Mercury (THg), Inorganic Mercury (IHg), Polychlorinated Biphenyl (PCB) Congeners 118, 138, 153 and 180

[2]NHANES Interviewers' Rating: Reliable and Meets Minimum Criteria for Dietary Recall (DRDDRSTS = 1)

Table 13
Bivariate Analyses of Season, Time of Day, Food Fast and Food Consumption on Exposure as Outcome with Two Categories
(unweighted and weighted data 1999-2004)

Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30					χ^2 *p value unweighted*	χ^2 *p value weighted*
	Exposure					
	0 and 1		2 and 3			
Season (ridexmon)						
November - April	758.00 (45.31%) 19,285,399.56 (34.39%)		674.00 (44.93%%) 29,365,343.47 (37.44%)		0.045 *0.83*	0.71 *0.40*
May - October	915.00 (54.69%) 36,779,061.09 (65.61%)		826.00 (55.07%%) 49,072,229.30 (62.56%)			
missing	n = 0 dropped					
Time of Day (time)						
Morning	784.00 (46.86%) 23,790,488.75 (42.43%)		740.00 (49.33%) 39,470,876.92 (50.32%)			
Afternoon	529.00 (31.62%) 18,079,471.03 (32.25%)		497.00 (33.13%) 25,478,512.93 (32.48%)		7.96 *0.019*	1.79 *0.18*
Evening	360.00 (21.52%) 14,194,500.87 (25.32%)		263.00 (17.53%) 13,488,182.92 (17.20%)			
(missing)	n = 0 dropped					
Food Fast prior 24 hours (fdfstcat)						
more than 24h	* (0.00%) 218,749.31 (0.39%)		* (0.00%) 278,855.06 (0.36%)			
16 to <24	224.00 (13.39%) 7,576,698.02 (13.51%)		188.00 (12.53%) 9,937,588.75 (12.67%)			
8 to <16	850.00 (50.81%) 28,726,744.41 (51.24%)		820.00 (54.67%) 434,029,49.11 (55.33%)		9.68 *0.085*	0.69 *0.63*
4 to <8	434.00 (25.94%) 16,101,400.57 (28.72%)		383.00 (25.53%) 21,902,865.77 (27.92%)			
1 to <4	140.00 (8.37%) 3,150,722.27 (5.62%)		89.00 (5.93%) 2,454,735.57 (3.13%)			
< 1 hr	* (0.00%) 290,146.07 (0.52%)		* (0.00%) 460,578.49 (0.59%)			
(missing)	n = 0 dropped					
Food Consumption prior 24-hours (fdc3cat)						
usual R	976.00 (58.48%) 33,526,112.21 (59.92%)		917.00 (61.13%) 51,357,582.93 (65.48%)			
less than usual	462.00 (27.68%) 15,416,115.31 (27.55%)		399.00 (26.60%) 19,344,149.41 (%)		6.36 *0.095*	0.687 *0.51*
more than usual	231.00 (13.84%) 7,007,328.05 (12.52%)		184.00 (12.27%) 7,735,840.43 (9.86%)			
(missing)	n = 4 dropped					

Table 14. Percent Bias in Estimating the Geometric Mean and Standard Deviation by Imputation Method

Method	% Nondetectable	Percent Bias in Estimating the Geometric Mean				Percent Bias in Estimating the Geometric Standard Deviation			
		Geometric Standard Deviation				Geometric Standard Deviation			
		1.5	2.0	2.5	3.0	1.5	2.0	2.5	3.0
	0	0.05	-0.04	-0.2	0.02	0.12	-0.04	-0.01	-0.05
1	15	0.4	0.5	0.0	-0.4	0.3	0.3	-0.3	0.4
2		-7.2	-5.2	-3.2	-1.8	13.4	8.1	4.2	1.0
3		-2.2	-0.1	1.9	3.4	2.9	-1.0	-4.2	-6.6
1	30	-0.3	0.2	-0.1	0.2	-0.3	0.3	-0.6	-0.4
2		-12.3	-7.4	-3.8	11.2	16.8	7.2	0.4	-4.5
3		-2.6	2.8	6.9	11.2	1.7	-5.8	-11.4	-15.4
1	45	0.3	-0.1	0.2	0.2	0.4	-0.3	0.3	0.4
2		-16.0	-7.2	0.3	6.1	15.3	1.9	-6.9	-13.5
3		-1.8	8.4	17.1	24.1	-1.5	-12.2	-19.4	-24.8
1	60	-	-	-	-	-	-	-	-
2		-17.9	-4.2	8.4	19.8	10.1	-6.1	-16.6	-24.2
3		1.1	17.9	33.4	47.3	-6.1	-19.3	-27.9	-34.4

Note : 1 = Hald, 2 = Nehls & Akland, 3 = Hornung & Reed. Adapted from "Estimation of Average Concentration in the Presence of Nondetectable Values," by R. Hornung and L. Reed, 1990, *Applied Occupational and Environmental Hygiene. 5* (1), p. 49-50. Copyright Taylor and Francis, Ltd.

Table 15
Limits of Detection and Imputed Values of Xenobiotic Blood Levels for Hornung and Reed (1990) Calculations: NHANES Sample Population by Each Two-Year Period[1]

Dependent Variable Components	1999 - 2000	2001-2002	2003-2004
Lead LBXBPB Non-Detectables[2] = 385/45,195 (0.9%) Detectables = 44,810/45,195 (99.1%)	LBXBPB = 0.3/√2 = 0.212 or 0.2 µg/dl Non-Detectables[2] = 75/12,582 (0.6%) Mode of Detectable Values Only: 0.80 GStandard Deviation of Log Detectable Values: 0.65 Skewness/GSkewness: 8.97/0.397	LBXBPB = 0.3/√2 = 0.212 or 0.2 µg/dl Non-Detectables[2] = 271/16,828 (1.6%) Mode of Detectable Values Only: 0.90 GStandard Deviation of Log Detectable Values: 0.67 Skewness/GSkewness: 7.29/0.44	LBXBPB = 0.3/√2 = 0.212 or 0.2 µg/dl Non-Detectables[2] = 39/15,785 (0.25%) Mode of Detectable Values Only: 0.90 GStandard Deviation of Log Detectable Values: 0.65 Skewness/GSkewness: 9.32/0.53
Total Mercury LBXTHG Non-Detectables[2] = 1,233/24,362 (5.1%) Detectables = 23,129/24,362 (94.9%)	LBXTHG = 0.137/√2 = 0.097 or 0.1 µg/dl Non-Detectables[2] = 308/3,605 (8.5%) Mode of Detectable Values Only: 0.30 GStandard Deviation of Log Detectable Values: 1.01 Skewness/GSkewness: 4.92/0.47	LBXTHG = 0.1/√2 = 0.07 µg/dl Non-Detectables[2] = 262/4,972 (5.3%) Mode of Detectable Values Only: 0.20 GStandard Deviation of Log Detectable Values: 1.01 Skewness/GSkewness: 5.17/0.22	LBXTHG = 0.14/√2 = 0.099 or 0.1 µg/dl Non-Detectables[2] = 663/15,785 (4.2%) Mode of Detectable Values Only: 0.30 GStandard Deviation of Log Detectable Values: 1.01 Skewness/GSkewness: 5.85/0.31
Inorganic Mercury LBXIHG Non-Detectables[2] = 20,029/23,929 (83.7%) Detectables = 3,900/23,929 (16.3%)	LBXIHG = 0.446/√2 = 0.315 or 0.3 µg/dl Non-Detectables[2] = 3,491/3,582 (97.5%) Mode of Detectable Values Only: 0.50 GStandard Deviation of Log Detectable Values: 0.83 Skewness/GSkewness: 3.26/2.06	LBXIHG = 0.396/√2 = 0.28 µg/dl Non-Detectables[2] = 4,641/4,901 (94.7%) Mode of Detectable Values Only: 0.50 GStandard Deviation of Log Detectable Values: 0.45 Skewness/GSkewness: 8.86/3.08	LBXIHG = 0.446/√2 = 0.315 or 0.3 µg/dl Non-Detectables[2] = 11,897/15,446 (77.0%) Mode of Detectable Values Only: 0.40 GStandard Deviation of Log Detectable Values: 0.36 Skewness/GSkewness: 41.81/2.09
PCB 118 LBX118 Non-Detectables[2] = 4,749/12,261 (38.7%) Detectables = 7,512/12,261 (61.3%)	LBD118LC = 1[3] Non-Detectables[2] = 2,181/3,322 (65.65%) Mode of Detectable Values Only: 0.06 GStandard Deviation of Log Detectable Values: 0.74 Skewness/GSkewness: 8.34/0.69	LBD118LC = 1[3] Non-Detectables[2] = 2,568/4,922 (52.17%) Mode of Detectable Values Only: 0.04 GStandard Deviation of Log Detectable Values: 0.74 Skewness/GSkewness: 7.26/0.91	LBD118LC = 1[3] Non-Detectables[2] = 0/4,017 (0%) Mode of Detectable Values Only: 0.01 GStandard Deviation of Log Detectable Values: 0.95 Skewness/GSkewness: 12.29/1.15
PCB 138 LBX138 Non-Detectables[2] = 3,473/12,243 (28.4%) Detectables = 8,770/12,243 (71.6%)	LBD138LC = 1[3] Non-Detectables[2] = 2,318/3,326 (69.7%) Mode of Detectable Values Only: 0.16 GStandard Deviation of Log Detectable Values: 0.58 Skewness/GSkewness: 7.58/0.90	LBD138LC = 1[3] Non-Detectables[2] = 1,155/4,880 (23.7%) Mode of Detectable Values Only: 0.05 GStandard Deviation of Log Detectable Values: 0.92 Skewness/GSkewness: 4.29/0.50	LBD138LC = 1[3] Non-Detectables[2] = 4,037/4,037 (0%) Mode of Detectable Values Only: 0.02 GStandard Deviation of Log Detectable Values: 1.13 Skewness/GSkewness: 8.81/0.58
PCB 153 LBX153 Non-Detectables[2] = 3,019/12,265 (24.6%) Detectables = 9,246/12,265 (75.4%)	LBD153LC = 1[3] Non-Detectables[2] = 2,156/3,313 (65.1%) Mode of Detectable Values Only: 0.37 GStandard Deviation of Log Detectable Values: 0.59 Skewness/GSkewness: 6.83/0.65	LBD153LC = 1[3] Non-Detectables[2] = 863/4,921 (17.5%) Mode of Detectable Values Only: 0.03 GStandard Deviation of Log Detectable Values: 1.01 Skewness/GSkewness: 3.88/0.34	LBD153LC = 1[3] Non-Detectables[2] = 4,031/4,031 (0%) Mode of Detectable Values Only: 0.03 GStandard Deviation of Log Detectable Values: 1.19 Skewness/GSkewness: 7.49/0.45
PCB 180 LBX180 Non-Detectables[2] = 3,801/12,251 (31.0%) Detectables = 8,450/12,251 (69%)	LBD180LC = 1[3] Non-Detectables[2] = 2,043/3,304 (61.8%) Mode of Detectable Values Only: 0.26 GStandard Deviation of Log Detectable Values: 0.65 Skewness/GSkewness: 4.70/0.30	LBD180LC = 1[3] Non-Detectables[2] = 1,719/4,908 (35.0%) Mode of Detectable Values Only: 0.03 GStandard Deviation of Log Detectable Values: 0.96 Skewness/GSkewness: 3.58/0.19	LBD180LC = 1[3] Non-Detectables[2] = 39/4,039 (0.9%) Mode of Detectable Values Only: 0.01 GStandard Deviation of Log Detectable Values: 1.37 Skewness/GSkewness: 5.18/0.26

[1]with no missing data [2]Non-Detectables = Below Level of Detection [3]PCBs' Level of Detection is Sample-Specific G = Geometric Values

Table 16
Initial Calculations for Methylmercury (MeHg) Blood Levels NHANES Sample Population by Each Two-Year Period

	1999-2000					2001-2002					2003-2004				1999-2004
MeHg < 0	IHg^{1} (<LoD) 0.3	IHg (LoD) 0.446	IHg (>LoD) > 0.446	Subtotal	MeHg < 0	IHg^{1} (<LoD) 0.28	IHg (LoD) 0.396	IHg (>LoD) > 0.396	Subtotal	MeHg < 0	IHg^{1} (<LoD) 0.3	IHg (LoD) 0.446	IHg (>LoD) > 0.446	Subtotal	Total MeHg < 0
THg^{1} (< LoD) 0.1	302	0	2	304	THg^{1} (< LoD) 0.07	254	0	5	259	THg^{1} (< LoD) 0.1	597	14	30	641	1,204
THg (LoD) 0.137	258	1	4	263	THg (LoD) 0.1	192	0	1	193	THg (LoD) 0.137	997	70	76	1,143	1,599
THg >IHgLoD - IHg >LoD > 0.137 to 0.446	0	0	0	0	THg >IHgLoD - IHg >LoD > 0.1 to 0.396	415	8	27	450	THg >IHgLoD - IHg >LoD > 0.137 to 0.446	710	134	246	1,090	1,540
THg (>IHgLoD) > 0.446	0	0	0	0	THg (>IHgLoD) > 0.396	0	0	5	5	THg (>IHgLoD) > 0.446	0	0	60	60	65
Total	560	1	6	567	Total	861	8	38	907	Total	2,304	218	412	2,934	4,408 (18.44%)

	1999-2000					2001-2002					2003-2004				1999-2004
MeHg = 0	IHg^{1} (<LoD) 0.3	IHg (LoD) 0.446	IHg (>LoD) > 0.446	Subtotal	MeHg = 0	IHg^{1} (<LoD) 0.28	IHg (LoD) 0.396	IHg (>LoD) > 0.396	Subtotal	MeHg = 0	IHg^{1} (<LoD) 0.3	IHg (LoD) 0.446	IHg (>LoD) > 0.446	Subtotal	Total MeHg = 0
THg^{1} (< LoD) 0.1	0	0	0	0	THg^{1} (< LoD) 0.07	0	0	0	0	THg^{1} (< LoD) 0.1	0	0	0	0	0
THg (LoD) 0.137	0	0	0	0	THg (LoD) 0.1	0	0	0	0	THg (LoD) 0.137	0	0	0	0	0
THg >IHgLoD - IHg >LoD > 0.137 to 0.446	292	0	0	292	THg >IHgLoD - IHg >LoD > 0.1 to 0.396	0	1	0	1	THg >IHgLoD - IHg >LoD > 0.137 to 0.446	1,232	89	0	1,321	1,614
THg (>IHgLoD) > 0.446	0	0	0	0	THg (>IHgLoD) > 0.396	0	0	6	6	THg (>IHgLoD) > 0.446	0	0	87	87	93
Total	292	0	0	292	Total	0	1	6	7	Total	1,232	89	87	1,408	1,707 (7.14%)

	1999-2000					2001-2002					2003-2004				1999-2004
MeHg > 0	IHg^{1} (<LoD) 0.3	IHg (LoD) 0.446	IHg (>LoD) > 0.446	Subtotal	MeHg > 0	IHg^{1} (<LoD) 0.28	IHg (LoD) 0.396	IHg (>LoD) > 0.396	Subtotal	MeHg > 0	IHg^{1} (<LoD) 0.3	IHg (LoD) 0.446	IHg (>LoD) > 0.446	Subtotal	Total MeHg > 0
THg^{1} (< LoD) 0.1	0	0	0	0	THg^{1} (< LoD) 0.07	0	0	0	0	THg^{1} (< LoD) 0.1	0	0	0	0	0
THg (LoD) 0.137	0	0	0	0	THg (LoD) 0.1	0	0	0	0	THg (LoD) 0.137	0	0	0	0	0
THg >IHgLoD - IHg >LoD > 0.137 to 0.446	238	0	0	238	THg >IHgLoD - IHg >LoD > 0.1 to 0.396	786	0	0	786	THg >IHgLoD - IHg >LoD > 0.137 to 0.446	1,146	0	0	1,146	2,170
THg (>IHgLoD) > 0.446	2,396	6	75	2,477	THg (>IHgLoD) > 0.396	2,983	28	179	3,190	THg (>IHgLoD) > 0.446	7,214	901	1,838	9,953	15,620
Total	2,634	6	75	2,715	Total	3,769	28	179	3,976	Total	8,360	901	1,838	11,099	17,790 (74.42%)

MeHg = (THg-IHg) THg = Total Mercury LoD = Level of Detection n = 23,905

IHg = Inorganic Mercury [1] = NHANES' imputed value = LoD/√2

Table 17
Operational Definitions of Independent Variables

Variable Name [R] = Reference Group (missing) = *not* separate category	Operational Definitions
Susceptibility-Related Attributes	
Age	
Age (age4cat)	age in months (RIDAGEMN)
16y - 19y[R]	192 - 239 months
20y - 29y	240 - 359 months
30y - 39y	360 - 479 months
40y - 49y	480 - 599 months
(missing)	n = 0 dropped
Health Status	
Perceived Health Status (huq2cat)	"Would you say your health in general is ..." (HUQ010)
excellent, very good, good[R]	
fair, poor	
(missing)	n = 1 dropped
Co-Morbidities (CCMS3cat)	"Has a doctor or other health professional ever told you that you have…" (See Table 23)
(missing)	recoded as no
Iron Deficiency (FeD2cat)	(See Table 24)
Treatment for Iron Deficiency past 3 mo (FeTx2cat)	"During the past 3 months, have you been on treatment for anemia, sometimes called 'tired blood' or 'low blood'? [include diet, iron pills, iron shots, transfusions as treatment]" (MCQ053)
yes	
no[R]	
(missing)	n = 1 dropped
Iron Deficiency and Treatment (FeDTx)	(FeD2cat * FeTx2cat)
(missing)	n = 1 dropped
Health Insurance (hi2cat)	"What kind of health insurance or health care coverage do you have? Include those that pay for only one type of service (nursing home care, accidents, or dental care). Exclude private plans that only provide extra cash while hospitalized. If {you have/he/she has} more than one kind of health insurance, just tell me about the first kind." (HID010, HID030)
private[R]	plan from employer, purchased directly from insurance, state/local government or community programs
public	medicare, medi-gap, medicaid, CHIP, military, tricare, Indian health service, state plan, other gov't
none	
missing	n = 73
Regular Source of Healthcare (hp2cat)	Is there a place that you usually go when you are sick or you need advice about your health?(HUQ030)
yes[R]	one or more places
no	
(missing)	n = 0
Source of Healthcare (hcsre)	"What kind of place do you go to most often: is it a clinic, doctor's office, emergency room, or some other place?" (HUQ040)
healthcare provider[R]	doctor's office or HMO
clinic	clinic or health center
ER or none	emergency room, hospital outpatient department, other unnamed source or none
missing	n = 48
Nutritional Status	
Food Security (food2cat)	household food security (FSDHH) + adult food security (FSDAD) + child food security (FSDCH)
food secure[R]	household fully or marginally secure or exceeds poverty income ratio (INDFMPIR ≥ 5);
food insecure	household insecure without hunger or household insecure with hunger
missing	n = 142
Body Mass Index (bmi30cat)	(BMXBMI)
<30.0[R] underweight, normal, overweight	underweight 00.0 to < 18.5 normal 18.5 to < 25.0 overweight 25.0 to < 30.0
30.0+ obese	obese I 30.0 to < 35.0 obese II 35.0 to < 40.0 obese III 40.0 or more
missing	n = 39

Table 17
Operational Definitions of Independent Variables

Variable Name R = Reference Group . = missing or (missing) = *not* separate category	Operational Definitions
Fat Intake/AMDR (fat3cat)	(fat intake 24h * 9 g/cal) /(total caloric intake 24h) (DR1TTFAT*9/DR1TKCAL) See Table 25
recommended or less[R]	0.00 to 0.35
more than recommended	> 0.35
(missing)	n = 0
Protein Intake in past 24h/AMDR (prot3cat)	(protein intake 24h * 4 g/cal) /(total caloric intake 24h) (DR1TPROT*4/DR1TKCAL) See Table 25
recommended or more[R]	0.10 or more
less than recommended	0.00 to < 0.10
(missing)	n = 0
Iron Intake in past 24h/RDA (iron2cat)	(dr1tiron/RDA) See Table 25
recommended or more[R]	≥ 1.0
less than recommended	< 1.0
(missing)	n = 0
Calcium Intake in past 24h/RDA (calc2cat)	(dr1tcalc/RDA) See Table 25
recommended or more[R]	≥ 1.0
less than recommended	< 1.0
(missing)	n = 0
Selenium Intake in past 24h/RDA (sele2cat)	(dr1tsele/RDA) See Table 25
recommended or more[R]	≥ 1.0
less than recommended	< 1.0
(missing)	n = 0
Reproductive Status	
Current Pregnancy (pregnant)	urine pregnancy test (URXPREG) and trimester of pregnancy (RHD152)
pregnant	urine pregnancy test (URXPREG = 1) OR if urine pregnancy test (URXPREG = .) AND trimester of pregnancy (RHD152 = 4, 5, 6, 7, 8, or 9)
not pregnant[R]	urine pregnancy test (URXPREG = 2)
missing	urine pregnancy test (URXPREG = .) OR if urine pregnancy test (URXPREG = .) AND trimester of pregnancy (RHD152 = 1, 2, or 3) unknown n = 141
Trimester of Pregnancy (tripcorr)	"Think that you are pregnant now?" (RHQ140 = 1) THEN "What month of pregnancy are you in?" (RHD152) and urine pregnancy test (URXPREG=1)
1st trimester	trimester of pregnancy (RHD152 = 1, 2, or 3) AND urine pregnancy test (URXPREG ≠ 2) OR urine pregnancy test (URXPREG = 1) AND trimester of pregnancy (RHD152 ≠ 4-9)
2nd trimester	trimester of pregnancy (RHD152 = 4, 5, 6)
3rd trimester	trimester of pregnancy (RHD152 = 7, 8, 9)
not pregnant[R]	urine pregnancy test (URXPREG = 2 or .) OR trimester of pregnancy (RHD152 = .) if unknown, recoded as not pregnant
Ever Pregnant (tprg2cat)	"The next questions are about your pregnancy history. {Have you ever been pregnant? Please include current pregnancy, live births, miscarriages, stillbirths, tubal pregnancies and abortions." (RHQ131)
never pregnant[R]	(RHQ131 = 2) OR (PREGNANT = 2)
one or more pregnancies	(RHQ131 = 1) OR (PREGNANT = 1)
(missing)	recoded as never pregnant
Live Births (live)	"How many of your pregnancies resulted in a live birth?" (RHD170)
no live births[R]	
one or more live births	
missing	recoded as no live births
Ever Breastfed (brstfda)	"Are you now breastfeeding a child?" (RHQ200) AND/OR "How many of your children did you breastfeed for at least one month?" (RHD230)
never breastfed[R]	
breastfed more than one month or currently	
missing	recoded as never breastfed

Table 17
Operational Definitions of Independent Variables

Variable Name R = Reference Group . = missing or (missing) = *not* separate category	Operational Definitions
Exposure-Related Attributes	
Acculturation	
Birthplace (born2cat)	"In what country were you born?" (DMDBORN)
U.S.R	
outside U.S.	
(missing)	n = 0
Years in U.S. (yrus5)	"In what month and year did you come to the United States to stay?" (DMDYRSUS)
born in U.S.R	(DMDBORN = 1)
five or more years	survey year - year arrived in U.S. (SDDSRVYR - DMDYRSUS)
less than five years	survey year - year arrived in U.S. (SDDSRVYR - DMDYRSUS)
(missing)	n = 6 dropped
Language Spoken at Home (lang2cat)	In general, what language(s) do you speak at home? (ACD010) For Hispanics Only: Would you say you speak/read . . . (ACQ020)
EnglishR	Only English OR English AND another language OR more English than Spanish OR both English and Spanish equally
Other	one or more languages (neither English) OR more Spanish than English
(missing)	n = 2 dropped
U.S. Citizenship (usczn2cat)	"Are you a citizen of the United States?" [Information about citizenship is being collected by the U.S. Public Health Service to perform health related research. Providing this information is voluntary and is collected under the authority of the Public Health Service Act. There will be no effect on pending immigration or citizenship petitions.]
U.S. citizenR	
non-U.S. citizen	
(missing)	n = 1 dropped
Dietary Consumption	
Seafood Eaten in Past 30 Days (smpw2cat)	see below fish eaten in past 30 days (fish2cat) AND shellfish eaten in past 30 days (shell2cat)
noneR	
any	
(missing)	recoded as none
Fish Eaten in Past 30 Days (fish2cat)	"During the past 30 days did you eat any types of fish listed on this card? Include any foods that had fish in them such as sandwiches, soups, or salads." (DRD360 AND DRD370)
noneR	
any	
(missing)	recoded as none
Shellfish Eaten in Past 30 Days (shell2cat)	"During the past 30 days did you eat any types of shellfish listed on this card? Include any foods that had shellfish in them such as sandwiches, soups, or salads." (DRD340 AND DRD350)
noneR	
any	
(missing)	recoded as none
Tap Water Consumed Prior 24h (tap2kct)	"How much of the plain water you drank was home tap water (1 gram = 1 milliliter)?" (DR1_330)
noneR	
< 2,000 ml	
2,000+ ml	
missing	n = 211
Alcohol Consumption	
Alcohol Consumption (retohuse)	
never, seldom drinkerR *including 16-19 y/o*	Never: "In your entire life, have you had at least 12 drinks of any type of alcoholic beverage?"(ALQ101 = 2) Included are liquor (such as whiskey or gin), beer, wine, wine coolers, and any other type of alcoholic beverage. Seldom: "In any one year, have you had at least 12 drinks of any type of alcoholic beverage? By a drink, I mean a 12 oz. beer, a 4 oz. glass of wine, or an ounce of liquor." (ALQ100 = 2) information confidential; assumed never or seldom drinker
drinker	"In any one year, have you had at least 12 drinks of any type of alcoholic beverage?"(ALQ100 = 1)
heavy drinker	"In the past 12 months, on how many days did you have five or more drinks of any alcoholic beverage?" (ALQ140Q/ALQ140U > 1) Was there ever a time or times in your life when you drank five or more drinks of any kind of alcoholic beverage almost every day?" (ALQ150 = 1)
missing	n = 145

Table 17
Operational Definitions of Independent Variables

Variable Name R = Reference Group . = missing or (missing) = *not* separate category	Operational Definitions
Tobacco Use	
Self-Reported Tobacco Use (tobuse)	cigarettes, pipe, cigars, snuff, chaw, other nicotine products ever/never (smq020 + smq120 + smq150 + smq180 + smq210 + smq840) current/former (smq040 + smq140 + smq170 + smq200 + smq230 + smq840)
age restricted[R]	(smq020 = missing + age4cat = 1)
never	
former	
current	
missing	n = 15 dropped
Serum Cotinine (cot3cat)	(lbxcot)
< 1.0 ng/ml[R]	
1.0 - 10.0 ng/ml	
> 10.0 ng/ml	
(missing)	n = 15 dropped
Environmental Tobacco Smoke (ETS)	"I would now like to ask you a few questions about smoking. Does anyone who lives here smoke cigarettes, cigars, or pipes anywhere inside this home?" (SMD410 = 1) "What is the total number of (cigarette, cigar, pipe) smokers in home?" (SMD415 > 0) "At your job or business, how many hours per day can you smell the smoke from other people's cigarettes, cigars, and/or pipes?" (OCQ290G ≥ 1)
no ETS[R]	
ETS at home or work	
ETS at home and work	
(missing)	recoded as no ETS
Residence	
Tap Water Source (h2os2cat)	"What is the source of tap water in this home? Is it a private or public water company, a private or public well, or something else?" (HOQ070)
public[R]	municipality or company
private	well or something else
missing	n = 81
Residential Tap Water Treatment (h2ox2cat)	"Are any of these water treatment devices used in your home (listed)?" (HOQ080)
yes	
no[R]	
missing	n = 71
Type of Residence (res3cat)	"I'd like to ask you a few questions about your home. Is your home . . ." (HOD011)
attached or detached house[R]	
mobile home or trailer	
all other types	
(missing)	recoded as all other types
Age of Residence (resb60cat)	"When was this {mobile home/house/building} originally built?" (HOD040)
1960 or newer[R]	
older than 1960	
missing/unknown	n = 812 (25.59%)
Age of Residence (resb78cat)	"When was this {mobile home/house/building} originally built?" (HOD040)
1978 or newer[R]	
older than 1978	
missing/unknown	n = 812 (25.59%)
Resident Status (resd3cat)	"Is this {mobile home/house/apartment} owned, being bought, rented, or occupied by some other arrangement by you or someone else in your family?" (HOQ065)
own[R]	
rent	
other	
(missing)	recoded as other
Years at Current Residence (re5yrcat)	"How many years {have you/has your family} lived at this address?" (HOD060/5)
more than five years[R]	
five years or less	
missing	n = 53
Household Size (hsize)	Total number of people in the Household (DMDHHSIZ)
four persons or less[R]	
more than four persons	

Table 17
Operational Definitions of Independent Variables

Variable Name R = Reference Group . = missing or (missing) = *not* separate category	Operational Definitions
Rooms in Residence (rm3cat)	"How many rooms are in this home? Count the kitchen but not the bathroom." (HOD050)
7+ rooms[R]	
4-6 rooms	
1-3 rooms	
missing	n = 71
Occupation	
Current Occupation (cocc2cat)	"What kind of work were you doing?" (OCD240) See Tables 23 and 24
not working[R]	
management, professional & sales	
services & goods	
(missing)	n = 0
Time in Current Employment (cjt)	"About how long have you worked for {EMPLOYER} as a(n) {OCCUPATION)?" (OCD270/5)
not working[R]	
less than five years	
five or more years	
(missing)	n = 0
Total Hours Worked Prior Week (hrwk)	"How many hours did you work last week at all jobs or businesses?" (OCD180/35) "Do you usually work 35 hours or more per week in total at all jobs or businesses?" (OCD210)
not employed[R]	employment status (emp3cat)
less than 35 hours	
35+ hours	
(missing)	n = 2 dropped
Longest Held Occupation (locc2cat)	"Thinking of all the paid jobs or businesses you ever had, what kind of work were you doing the longest? (OCD390) See Table
not applicable[R]	
management, professional & sales	
services & goods	
(missing)	n = 0
Time in Longest Employment (ljt)	"About how long did {you/SP} work at that job or business?" (OCD395)
not applicable[R]	
less than five years	
five or more years	
(missing)	n = 0
Socioeconomic Factors	
Education	
Highest Education (educ2)	"What is the highest grade or level of school {you have/SP has} completed or the highest degree {you have/s/he has} received?" (DMDEDUC2, DMDEDUC3)
high school diploma, GED or higher[R]	
less than high school diploma	
(missing)	n = 1 dropped
Employment	
Employment Status (emp3cat)	"In this part of the survey I will ask you questions about your work experience. Which of the following were you doing last week… (OCD150)
employed	
not employed[R]	
(missing)	n = 2 dropped
Reason for Unemployment (unem2cat)	"What is the main reason you did not work last week?" (OCD380)
working[R]	employment status (emp3cat)
voluntary unemployment	taking care of house or family; going to school; retired
involuntary unemployment	unable to work for health reasons; on layoff; disabled; other
missing	n = 101
Work History (wkcp)	longest held occupation (locc2cat) AND current occupation (cocc2cat) AND employment status (emp3cat)
never employed[R]	
currently employed	
employed in the past but not currently	
employed now and in the past	
(missing)	recoded as never employed

Table 17
Operational Definitions of Independent Variables

Variable Name R = Reference Group . = missing or (missing) = *not* separate category	Operational Definitions
Income	
U.S. Poverty Threshold (pov2cat)	Family Poverty Income Ratio (INDFMPIR) See Table 25
more than 1.00[R]	
1.00 or less	
missing	n = 216
Marital Status	
Marital Status (marr3cat)	"Are you now married, widowed, divorced, separated, never married or living with a partner?"
married or living with partner	
widowed, divorced or separated	
never married[R]	
missing	n = 77
Race-Ethnicity	
Race-Ethnicity (race5cat)	"Which one of these groups would you say best represents your race?" (RIDRETH1)
Non-Hispanic White[R]	
Non-Hispanic Black	
Mexican American	
Other Hispanic	
Asian, Native American, Pacific Islander & Multi-Racial	includes "cannot choose 1 race"
(missing)	n = 0
Race-Ethnicity/Hispanic Grouping (race4cat)	"Which one of these groups would you say best represents your race?" (RIDRETH1)
Non-Hispanic White[R]	
Non-Hispanic Black	
Hispanic	Mexican American AND Other Hispanic
Asian, Native American, Pacific Islander & Multi-Racial	includes "cannot choose 1 race"
(missing)	n = 0

Table 18
Composition and Frequencies of Independent Variables that Comprise the Charleson Co-Morbidity Index (1999-2004)

Index Points Per Diagnosis Sample Frequency * = cell size less than 30	0	1	2	3	6
Myocardial Infarction (MCQ160E)	3,166.00	*			
Congestive Heart Failure (MCQ160B)	3,169.00	*			
Cerebrovascular Disease (MCQ160F)	3,156.00	*			
Chronic Pulmonary Disease asthma + chronic bronchitis + emphysema (MCQ010+MCQ030) + (MCQ160K+MCQ170K) + (MCQ160G)	2,920.00	253.00			
Diabetes (DIQ010)	3,110.00	63.00			
Connective Tissue Disease[1]					
Peripheral Vascular Disease[1]					
Liver Disease - mild CTP[2] score + MCQ160L + MCQ170L = 5 points or less	3,173.00	0.00			
Peptic Ulcer[3] (MCQ200 1 = Yes 2 = No)	3,162.00	*			
Dementia[1,4]					
Renal Disease moderate to severe kidney failure/dialysis or serum creatinine ≤ 3.0 mg/dL (KIQ020 or LBXSCR)	3,171.00		*		
Diabetes with End-Organ Disease diabetes with retinopathy or dialysis (DIQ010+ DIQ080 or DIQ090 or KIQ200)	3,158.00		*		
Hemiplegia/Paraplegia[1]					
Solid Tumor with no metastases within five years[5] (MCQ220 + MCQ230A-DD) + (RIDAGEYR-MCQ240A-DD)	3,152.00		*		
History of Leukemia (MCQ220 + MCQ230 A/ B/ C or D = 21)	3,172.00		*		
History of Lymphoma (MCQ220 + MCQ230 A/ B/ C or D = 24)	3,172.00		*		
Liver Disease - moderate to severe CTP2 score + MCQ160L + MCQ170L = 6 points or more	3,173.00			0.00	
Solid Tumor with metastases[5]	3,168.00				*
AIDS-Complex HIV positive + CD4 < 200 cells/mm^3	3,171.00				*

References

Charlson, Charlson, Peterson, Marinopoulos, Briggs, & Hollenberg, (2008). The Charlson comorbidity index is adapted to predict costs of chronic disease in primary care patients. *Journal of Clinical Epidemiology, 61* , 1234-1240. doi:10:1016/j.jclinepi.2008.01.006

Charlson, Pompei, Ales, & MacKenzie, (1987). A new method of classifying prognostic comorditiy in longitudinal studies: development and validation. *Journal of Chronic Diseases, 40* , 377, 382-383.

Table 18
Composition and Frequencies of Independent Variables that Comprise the Charleson Co-Morbidity Index (1999-2004)

[1]Information Not Available in NHANES (1999 - 2004)					
Score Points Sample Frequency * = cell size less than 30	1	2	3	4	Total
[2]Childs-Turcotte-Pugh Score (CTP) serum albumin + serum total bilirubin + (prothrombin time[1] + ascites[1] + hepatic encephalopathy[1])		2,941.00	227.00	*	3,173.00
Serum Albumin (sal3cat)					
more than 35.0 mg/dl	2,941.00	*	*		2,957.00
28.0 - 35.0 mg/dl	0.00	212.00	0.00		212.00
less than 28.0 mg/dl	0.00	0.00	*		*
Serum Total Bilirubin (stb3cat)					
less than 34.0	2,799.00	302.00	56.00		3,157.00
34.0 - 50.0	*	0.00	0.00		*
more than 50.0	0.00	*	0.00		*
Prothrombin Time[1] 1 (13.0 - 16.9) or 2(17.0 - 19.0) or 3(> 19.0)					
Ascites[1] (1 = None or 2 = Mild/Controlled or 3 = Severe/Refractory)					
Hepatic Encephalopathy[1] (1 = None or 2 = Grades I-II/Controlled or 3 = Grades III-IV/Refractory)					
Sample Frequency * = cell size less than 30	0 never	1 ever			
[3]Peptic Ulcer (MCQ200)					
1999 - 2000	787.00	*			
2001 - 2004[1]	2,375.00	N/A			
[4]Dementia (chronic cognitive deficit) memory problems (PFQ056 + PFQ059) + unable to manage money (PFQ059)					
16 - 19[1]	1,085.00	N/A			
20 - 29	880.00	*			
30 -39	702.00	0.00			
40 - 49	501.00	*			
[5]Solid Tumor excludes leukemia, lymphoma, blood and bone cancers (MCQ220 + MCQ230A-DD)					
Solid Tumor with no metastases within five years (MCQ220 + MCQ230A-DD) + (RIDAGEYR-MCQ240A-DD)	3,152.00	21.00			
Solid Tumor with metastases (MCQ220 + MCQ230A-DD + 66)	3,168.00	*			
	Primary Site	Secondary Site	Secondary-to-Tertiary Site		
Metastasic Cancers in childbearing-aged female participants		nervous system	lung-to-breast		
	cervix	*			
	ovary	*			
	uterus		*		

[1]Information Not Available in NHANES (1999 - 2004)

References

Brodaty, H., Pond, D., Kemp, N., Luscombe, G., Harding, L., Berman, K., & Huppert, F. (2002). The GPCOG: a new screening test for dementia designed for general practice. *Journal of American Geriatric Society, 50* (3), 530-534. doi:10.1046/j.1532-5415.2002.50122.x

Child, C. & Turcotte, J. (1964). Surgery and portal hypertension. In C. Child (Ed.), *Liver and Portal Hypertension* (pp. 50-64). Philadelphia, PA: W. Saunders.

Hanson, D., Chu, S., Farizo, K., & Ward, J. (1995). Distribution of CD4+T Lymphocytes at Diagnosis of Acquired Immunodeficiency Syndrome – Defining and Other Human Immunodeficiency Virus-Related Illnesses. Archives of Internal Medicine, 155(14), 1537-1542.

Pugh, R., Murray-Lyon, I., Dawson, J., Pietroni, M. & Williams, R. (1973, August). Transection of the oesophagus for bleeding oesophageal varices. *The British Journal of Surgery, 60* (8), 646-649.

Table 19
Composition and Frequencies of Independent Variables that Comprise Iron Deficiency (1999-2004)

Points Sample Frequency * = cell size less than 30	0	1	2+	Total
Iron Deficiency ≥ 2 points	2,021.00 (63.69%)	703.00 (22.16%)	449.00 (14.15%)	3,173.00
Mean Cell Volume (LBXMCVSI)				
81.0 fL or more	2,021.00	651.00	272.00	2,944.00
less than 81.0 fL	0.00	52.00	177.00	229.00
Transferin Saturation (LBXPCT) (serum iron / serum total iron binding capacity x 100%)				
15% or more	2,021.00	276.00	*	2,316.00
less than 15%	0.00	427.00	430.00	857.00
Serum Ferritin (LBXFERSI)				
12 or more µg/L	2,021.00	479.00	41.00	2,541.00
less than 12 µg/L	0.00	224.00	408.00	632.00

fL = femtoliters = 10^{-15} liters

References

Móron, C., & Viteri, F. (2009). Update on common indicators of nutritional status: food access, food consumption and biochemical measures of iron and anemia. *Nutrition Reviews, 67* (Supplement 1), S31-S35. doi:10.1111/j.1753-4887.2009.00156.x

Ross, E. (2002). Evaluation and Treatment of Iron Deficiency in Adults. *Nutrition in Clinical Care* , 5(5), 220-224.

Table 20
Composition and Frequencies of Independent Variables that Comprise Acceptable Macronutrient Distribution Range (AMDR) and Recommended Daily Allowances (RDA) for Specific Nutrients (1999-2004)

	Age 192 to < 240 mo	Age ≥ 240 to < 600 mo
Fat AMDR (fat3cat)	0.25 to 0.35	0.20 to 0.35
Protein AMDR (prot3cat)	0.10 to 0.30	0.10 to 0.35
Iron RDA (iron2cat)		
Pregnant (pregnant = 1)	27 mg	27 mg
Breastfeeding (RHQ200 = 1)	10 mg	9 mg
Neither Pregnant (pregnant = 2 or 3) Nor Breastfeeding (RHQ200 = 2)	15 mg	18 mg
Calcium RDA (calc2cat)		
Pregnant (pregnant= 1)	1,300 mg	1,000 mg
Breastfeeding (RHQ200= 1)	1,300 mg	1,000 mg
Neither Pregnant (pregnant = 2 or 3) Nor Breastfeeding (RHQ200 = 2)	1,300 mg	1,000 mg
Selenium RDA (sele2cat)		
Pregnant (pregnant = 1)	60 µg	60 µg
Breastfeeding (RHQ200 = 1)	70 µg	70 µg
Neither Pregnant (pregnant = 2 or 3) Nor Breastfeeding (RHQ200 = 2)	55 µg	55 µg
Water RDA all sources		
Pregnant (pregnant = 1)	3,000	3,000
Breastfeeding (RHQ200 = 1)	3,800	3,800
Neither Pregnant (pregnant = 2 or 3) Nor Breastfeeding (RHQ200 = 2)	2,300	2,700

References

Institute of Medicine. (2005). Dietary Reference Intakes. Washington, DC: National Academy Press.

Table 21
NHANES Post-Recall Dietary Questionnaire Specific
Fish/Shellfish (1999-2004)

Shellfish Meals
Clams
Crab
Crayfish
Lobster
Mussels
Oysters
Scallops
Shrimp
Other Shellfish
Other Unknown Shellfish
Fish Meals
Breaded Fish Products
Tuna
Bass
Catfish
Cod
Flatfish
Haddock
Mackerel
Perch
Pike
Pollock
Porgy
Salmon
Sardines
Sea Bass
Shark
Swordfish
Trout
Walleye
Other Fish
Other Fish Unknown

References

Centers for Disease Control and Prevention, National Center for Health Statistics. (2007b, November). NHANES 2003-2004 Data Documentation: Dietary Interview Total Nutrient Intakes (First Day). Retrieved February 7, 2011 from http://www.cdc.gov/nchs/data/nhanes/nhanes_03_04/dr1tot_c.pdf

Table 22

Limits of Detection and Imputed Values of Serum Cotinine Levels for Hornung and Reed (1990) Calculations NHANES Sample Population by Each Two-Year Period[1]

Independent Variable	1999 - 2000	2001-2002	2003-2004
Serum Cotinine LBXCOT Non-Detectables[2] = 10,763/42,931 (25.1%) Detectables = 32,168/42,931 (74.9%)	LBXCOT = 0.05/√2 = 0.035 µg/dl Non-Detectables[2] = 4,480/11,767 (38.07%) Mode of Detectable Values Only: 0.035 GStandard Deviation of Log Detectable Values: 2.944 Skewness: 0.676	LBXCOT= 0.05/√2 = 0.035 µg/dl or LBXCOT = 0.15/√2 = 0.011 µg/dl Non-Detectables[2] = 3,937/16,071 (24.49%) Mode of Detectable Values Only: 0.011 GStandard Deviation of Log Detectable Values: 3.219 Skewness: 0.791	LBXCOT = 0.15/√2 = 0.011 µg/dl Non-Detectables[2] = 2,346/15,093 (15.54%) Mode of Detectable Values Only: 0.011 GStandard Deviation of Log Detectable Values: 3.214 Skewness: 0.819

[1]with no missing data [2]Non-Detectables = Below Level of Detection

Table 23
Composition of Independent Variables that Comprise Industrial Categories
(1999-2004)

Industrial Categories	NAICS Census 2000
Goods-Producing Industries	
Natural Resources & Mining	001 - 056
agriculture production	
agricultural services, forestry and fishing	
mining	
Construction	077 - 106
construction	
Manufacturing	107 - 406
food and kindred products	
textile mill products	
apparel and other finished textile products	
paper products, printing, publishing and allied industries	
chemicals, petroleum and coal products	
rubber, plastics and leather products	
lumber and wood products including furniture	
metal industires	
machiery except electrical	
electrical machinery, equipment and supplies	
transportation equipment	
miscellaneous and not specified manufacturing industries	
Services-Producing Industries	
Trade, Transportation & Utilities [1]	407 - 646; 057 - 076
trucking service	
transportation except trucking	
utilities	
wholesale trade, durable goods	
whoelsale trade, non-durable and not specified goods	
retail department stores	
retail food stores	
retail vehicle dealers, supply and service stores	
retail eating and drinking places	
other retail trade	
Information/Communications	647 - 686
information/communications	
Financial Activities	687 - 726
banking and other finance	
insurance and real estate	

Table 23
Composition of Independent Variables that Comprise Industrial Categories
(1999-2004)

Industrial Categories	NAICS Census 2000
Professional & Business Services	727 - 785
business services	
other professional and related services	
Other Services	877 - 936
repair services	
private households	
Leisure & Hospitality	856 - 876
lodging places	
personal services except private households and lodging	
entertainment and recreation services	
Education & Health Services	786 - 855
offices of health practitioners	
hospitals	
health services, n.e.c.	
educational services	
social services	
Public Administration	937 - 966
justice, public order and safety	
public administration except justice, public order and safety	
military and national security	
Unemployed	992
blank but applicable	

[1]Utilities reclassified from goods to services by NAICS in 1997

References

U.S. Census Bureau. (2001b, October). Occupation Detailed Code List: Decennial 2000 SOC and U.S. Census 2000. Retrieved February 7, 2011 from http://factfinder.census.gov/metadoc/occupation.pdf

U.S. Census Bureau. (2003a, March). North American Industry Classification System (NAICS) Index of Industry and Occupations: Alternate Aggregation Structure. Retrieved February 7, 2011 from http://www.dlt.ri.gov/lmi/pdf/alternate.pdf

Table 24
Composition of Independent Variables that Comprise Occupational Categories
(1999-2004)

Occupational Categories	Census 2000
Managerial and Professional Occupations	001-359
executive, administrators and managers	
management-related occupations	
farm operators, managers and supervisors	
engineers, architects and scientists	
health diagnosing, assessing and treating occupations	
teachers	
writers, artists, entertainers and athletes	
other professional specialty occupations	
technicians and related support occupations	
Sales-Related Occupations	470-599
supervisors and proprietors, sales occupations	
sales representatives, finance, business and commodities excluding retail	
sales workers, retail and personal services	
secretaries, stenographers and typists	
information clerks	
records processing occupations	
material recording, scheduling and distributing clerks	
miscellaneous administrative support occupations	
Services-Related Occupations	360-469
private household occupations	
protective service occupations	
waiters and waitresses	
cooks	
miscellaneous food preparation and service occupations	
health service occupations	
cleaning and building service occuaptions	
personal service occupations	
Farming, Fishing and Forestry Occupations	600-613
farm and nursery workers	
related agricultural, forestry and fishing occupations	
vehicle and mobile equipment mechanics and repairers	
other mechanics and repairers	
Construction, Extraction and Maintenance Occupations	620-769
construction trades	
extractive and prercision production occupations	
textile, apparel and furnishings machine operators	
machine operators, assorted materials	

Table 24
Composition of Independent Variables that Comprise Occupational Categories (1999-2004)

Occupational Categories	Census 2000
Production, Transportation and Material Moving Occupations	770-979
fabricators, assemblers, inspectors and samplers	
motor vehicle operators	
other transportation and material moving occupations	
construction laborers	
laborers excluding construction	
freight, stock and material movers, hand	
other helpers, equipment cleaners, hand packagers and laborers	

References

U.S. Census Bureau. (2001b, October). Occupation Detailed Code List: Decennial 2000 SOC and U.S. Census 2000. Retrieved February 7, 2011 from http://factfinder.census.gov/metadoc/occupation.pdf

U.S. Census Bureau. (2003a, March). North American Industry Classification System (NAICS) Index of Industry and Occupations: Alternate Aggregation Structure. Retrieved February 7, 2011 from http://www.dlt.ri.gov/lmi/pdf/alternate.pdf

Table 25
Composition and Frequencies of Independent Variables that Comprise Income-Related Variables (1999-2004)

	1999-2000	2001-2002	2003-2004
Family Poverty Income Ratio (INDFMPIR)[1]	1.00	1.00	1.00
Low Income Status (200%*INDFMPIR)	1.00	1.00	1.00
data missing or incomplete	n = 216 (6.81%)		
Median Family Income (MFI)	$ 49,628.00	$ 52,742.00	$ 53,692.00
Relative Poverty (60%*MFI)	$ 29,776.80	$ 31,645.00	$ 32,215.20
Housing Assistance Eligibility (50%*MFI)	$ 24,814.00	$ 26,371.00	$ 26,846.00
Annual Household Income (INDHHINC)	reported in ranges		
data missing or incomplete	n = 305 (10.40%)		
Annual Family Income (INDFMINC)	reported in ranges		
data missing or incomplete	n = 216 (6.81%)		

[1]before tax money income; poverty income threshold varies with age and family size

References

Assistant Secretary for Planning and Evaluation, U.S. Department of Health and Human Services. (January 25, 2010). *Further Resources on Poverty Measurement, Poverty Lines and Their History* . Retrieved February 7, 2011 from http://aspe.hhs.gov/poverty/contacts.shtml

Table 26
Frequencies of Independent Variables
(unweighted and weighted data 1999-2004)

Independent Variable Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30	Total
Susceptibility-Related Attributes	
Age (age4cat)	
	1,085.00
	(34.19%)
	18,510,468.72
16-19[R]	(13.76%)
	884.00
	(27.86%)
	45,347,514.91
20-29	(33.72%)
	702.00
	(22.13%)
	36,357,836.50
30-39	(27.03%)
	502.00
	(15.82%)
	34,286,213.30
40-49	(25.49%)
Health Status	
Perceived Health Status (huq2cat)	
	2,634.00
	(89.53%)
	124,005,245.09
excellent, very good, good[R]	(92.22%)
	332.00
	(10.47%)
	10,465,879.59
fair, poor	(7.78%)
missing = 1	
Charleson Co-Morbidity Scale (CCMS3cat)	
	2,814.00
	(88.69%)
	118,257,021.43
none[R]	(87.93%)
	303.00
	(9.55%)
	13,146,733.31
one co-morbidity	(9.77%)
	56.00
	(1.76%)
	3,098,278.69
more than one co-morbidity	(2.30%)
Iron Deficiency (FeD2cat)	
	2,724.00
	(85.85%)
	122,836,758.63
within normal limits[R]	(91.33%)
	449.00
	(14.15%)
	11,665,274.81
iron deficient	(8.67%)
Treatment for Iron Deficiency past 3 mo (FeTx2cat)	
	171.00
	(5.39%)
	5,146,295.97
yes	(3.83%)
	3,001.00
	(94.61%)
	129,342,158.39
no[R]	(96.17%)
missing = 1	

Table 26
Frequencies of Independent Variables
(unweighted and weighted data 1999-2004)

Independent Variable Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30	Total
Iron Deficiency and Treatment (FeDTx)	
	2,608.00
	(82.22%)
	119,442,698.68
normal/no treatment[R]	(88.81%)
	115.00
	(3.63%)
	3,380,480.87
normal w/treatment	(2.51%)
	56.00
	(1.76%)
	1,765,815.10
deficient w/treatment	(1.32%)
	393.00
	(12.39%)
	9,899,459.71
deficient/no treatment	(7.36%)
missing = 1	
Health Insurance (hi2cat)	
	2,042.00
	(64.35%)
	100,132,778.47
private[R]	(74.45%)
	439.00
	(13.83%)
	9,791,420.08
public	(7.28%)
	619.00
	(19.51%)
	21,762,804.37
none	(16.18%)
	73.00
	(2.31%)
	2,815,030.52
missing	(2.09%)
Regular Source of Healthcare (hp2cat)	
	2,673.00
	(84.84%)
	115,458,282.32
yes[R]	(85.58%)
	500.00
	(15.76%)
	19,043,751.11
no	(14.16%)
Source of Healthcare (hcsre)	
	1,807.00
	(56.95%)
	85,161,839.99
healthcare provider[R]	(63.32%)
	676.00
	(21.31%)
	23,768,096.88
clinic	(17.67%)
	642.00
	(20.23%)
	22,962,656.75
ER or none	(17.07%)
	48.00
	(1.51%)
	2,609,439.80
missing	(1.94%)

Table 26
Frequencies of Independent Variables
(unweighted and weighted data 1999-2004)

Independent Variables with Exposure Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30	Total
Nutritional Status	
Food Security (food2cat)	
	2,572.00
	(81.05%)
	114,043,912.66
food secure[R]	(84.79%)
	459.00
	(14.47%)
	14,234,522.05
food insecure	(10.58%)
	142.00
	(4.48%)
	6,223,598.72
missing	(4.63%)
Body Mass Index (bmi30cat)	
<30.0[R]	2,357.00
underweight	(74.28%)
normal	102,843,896.92
overweight	(76.46%)
	777.00
	(24.49%)
30.0+	30,216,437.80
obese	(22.47%)
	39.00
	(1.23%)
	1,441,678.71
missing	(1.07%)
Fat Intake/AMDR (fat3cat)	
	1,898.00
	(59.82%)
	81,144,342.04
recommended or less[R]	(60.33%)
	1,275.00
	(40.18%)
	53,357,691.39
more than recommended	(39.67%)
Protein Intake/AMDR (prot3cat)	
	2,712.00
	(85.47%)
	118,763,765.28
recommended or more[R]	(88.30%)
	461.00
	(14.53%)
	15,738,268.16
less than recommended	(11.70%)
Iron Intake/RDA (iron2cat)	
	929.00
	(29.28%)
	33,509,679.62
recommended or more[R]	(24.91%)
	2,244.00
	(70.72%)
	100,992,353.82
less than recommended	(75.09%)
Calcium Intake/RDA (calc2cat)	
	845.00
	(26.63%)
	42,562,543.92
recommended or more[R]	(31.64%)
	2,328.00
	(73.37%)
	91,939,489.51
less than recommended	(68.36%)

Table 26
Frequencies of Independent Variables
(unweighted and weighted data 1999-2004)

Independent Variables with Exposure Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30	Total
Selenium Intake/RDA (sele2cat)	
recommended or more[R]	2,559.00 (80.65%) 112,678,482.86 (83.77%)
less than recommended	614.00 (19.35%) 21,823,550.58 (16.23%)
Reproductive Status	
Current Pregnancy (pregnant)	
pregnant	391.00 (12.32%) 4,842,189.09 (3.60%)
not pregnant[R]	2,641.00 (83.23%) 126,376,518.93 (93.96%)
missing	141.00 (4.44%) 3,283,325.40 (2.44%)
Trimester of Pregnancy (tripcorr)	
not pregnant[R]	2,782.00 (87.68%) 129,659,844.35 (96.40%)
1st trimester	149.00 (4.69%) 1,991,566.11 (1.48%)
2nd trimester	132.00 (4.16%) 1,523,495.53 (1.13%)
3rd trimester	110.00 (3.47%) 1,327,127.44 (0.99%)
Ever Pregnant (tprg2cat)	
never pregnant[R]	1,535.00 (48.38%) 59,565,096.98 (44.29%)
one or more pregnancies	1,638.00 (51.62%) 74,936,936.45 (55.71%)
Live Births (live)	
no live births[R]	1,820.00 (57.36%) 67,420,238.21 (50.13%)
one or more live births	1,353.00 (42.64%) 67,081,795.21 (49.87%)

Table 26
Frequencies of Independent Variables
(unweighted and weighted data 1999-2004)

Independent Variables with Exposure Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30	Total
Ever Breastfed (brstfda)	
never breastfed[R]	2,323.00 (73.12%) 91,954,322.30 (68.37%)
breastfed more than one month and/or currently	850.00 (26.79%) 42,547,711.13 (31.63%)
Exposure-Related Attributes	
Acculturation	
Birthplace (born2cat)	
U.S.[R]	2,673.00 (84.24%) 120,303,696.80 (89.44%)
outside U.S.	500.00 (15.76%) 14,198,336.63 (10.56%)
Years in U.S. (yrus5)	
born in U.S.[R]	2,673.00 (84.40%) 120,303,696.80 (89.56%)
five or more years	352.00 (11.12%) 11,073,519.21 (8.24%)
less than five years	142.00 (4.48%) 2,960,966.91 (2.20%)
missing = 6	
Language Spoken at Home (lang2cat)	
English[R]	2,844.00 (89.69%) 126,761,194.36 (97.72%)
Other	327.00 (10.31%) 2,960,966.91 (2.28%)
missing = 2	
U.S. Citizenship (usczn2cat)	
U.S. citizen[R]	2,814.00 (88.71%) 126,825,271.91 (94.31%)
non-U.S. citizen	358.00 (11.29%) 7,654,892.84 (5.69%)
missing = 1	
Diet	
Seafood Eaten in Past 30 Days (smpw2cat)	
none[R]	686.00 (21.62%) 22,870,840.79 (17.01%)
any	2,487.00 (78.38%) 111,631,192.65 (82.99%)

Table 26
Frequencies of Independent Variables
(unweighted and weighted data 1999-2004)

Independent Variables with Exposure Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30	Total
Fish Eaten in Past 30 Days (fish2cat)	
	1,040.00 (32.78%) 36,809,739.68 (27.37%)
none[R]	
	2,133.00 (67.22%) 97,692,293.76 (72.63%)
any	
Shellfish Eaten in Past 30 Days (shell2cat)	
	1,557.00 (49.07%) 63,018,639.18 (46.85%)
none[R]	
	1,616.00 (50.93%) 71,483,394.25 (53.15%)
any	
Tap Water Consumed Prior 24h (tap2kct)	
	1,129.00 (35.58%) 40,504,828.24 (30.12%)
none[R]	
	1,538.00 (48.47%) 71,045,485.42 (52.82%)
< 2,000 ml	
	295.00 (9.29%) 15,529,552.50 (11.55%)
2,000+ ml	
	211.00 (6.66%) 7,422,167.26 (5.51%)
missing	
Alcohol Consumption	
Alcohol Consumption (retohuse)	
	1,743.00 (54.93%) 52,220,515.36 (38.83%)
never, seldom drinker[R] *including 16-19 y/o*	
	730.00 (23.01%) 40,670,079.57 (30.24%)
drinker	
	555.00 (17.49%) 35,765,379.44 (26.59%)
heavy drinker	
	145.00 (4.57%) 5,846,059.05 (4.34%)
missing	

Table 26
Frequencies of Independent Variables
(unweighted and weighted data 1999-2004)

Independent Variables with Exposure Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30	Total
Tobacco Use	
Serum Cotinine (cot3cat)	
< 1.0 ng/ml[R]	2,368.00 (74.99%) 98,871,473.57 (73.86%)
1.0 - 10.0 ng/ml	190.00 (6.02%) 5,250,301.62 (3.92%)
> 10.0 ng/ml	600.00 (18.99%) 29,750,341.24 (22.22%)
missing = 15	
ETS (ETS)	
no ETS[R]	2,417.00 (76.17%) 101,797,371.58 (75.68%)
ETS at home or work	650.00 (20.49%) 26,706,869.60 (19.86%)
ETS at home and work	106.00 (3.34%) 5,997,792.25 (4.46%)
Residence	
Tap Water Source (h2os2cat)	
public[R]	2,826.00 (89.06%) 116,735,908.16 (86.79%)
private	266.00 (8.38%) 14,491,535.02 (10.77%)
missing	81.00 (2.56%) 3,274,590.25 (2.44%)
Residential Tap Water Treatment (h2ox2cat)	
yes	863.00 (27.19%) 45,308,234.67 (33.69%)
no[R]	2,239.00 (70.56%) 86,545,337.36 (64.34%)
missing	71.00 (2.24%) 264,861.40 (1.98%)

Table 26
Frequencies of Independent Variables
(unweighted and weighted data 1999-2004)

Independent Variables with Exposure Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30	Total
Type of Residence (res3cat)	
	2,072.00
	(65.30%)
	89,305,970.56
attached or detached house[R]	(66.39%)
	202.00
	(6.37%)
	8,401,777.80
mobile home or trailer	(6.25%)
	899.00
	(28.33%)
all other types	36,794,285.07
including missing/unknown	(27.36%)
Age of Residence (resb60cat)	
	1,595.00
	(50.27%)
	78,044,524.02
1960 or newer[R]	(58.03%)
	766.00
	(24.14%)
	32,092,200.10
older than 1960	(23.86%)
	812.00
	(25.59%)
	24,365,309.31
missing/unknown	(18.11%)
Age of Residence (resb78cat)	
	1,087.00
	(34.26%)
	55,388,048.85
1978 or newer[R]	(41.18%)
	1,274.00
	(40.15%)
	54,748,695.27
older than 1978	(40.70%)
	812.00
	(25.59%)
	24,365,309.31
missing/unknown	(18.12)
Resident Status (resd3cat)	
	1,727,00
	(54.43%)
	77,250,307.12
own[R]	(57.43%)
	1,278.00
	(40.28%)
	51,163,895.57
rent	(38.04%)
	168.00
	(5.29%)
other	6,087,830.73
including missing	(4.53%)
Years at Current Residence (re5yrcat)	
	1,113.00
	(35.08%)
	45,894,318.68
more than five years[R]	(34.12%)
	2,007.00
	(63.25%)
	86,455,612.94
five years or less	(64.28%)
	53.00
	(1.67%)
	2,152,010.81
missing	(1.60%)

Table 26
Frequencies of Independent Variables
(unweighted and weighted data 1999-2004)

Independent Variables with Exposure Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30	Total
Household Size (hsize)	
four persons or less[R]	2,182.00 (68.77%) 106,454,028.39 (79.15%)
more than four persons	991.00 (31.23%) 28,048,005.05 (20.85%)
Rooms in Residence (rm3cat)	
7+ rooms[R]	1,148.00 (36.18%) 52,616,512.81 (39.12%)
4-6 rooms	1,691.00 (53.29%) 69,100,132.59 (51.37%)
1-3 rooms	263.00 (8.29%) 10,175,173.11 (7.57%)
missing	71.00 (2.24%) 2,610,214.92 (1.94%)
Occupation	
Current Occupation (cocc2cat)	
not working[R]	1,324.00 (41.73%) 42,172,957.57 (31.36%)
management, professional & sales	1,243.00 (39.17%) 67,758,891.74 (50.38%)
services & goods	606.00 (19.10%) 24,570,184.12 (18.26%)
Time in Current Employment (cjt)	
not working[R]	1,324.00 (41.73%) 42,172,957.57 (31.36%)
less than five years	1,434.00 (45.19%) 67,241,639.72 (49.99%)
five or more years	415.00 (13.08%) 25,087,436.13 (18.65%)
Total Hours Worked Prior Week (hrwk)	
not employed[R]	1,381.00 (43.55%) 45,808,038.02 (34.09%)
less than 35 hours	736.00 (23.21%) 33,367,433.79 (24.84%)
35+ hours	1,054.00 (33.24%) 55,181,521.00 (41.07%)
missing = 2	

Table 26
Frequencies of Independent Variables
(unweighted and weighted data 1999-2004)

Independent Variables with Exposure Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30	Total
Longest Held Occupation (locc2cat)	
not applicable[R]	1,562.00 (49.23%) 64,117,356.51 (47.67%)
management, professional & sales	903.00 (28.46%) 41,415,186.15 (30.79%)
services & goods	708.00 (22.31%) 28,969,490.47 (21.54%)
Time in Longest Employment (ljt)	
not applicable[R]	1,562.00 (49.23%) 64,117,356.51 (47.67%)
less than five years	997.00 (31.42%) 34,542,268.85 (25.68%)
five or more years	614.00 (19.35%) 35,842,408.07 (26.65%)
Socioeconomic Factors	
Education	
Highest Education (educ2)	
high school diploma, GED or higher[R]	2,037.00 (64.22%) 106,907,161.34 (79.52%)
less than high school diploma	1,135.00 (35.78%) 27,527,733.30 (20.48%)
missing = 1	
Employment	
Employment Status (emp3cat)	
employed	1,853.00 (58.44%) 92,468,799.96 (68.76%)
not employed[R]	1,318.00 (41.56%) 42,022,013.95 (31.24%)
missing = 2	
Reason for Unemployment (unem2cat)	
working[R]	1,853.00 (58.40%) 92,468,799.96 (68.75%)
voluntary unemployment	924.00 (29.12%) 28,265,021.67 (21.01%)
involuntary unemployment	295.00 (9.30%) 10,263,114.01 (7.63%)
missing	101.00 (3.18%) 3,505,097.78 (2.61%)

Table 26
Frequencies of Independent Variables
(unweighted and weighted data 1999-2004)

Independent Variables with Exposure Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30	TOTAL
Work History (wkcp)	
never employed[R]	408.00 (12.86%) 8,238,810.80 (6.12%)
currently employed	1,154.00 (36.37%) 55,878,545.71 (41.55%)
employed in the past but not currently	916.00 (28.87%) 33,934,146.78 (25.23%)
employed now and in the past	695.00 (21.90%) 36,450,530.14 (27.10%)
Income	
U.S. Poverty Threshold (pov2cat)	
more than 1.00[R]	2,227.00 (70.19%) 103,953,623.19 (77.29%)
1.00 or less	730.00 (23.00%) 22,587,197.47 (16.79%)
missing	216.00 (6.81%) 7,961,212.77 (5.92%)
Marital Status	
Marital Status (marr3cat)	
married or living with partner	1,198.00 (37.75%) 61,800,648.25 (45.95%)
widowed, divorced or separated	261.00 (8.23%) 14,353,952.38 (10.67%)
never married[R]	1,637.00 (51.59%) 53,492,951.49 (39.77%)
missing	77.00 (2.43%) 4,854,481.31 (3.61%)

Table 26
Frequencies of Independent Variables
(unweighted and weighted data 1999-2004)

Independent Variables with Exposure Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30	TOTAL
Race-Ethnicity	
Race-Ethnicity (race5cat)	
Non-Hispanic White[R]	1,493.00 (47.05%) 97,887,544.16 (72.78%)
Non-Hispanic Black	623.00 (19.63%) 12,747,178.37 (9.48%)
Mexican American	745.00 (23.49%) 8,670,575.81 (6.45%)
Other Hispanic	178.00 (5.61%) 7,525,992.22 (5.59%)
Asian, Native American, Pacific Islander & Multi-Racial	134.00 (4.22%) 7,670,742.88 (5.70%)
Race-Ethnicity/Hispanic Grouping (race4cat)	
Non-Hispanic White[R]	1,493.00 (47.05%) 97,887,544.16 (72.78%)
Non-Hispanic Black	623.00 (19.63%) 12,747,178.37 (9.48%)
Hispanic	923.00 (29.10%) 16,196,568.03 (12.04%)
Asian, Native American, Pacific Islander & Multi-Racial	134.00 (4.22%) 7,670,742.88 (5.70%)

Table 27
Bivariate Analyses of Selected Independent Variable Pairs Among Childbearing-Aged Participants
(unweighted and weighted data 1999-2004)

Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30					χ^2 *p value* *unweighted*	χ^2 *p value* *weighted*
	Current Pregnancy (pregnant)					
Age (age4cat)	pregnant	not pregnant^{R}	missing			
16-19^{R}	(15.60%) 404,786.28 (8.36%)	(34.12%) 15,916,106.14 (12.59%)	(87.23%) 2,189,576.30 (66.69%)		388.32 *<0.0001*	7.77 *0.0000*
20-29	(50.64%) 2,562,930.76 (52.93%)	(25.60%) 42,139,552.13 (33.34%)	(0.00%) 645,032.0 (19.65%)			
30-39	(33.25%) 1,687,710.61 (34.85%)	(21.58%) 34,543,369.03 (27.33%)	(0.00%) 126,756.87 (3.86%)			
40-49	(0.00%) 186,761.44 (3.86%)	(18.71%) 33,777,491.63 (26.73%)	(0.00%) 321,960.23 (9.81%)			
	Live Births (live)					
Age (age4cat)	no live births^{R}	one or more live births				
16-19^{R}	992.00 (54.51%) 17,576,095.72 (26.07%)	93.00 (6.87%) 934,373.00 (1.39%)				
20-29	546.00 (30.00%) 33,032,255.38 (48.99%)	338.00 (24.98%) 12,315,259.53 (18.36%)			1,097.32 *<0.0001*	37.14 *0.0000*
30-39	189.00 (10.38%) 10,485,224.99 (15.55%)	513.00 (37.92%) 25,872,611.51 (38.57%)				
40-49	93.00 (5.11%) 6,326,662.12 (9.38%)	409.00 (30.23%) 27,959,551.18 (41.68%)				
	Ever Breastfed (brstfda)					
Age (age4cat)	never breastfed^{R}	breastfed more than one month or currently				
16-19^{R}	1,047.00 (45.07%) 18,135,863.89 (19.72%)	38.00 (4.47%) 374,604.83 (0.88%)				
20-29	669.00 (28.80%) 37,884,359.67 (41.20%)	215.00 (25.29%) 7,463,155.24 (17.54%)			621.82 *<0.0001*	26.89 *0.0000*
30-39	352.00 (15.15%) 18,595,942.83 (20.22%)	350.00 (41.18%) 17,761,893.68 (41.75%)				
40-49	255.00 (10.98%) 17,338,155.92 (18.86%)	247.00 (29.06%) 16,948,057.38 (39.83%)				

Table 27
Bivariate Analyses of Selected Independent Variable Pairs Among Childbearing-Aged Participants (unweighted and weighted data 1999-2004)

Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30					χ^2 *p value unweighted*	χ^2 *p value weighted*
	U.S. Poverty Threshold (pov2cat)					
Age (age4cat)	more than 1.00	1.00 or less	missing			
16-19^{R}	647.00 (29.05%) 12,250,212.93 (11.78%)	357.00 (48.90%) 4,716,875.65 (20.88%)	81.00 (37.50%) 1,543,380.14 (19.39%)			
20-29	639.00 (28.69%) 32,516,350.52 (31.28%)	183.00 (25.07%) 9,634,923.63 (42.66%)	62.00 (28.70%) 3,196,240.76 (40.15%)		107.59 *<0.0001*	4.16 *0.0021*
30-39	546.00 (24.52%) 29,389,193.20 (28.27%)	108.00 (14.79%) 4,451,494.54 (19.71%)	48.00 (22.22%) 2,517,148.76 (31.62%)			
40-49	395.00 (17.74%) 29,797,866.54 (28.66%)	82.00 (11.23%) 3,783,903.65 (16.75%)	* (0.00%) 704,443.11 (8.85%)			
	Time in Current Employment (cjt)					
Age (age4cat)	not working^{R}	less than five years	five or more years			
16-19^{R}	659.00 (49.77%) 10,401,116.38 (24.66%)	413.00 (28.80%) 7,917,836.01 (11.78%)	* (0.00%) 191,516.34 (0.76%)			
20-29	287.00 (21.68%) 11,681,897.31 (27.70%)	532.00 (37.10%) 30,433,333.24 (45.26%)	65.00 (15.66%) 3,232,284.36 (12.88%)		608.37 *<0.0001*	14.94 *0.0000*
30-39	214.00 (16.16%) 10,375,482.65 (24.60%)	332.00 (23.15%) 17,671,355.71 (26.28%)	156.00 (37.59%) 8,310,998.15 (33.13%)			
40-49	164.00 (12.39%) 9,714,461.24 (23.03%)	157.00 (10.95%) 11,219,114.77 (16.68%)	181.00 (43.61%) 13,352,637.29 (53.22%)			
	Time in Longest Employment (ljt)					
Age (age4cat)	not applicable^{R}	less than five years	five or more years			
16-19^{R}	584.00 (37.99%) 9,124,957.53 (14.23%)	497.00 (49.85%) 9,365,372.71 (27.11%)	* (0.00%) 20,138.48 (0.06%)			
20-29	398.00 (25.48%) 19,804,736.22 (30.89%)	367.00 (36.81%) 18,212,333.88 (52.72%)	119.00 (19.38%) 7,330,444.80 (20.45%)		778.16 *<0.0001*	19.79 *0.0000*
30-39	320.00 (20.49%) 16,623,205.52 (25.93%)	79.00 (7.92%) 3,839,684.30 (11.12%)	303.00 (49.35%) 15,894,946.68 (44.35%)			
40-49	260.00 (16.65%) 18,564,457.23 (28.95%)	54.00 (5.42%) 3,124,877.96 (9.05%)	188.00 (30.62%) 12,596,878.11 (35.15%)			

Table 27
Bivariate Analyses of Selected Independent Variable Pairs Among Childbearing-Aged Participants (unweighted and weighted data 1999-2004)

Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30					χ^2 *p value* *unweighted*	χ^2 *p value* *weighted*
	Marital Status (marr3cat)					
Age (age4cat)	married or living with partner	widowed, divorced or separated	never married	missing		
16-19^{R}	62.00 (5.18%) 704,745.98 (1.14%)	* (0.00%) 115,609.26 (0.81%)	1,014.00 (61.94%) 17,678,830.80 (33.05%)	* (0.00%) 11,282.68 (0.23%)		
20-29	363.00 (30.30%) 15,242,297.90 (24.66%)	32.00 (12.26%) 1,286,019.58 (8.96%)	461.00 (28.16%) 27,402,743.76 (51.23%)	* (0.00%) 1,416,453.67 (29.18%)	1,589.54 *<0.0001*	15.82 *0.0000*
30-39	468.00 (39.07%) 23,431,086.70 (37.91%)	91.00 (34.87%) 4,769,461.14 (33.23%)	110.00 (6.72%) 5,921,247.56 (11.07%)	33.00 (42.86%) 2,236,041.11 (46.06%)		
40-49	305.00 (25.46%) 22,422,517.68 (36.28%)	132.00 (50.57%) 8,182,862.39 (57.01%)	52.00 (3.18%) 2,490,129.38 (4.66%)	* (0.00%) 1,190,703.85 (24.53%)		
	Charleson Co-Morbidity Scale (CCMS3cat)					
Perceived Health Status (huq2cat)	none^{R}	one co-morbidity	greater than one co-morbidity			
excellent, very good, good^{R}	2,569.00 (91.33%) 110,842,654.41 (93.75%)	235.00 (77.56%) 10,845,849.86 (82.50%)	36.00 (64.29%) 2,316,740.82 (74.78%)		94.11 *<0.0001*	7.01 *0.0023*
fair, poor	244.00 (8.67%) 7,383,458.27 (6.25%)	68.00 (22.44%) 2,300,883.45 (17.50%)	* (0.00%) 781,537.87 (25.22%)			
	Treatment for Iron Deficiency past 3 months (FeTx2cat)					
Iron Deficiency (FeD2cat)	yes	no^{R}				
within normal limits^{R}	115.00 (67.25%) 3,380,480.87 (65.69%)	2,608.00 (86.90%) 119,442,698.68 (92.35%)			51.42 *<0.0001*	4.17 *0.047*
iron deficient	56.00 (32.75%) 1,765,815.10 (34.31%)	393.00 (13.10%) 9,899,459.71 (7.65%)				
	Source of Healthcare (hcsre)					
Health Insurance (hi2cat)	healthcare provider^{R}	clinic	ER or none	missing		
private^{R}	1,408.00 (77.92%) 70,148,220.11 (82.37%)	356.00 (52.66%) 15,925,109.07 (67.00%)	244.00 (38.01%) 11,763,693.79 (51.23%)	34.00 (70.83%) 2,295,755.50 (87.98%)		
public	207.00 (11.46%) 5,828,476.81 (6.84%)	136.00 (20.12%) 2,523,657.15 (10.62%)	92.00 (14.33%) 1,377,137.24 (6.00%)	* (0.00%) 62,148.88 (2.38%)	504.59 *<0.0001*	6.45 *0.0000*
none	150.00 (8.30%) 7,084,647.84 (8.32%)	179.00 (26.48%) 5,188,183.59 (21.83%)	281.00 (43.77%) 9,244,055.24 (40.26%)	* (0.00%) 245,917.70 (9.42%)		
missing	42.00 (2.32%) 2,100,495.24 (2.47%)	* (0.00%) 131,147.07 (0.55%)	* (0.00%) 577,770.48 (2.52%)	* (0.00%) 5,617.72 (0.22%)		

Table 27
Bivariate Analyses of Selected Independent Variable Pairs Among Childbearing-Aged Participants (unweighted and weighted data 1999-2004)

Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30					χ^2 *p value unweighted*	χ^2 *p value weighted*
	Regular Source of Healthcare (hp2cat)					
Source of Healthcare (hcsre)	yes^{R}	no				
healthcare provider^{R}	1,807.00 (67.60%) 85,161,840.00 (73.76%)	0.00 (0.00%) 0.00 (0.00%)			2,339.90 *<0.0001*	11.42 *0.0000*
clinic	676.00 (25.29%) 23,768,096.88 (20.59%)	0.00 (0.00%) 0.00 (0.00%)				
ER or none	142.00 (5.31%) 3,918,905.64 (3.39%)	500.00 (100.00%) 19,043,751.11 (100.00%)				
missing	48.00 (1.80%) 2,609,439.81 (2.26%)	0.00 (0.00%) 0.00 (0.00%)				
	Body Mass Index (bmi30cat)					
Food Security (food2cat)	<30.0^{R} underweight normal overweight	30.0+ obese	missing			
food secure^{R}	1,941.00 (82.35%) 89,256,111.99 (86.79%)	597.00 (76.83%) 23,497,629.27 (77.76%)	34.00 (87.18%) 1,290,171.40 (89.49%)		15.44 *0.004*	2.49 *0.056*
food insecure	310.00 (13.15%) 8,787,220.12 (8.54%)	145.00 (18.66%) 5,376,408.10 (17.79%)	* (0.00%) 70,893.83 (4.92%)			
missing	106.00 (4.50%) 4,800,564.81 (4.67%)	35.00 (4.50%) 1,342,420.43 (4.44%)	* (0.00%) 80,613.48 (5.59%)			
	Fat Intake/AMDR (fat3cat)					
Food Security (food2cat)	recommended or less^{R}	more than recommended				
food secure^{R}	1,513.00 (79.72%) 67,837,817.09 (83.60%)	1,059.00 (83.065%) 46206095.57 (86.60%)			5.70 *0.058*	0.81 *0.45*
food insecure	296.00 (15.60%) 9,215,828.85 (11.36%)	163.00 (12.78%) 5018693.20 (9.41%)				
missing	89.00 (4.69%) 4,090,696.09 (5.04%)	53.00 (4.16%) 2,132,902.62 (3.99%)				

Table 27
Bivariate Analyses of Selected Independent Variable Pairs Among Childbearing-Aged Participants (unweighted and weighted data 1999-2004)

Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30					χ^2 *p value* *unweighted*	χ^2 *p value* *weighted*
	U.S. Poverty Threshold (pov2cat)					
Food Security (food2cat)	more than 1.00^{R}	1.00 or less	missing			
food secure^{R}	1,932.00 (86.25%) 91,382,474.30 (87.91%)	489.00 (66.99%) 17,025,611.13 (75.33%)	151.00 (69.91%) 5,635,827.23 (70.79%)		390.73 *<0.0001*	8.42 *0.0000*
food insecure	206.00 (9.25%) 8,765,266.27 (8.43%)	234.00 (32.05%) 5,182,943.35 (22.95%)	* (0.00%) 286,312.42 (3.60%)			
missing	46.00 (21.30%) 2,039,073.12 (25.61%)	89.00 (4.00%) 3,805,882.62 (3.66%)	7.00 (0.96%) 378,642.99 (1.68%)			
	Fat Intake/AMDR (fat3cat)					
Body Mass Index (bmi30cat)	recommended or less^{R}	more than recommended				
<30.0^{R} underweight, normal, overweight	1,440.00 (75.87%) 63,977,815.31 (78.84%)	917.00 (71.92%) 38,866,081.61 (72.84%)			11.88 *0.0026*	2.08 *0.137*
30.0+ obese	429.00 (22.60%) 16,145,112.74 (19.89%)	348.00 (27.29%) 14,071,345.06 (26.37%)				
missing	29.00 (1.27%) 1,021,413.99 (1.26%)	10.00 (0.79%) 420,264.71 (0.79%)				
	Live Births (live)					
Body Mass Index (bmi30cat)	no live births^{R}	one or more live births				
<30.0^{R} underweight, normal, overweight	1,481.00 (81.37%) 55,529,169.26 (82.36%)	876.00 (64.75%) 47,314,727.66 (70.53%)			112.69 *<0.0001*	3.57 *0.0365*
30.0+ obese	321.00 (17.64%) 11,163,737.15 (16.56%)	456.00 (33.70%) 19,052,720.65 (28.40%)				
missing	18.00 (0.99%) 727,331.80 (1.08%)	21.00 (1.55%) 714,346.91 (1.06%)				
	Ever Breastfed (brstfda)					
Body Mass Index (bmi30cat)	never breastfed^{R}	breastfed more than one month				
<30.0^{R} underweight, normal, overweight	1,784.00 (76.80%) 71,780,209.53 (78.06%)	573.00 (67.41%) 31,063,687.39 (73.01%)			29.38 *<0.0001*	3.36 *0.0439*
30.0+ obese	511.00 (22.00%) 18,884,776.92 (20.54%)	266.00 (31.29%) 11,331,680.88 (26.63%)				
missing	28.00 (1.21%) 1,289,335.85 (1.40%)	11.00 (1.29%) 152,342.86 (0.36%)				

Table 27
Bivariate Analyses of Selected Independent Variable Pairs Among Childbearing-Aged Participants (unweighted and weighted data 1999-2004)

Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30					χ^2 *p value* *unweighted*	χ^2 *p value* *weighted*
	Seafood Meals (smpw2cat)					
Protein Intake/AMDR (prot3cat)	none^{R}	any				
recommended or more^{R}	598.00 (87.17%) 19,990,155.11 (87.40%)	2,114.00 (85.00%) 98,773,610.16 (88.48%)			2.04 *0.153*	0.07 *0.792*
less than recommended	88.00 (12.83%) 2,880,685.67 (12.60%)	373.00 (15.00%) 12,857,582.49 (11.52%)				
	Seafood Meals (smpw2cat)					
Selenium Intake/RDA (sele2cat)	none^{R}	any				
recommended or more^{R}	528.00 (76.97%) 17,412,417.38 (76.13%)	2,031.00 (81.66) 95,266,065.47 (85.34%)			7.60 *0.0058*	3.86 *0.056*
less than recommended	158.00 (23.03%) 5,458,423.40 (23.87%)	456.00 (18.34%) 16,365,127.18 (14.66%)				
	Trimester of Pregnancy (tripcorr)					
Current Pregnancy (pregnant)	not pregnant^{R}	1st trimester	2nd trimester	3rd trimester		
pregnant $n_{unweighted}$ = 391 $n_{weighted}$ = 4,842,189.09	0.00 (0.00%) 0.00 (0.00%)	149.00 (99.33%) 1,991,566.11 (100.00%)	132.00 (100.00%) 1,523,495.53 (100.00%)	110.00 (100.00%) 1,327,127.44 (100.00%)	3,173.00 *<0.0001*	16.06 *0.0000*
not pregnant^{R}	2,641.00 (94.93%) 1,26376,518.94 (93.96%)	0.00 (0.00%) 0.00 (0.00%)	0.00 (0.00%) 0.00 (0.00%)	0.00 (0.00%) 0.00 (0.00%)		
missing	141.00 (5.07%) 3,283,325.41 (2.44%)	0.00 (0.00%) 0.00 (0.00%)	0.00 (0.00%) 0.00 (0.00%)	0.00 (0.00%) 0.00 (0.00%)		
	Live Births (live)					
Ever Pregnant (tprg2cat)	no live births^{R}	one or more live births				
never pregnant^{R}	1,535.00 (84.34%) 59,565,096.98 (88.35%)	0.00 (0.00%) 0.00 (0.00%)			2,210.50 *<0.0001*	182.17 *0.0000*
one or more pregnancies	285.00 (15.66%) 7,855,141.24 (11.65%)	1,353.00 (100.00%) 67,081,795.22 (100.00%)				

Table 27
Bivariate Analyses of Selected Independent Variable Pairs Among Childbearing-Aged Participants (unweighted and weighted data 1999-2004)

Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30					χ^2 *p value* *unweighted*	χ^2 *p value* *weighted*
	Ever Breastfed (brstfda)					
Live Births (live)	never breastfed[R]	breastfed more than one month or currently				
no live births[R]	1,820.00 (78.35%) 67,420,238.21 (50.13%)	0.00 (0.00%) 0.00 (0.00%)			1561.76 *<0.0001*	130.73 *0.0000*
one or more live births	503.00 (21.65%) 24,534,084.08 (26.68%)	850.00 (100.00%) 42,547,711.13 (100.00%)				
	Language Spoken at Home (lang2cat)					
Years in U.S. (yrus5)	English[R]	Other				
born in U.S.[R]	2,648.00 (93.14%) 119,574,469.41 (94.37%)	* (0.00%) 502,160.04 (6.79%)			1749.39 *<0.0001*	56.38 *0.0000*
five or more years	173.00 (6.09%) 6,274,037.58 (4.95%)	179.00 (55.59%) 4,799,481.63 (64.88%)				
less than five years	* (0.00%) 859,771.07 (0.68%)	120.00 (37.27%) 2,096,195.84 (28.34%)				
	U.S. Citizenship (usczn2cat)					
Years in U.S. (yrus5)	U.S. citizen[R]	non-U.S. citizen				
born in U.S.[R]	2,673.00 (95.06%) 120,303,696.80 (94.93%)	0.00 (0.00%) 0.00 (0.00%)			2277.87 *<0.0001*	37.43 *0.0000*
five or more years	133.00 (4.73%) 6,126,948.25 (4.83%)	219.00 (61.69%) 4,946,570.95 (64.99%)				
less than five years	* (0.00%) 291,572.56 (0.23%)	136.00 (38.31%) 2,664,394.36 (35.01%)				
	U.S. Citizenship (usczn2cat)					
Language Spoken at Home (lang2cat)	U.S. citizen[R]	non-U.S. citizen				
English[R]	2,734.00 (97.23%) 124,256,122.31 (98.15%)	110.00 (30.73%) 2,505,072.05 (32.73%)			1522.13 *<0.0001*	77.39 *0.0000*
Other	78.00 (2.77%) 2,342,082.25 (1.85%)	248.00 (69.27%) 5,149,820.78 (67.27%)				

Table 27
Bivariate Analyses of Selected Independent Variable Pairs Among Childbearing-Aged Participants (unweighted and weighted data 1999-2004)

Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30					χ^2 *p value* *unweighted*	χ^2 *p value* *weighted*
	Fish Eaten in Past 30 Days (fish2cat)					
Shellfish Eaten in Past 30 Days (shell2cat)	none^{R}	any				
none^{R}	686.00 (65.96%) 22,870,840.78 (62.13%)	871.00 (40.83%) 40,147,798.40 (41.10%)			176.62 *<0.0001*	11.03 *0.0018*
any	354.00 (34.04%) 13,938,898.89 (37.87%)	1,262.00 (59.17%) 57,544,495.36 (58.90%)				
	Fish Eaten in Past 30 Days (fish2cat)					
Seafood Eaten in Past 30 Days (smpw2cat)	none^{R}	any				
none^{R}	686.00 (65.9%) 22,870,840.78 (62.13%)	0.00 (0.00%) 0.00 (0.00%)			1795.05 *<0.0001*	110.57 *0.0000*
any	354.00 (34.04%) 13,938,898.89 (37.87%)	2,133.00 (100.00%) 97,692,293.76 (100.00%)				
	Shellfish Eaten in Past 30 Days (shell2cat)					
Seafood Eaten in Past 30 Days (smpw2cat)	none^{R}	any				
none^{R}	686.00 (44.06%) 22,870,840.78 (36.29%)	0.00 (0.00%) 0.00 (0.00%)			908.39 *<0.0001*	92.67 *0.0000*
any	871.00 (55.94%) 40,147,798.40 (63.71%)	1,616.00 (100.00%) 71,483,394.25 (100.00%)				
	Tap Water Source (h2os2cat)					
Tap Water Consumed past 24h (tap2kct)	public^{R}	private	missing			
none^{R}	975.00 (34.50%) 33,223,428.63 (28.46%)	113.00 (42.48%) 6,025,957.02 (41.58%)	41.00 (50.62%) 1,255,442.59 (38.34%)			
< 2,000 ml	1,390.00 (49.19%) 63,041,240.20 (54.00%)	122.00 (45.86%) 6,595,092.42 (45.51%)	* (0.00%) 1,409,152.80 (43.03%)		23.57 *0.0006*	4.03 *0.0026*
2,000+ ml	263.00 (9.31%) 13,414,568.57 (11.49%)	* (0.00%) 1,701,154.02 (11.74%)	* (0.00%) 413,829.90 (12.64%)			
missing	198.00 (7.01%) 7,056,670.76 (6.04%)	* (0.00%) 169,331.56 (1.17%)	* (0.00%) 196,164.94 (5.99%)			

Table 27
Bivariate Analyses of Selected Independent Variable Pairs Among Childbearing-Aged Participants (unweighted and weighted data 1999-2004)

Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30					χ^2 *p value* *unweighted*	χ^2 *p value* *weighted*
	Residential Tap Water Treatment (h2ox2cat)					
Tap Water Consumed past 24h (tap2kct)	yes	no[R]	missing			
none[R]	213.00 (24.68%) 8,657,607.21 (19.11%)	888.00 (39.66%) 30,854,229.99 (35.65%)	* (0.00%) 992,991.04 (37.49%)			
< 2,000 ml	522.00 (60.49%) 30,205,324.44 (66.67%)	981.00 (43.81%) 39,474,226.06 (45.61%)	35.00 (49.30%) 1,365,934.92 (51.57%)		89.81 *<0.0001*	5.50 *0.0003*
2,000+ ml	89.00 (10.31%) 4,986,577.49 (11.01%)	205.00 (9.16%) 10,521,106.33 (12.16%)	* (0.00%) 21,868.68 (0.83%)			
missing	39.00 (4.52%) 1,458,725.52 (3.22%)	165.00 (7.37%) 5,695,774.98 (6.58%)	7.00 (9.86%) 267,666.76 (10.11%)			
	Serum Cotinine (cot3cat)					
Alcohol Consumption (retohuse)	< 1.0 ng/ml[R]	1.0 - 10.0 ng/ml	> 10.0 ng/ml			
never, seldom drinker[R] *including 16-19 y/o*	1,362.00 (57.52%) 41,883,173.64 (42.36%)	130.00 (68.42%) 2,207,900.23 (42.05%)	245.00 (40.83%) 7,996,189.95			
drinker	581.00 (24.54%) 31,478,549.38 (31.84%)	* (0.00%) 1,642,802.71 (31.29%)	113.00 (18.83%) 7,057,861.37 (23.72%)		192.77 *<0.0001*	2.74 *0.024*
heavy drinker	312.00 (13.18%) 20,883,010.05 (21.12%)	* (0.00%) 1,199,798.28 (22.85%)	218.00 (36.33%) 13,682,571.10 (45.99%)			
missing	113.00 (4.77%) 4,626,740.51 (4.68%)	* (0.00%) 199,800.39 (3.81%)	* (0.00%) 1,013,718.81 (3.41%)			
	Environmental Tobacco Smoke (ETS)					
Serum Cotinine (cot3cat)	no ETS[R]	ETS at home or at work	ETS at home and at work			
< 1.0 ng/ml[R]	2,040.00 (84.93%) 87,163,820.06 (86.16%)	309.00 (47.54%) 10,705,253.64 (40.08%)	* (0.00%) 1,002,399.86 (16.71%)			
1.0 - 10.0 ng/ml	107.00 (4.45%) 2,124,105.13 (2.10%)	78.00 (12.00%) 3,055,931.78 (11.44%)	* (0.00%) 70,264.71 (1.17%)		628.17 *<0.0001*	23.15 *0.0000*
> 10.0 ng/ml	255.00 (10.62%) 11,879,529.38 (11.74%)	263.00 (40.46%) 12945684.18 (48.47%)	82.00 (77.36%) 4,925,127.68 (82.12%)			

Table 27
Bivariate Analyses of Selected Independent Variable Pairs Among Childbearing-Aged Participants (unweighted and weighted data 1999-2004)

Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30					χ^2 *p value unweighted*	χ^2 *p value weighted*
	Self-Reported Tobacco Use (tobuse)					
Serum Cotinine (cot3cat)	age restricted^{R}	never	former	current		
< 1.0 ng/ml^{R}	830.00 (75.39%) 13,874,581.99 (74.01%)	1,248.00 (94.19%) 68,473,062.62 (94.76%)	257.00 (86.53%) 14,629,369.30 (88.96%)	33.00 (7.59%) 1,894,459.64 (7.17%)		
1.0 - 10.0 ng/ml	100.00 (9.08%) 1,221,535.37 (6.52%)	56.00 (4.23%) 2,615,754.04 (3.62%)	* (0.00%) 461,855.17 (2.81%)	* (0.00%) 951,157.01 (3.60%)	1,724.60 *<0.0001*	21.59 *0.0000*
> 10.0 ng/ml	171.00 (15.53%) 3,649,817.08 (19.47%)	* (0.00%) 1,171,855.77 (1.62%)	* (0.00%) 1,354,453.22 (8.24%)	387.00 (88.97%) 23,574,215.16 (89.23%)		
	Residential Tap Water Treatment (h2ox2cat)					
Tap Water Source (h2os2cat)	yes	no^{R}	missing			
public^{R}	706.00 (81.81%) 37,562,745.23 (82.90%)	2,097.00 (93.66%) 78,629,947.58 (90.85%)	* (0.00%) 543,215.36 (20.51%)		1,314.81 *<0.0001*	3.06 *0.026*
private	153.00 (17.73%) 7,422,870.19 (16.38%)	112.00 (5.00%) 6,989,566.08 (8.08%)	* (0.00%) 79,098.75 (2.99%)			
missing	* (0.00%) 322,619.25 (0.71%)	30.00 (1.34%) 925,823.70 (1.07%)	47.00 (66.20%) 2,026,147.29 (76.50%)			
	Resident Status (resd3cat)					
Type of Residence (res3cat)	own^{R}	rent	other *including missing*			
attached or detached house^{R}	1,548.00 (89.64%) 69,420,478.62 (89.86%)	476.00 (37.25%) 18,097,932.14 (35.37%)	48.00 (28.57%) 1,787,559.80 (29.36%)		1,291.91 *<0.0001*	24.07 *0.0000*
mobile home or trailer	143.00 (8.28%) 6,233,638.41 (8.07%)	50.00 (3.91%) 1,873,766.07 (3.66%)	* (0.00%) 294,373.32 (4.84%)			
all other types *including missing/unknown*	36.00 (2.08%) 1,596,190.10 (2.07%)	752.00 (58.84%) 31,192,197.36 (60.97%)	111.00 (66.07%) 4,005,897.61 (65.80%)			
	Resident Status (resd3cat)					
Age of Residence (resb60cat)	own^{R}	rent	other *including missing*			
1960 or newer^{R}	1,138.00 (65.89%) 55,261,058.05 (71.54%)	410.00 (32.08%) 21,314,206.66 (41.66%)	47.00 (27.98%) 1,469,259.30 (24.13%)		651.29 *<0.0001*	21.57 *0.0000*
older than 1960	449.00 (26.00%) 18,303,204.02 (23.69%)	265.00 (20.74%) 11,664,723.07 (22.80%)	52.00 (30.95%) 2,124,273.01 (34.89%)			
missing/unknown	140.00 (8.11%) 3,686,045.05 (4.77%)	603.00 (47.18%) 18,184,965.84 (35.54%)	69.00 (41.07%) 2,494,298.42 (40.97%)			

Table 27
Bivariate Analyses of Selected Independent Variable Pairs Among Childbearing-Aged Participants (unweighted and weighted data 1999-2004)

Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30					χ^2 *p value* *unweighted*	χ^2 *p value* *weighted*
	Resident Status (resd3cat)					
Years at Current Residence (re5yrcat)	own^{R}	rent	other *including missing*			
more than five years^{R}	897.00 (51.94%) 39,199,292.03 (50.74%)	189.00 (14.79%) 6,094,286.85 (11.91%)	* (0.00%) 600,739.80 (9.87%)		1,342.88 *<0.0001*	25.54 *0.0000*
five years or less	830.00 (48.06%) 38,051,015.10 (49.26%)	1,087.00 (85.05%) 45,001,738.00 (87.96%)	90.00 (53.57%) 3,402,859.84 (55.90%)			
missing	0.00 (0.00%) 0.00 (0.00%)	* (0.00%) 67,870.72 (0.13%)	51.00 (30.36%) 2,084,231.09 (34.24%)			
	Rooms in Residence (rm3cat)					
Household Size (hsize)	7+ rooms^{R}	4-6 rooms	1-3 rooms	missing		
four persons or less^{R}	683.00 (59.49%) 36,468,109.07 (69.31%)	1207.00 (71.38%) 58,206,284.87 (84.23%)	233.00 (88.59%) 9,435,232.73 (92.73%)	59.00 (83.10%) 2,344,401.71 (89.82%)	106.24 *<0.0001*	7.96 *0.0002*
more than four persons	465.00 (40.51%) 16,148,403.74 (30.69%)	484.00 (28.62%) 10,893,847.72 (15.77%)	30.00 (11.41%) 739,940.38 (7.27%)	* (0.00%) 265,813.21 (10.18%)		
	Time in Current Employment (cjt)					
Current Occupation (cocc2cat)	not working^{R}	less than five years	five or more years			
not working^{R}	1,324.00 (100.00%) 42,172,957.57 (100.00%)	0.00 (0.00%) 0.00 (0.00%)	0.00 (0.00%) 0.00 (0.00%)		3194.79 *<0.0001*	59.82 *0.0000*
management, professional & sales	0.00 (0.00%) 0.00 (0.00%)	934.00 (65.13%) 47,316,425.78 (70.37%)	309.00 (74.46%) 20,442,465.96 (81.48%)			
services & goods	0.00 (0.00%) 0.00 (0.00%)	500.00 (34.87%) 19,925,213.95 (29.63%)	106.00 (25.54%) 4,644,970.16 (18.52%)			
	Total Hours Worked Prior Week (hrwk)					
Current Occupation (cocc2cat)	not worked^{R}	less than 35 hours	35+ hours			
not working^{R}	1,323.00 (95.80%) 42,151,088.89 (92.02%)	0.00 (0.00%) 0.00 (0.00%)	0.00 (0.00%) 0.00 (0.00%)		2959.42 *<0.0001*	57.79 *0.0000*
management, professional & sales	58.00 (4.20%) 3,656,949.14 (7.98%)	460.00 (62.50%) 21,926,910.36 (65.71%)	725.00 (68.79%) 42,175,032.24 (76.43%)			
services & goods	0.00 (0.00%) 0.00 (0.00%)	276.00 (37.50%) 11,440,523.43 (34.29%)	329.00 (31.21%) 13,006,488.77 (23.57%)			

Table 27
Bivariate Analyses of Selected Independent Variable Pairs Among Childbearing-Aged Participants (unweighted and weighted data 1999-2004)

Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30					χ^2 *p value* *unweighted*	χ^2 *p value* *weighted*
	Time in Longest Employment (ljt)					
Longest Held Occupation (locc2cat)	not applicable^{R}	less than five years	five or more years			
not applicable^{R}	1,562.00 (100.00%) 64,117,356.51 (100.00%)	0.00 (0.00%) 0.00 (0.00%)	0.00 (0.00%) 0.00 (0.00%)		3191.73 *<0.0001*	92.59 *0.0000*
management, professional & sales	0.00 (0.00%) 0.00 (0.00%)	529.00 (53.06%) 19,500,975.82 (56.46%)	374.00 (60.91%) 21,914,210.33 (61.14%)			
services & goods	0.00 (0.00%) 0.00 (0.00%)	468.00 (46.94%) 1,504,1293.03 (43.54%)	240.00 (39.09%) 13,928,197.74 (38.86%)			
	Current Occupation (cocc2cat)					
Work History (wkcp)	not working^{R}	management, professional & sales	services & goods			
never employed^{R}	408.00 (30.82%) 8,238,810.80 (19.54%)	0.00 (0.00%) 0.00 (0.00%)	0.00 (0.00%) 0.00 (0.00%)			
currently employed	0.00 (0.00%) 0.00 (0.00%)	804.00 (64.68%) 42,694,958.49 (63.01%)	350.00 (57.76%) 13,183,587.23 (53.66%)		3187.29 *<0.0001*	44.91 *0.0000*
employed in the past but not currently	916.00 (69.18%) 33,934,146.78 (80.46%)	0.00 (0.00%) 0.00 (0.00%)	0.00 (0.00%) 0.00 (0.00%)			
employed now and in the past	0.00 (0.00%) 0.00 (0.00%)	439.00 (35.32%) 25,063,933.25 (36.99%)	256.00 (42.24%) 11,386,596.89 (46.34%)			
	Longest Held Occupation (locc2cat)					
Work History (wkcp)	not applicable^{R}	management, professional & sales	services & goods			
never employed^{R}	408.00 (26.12%) 8,238,810.80 (12.85%)	0.00 (0.00%) 0.00 (0.00%)	0.00 (0.00%) 0.00 (0.00%)			
currently employed	1,154.00 (73.88%) 55,878,545.72 (87.15%)	0.00 (0.00%) 0.00 (0.00%)	0.00 (0.00%) 0.00 (0.00%)		3,173.13 *<0.0001*	65.91 *0.0000*
employed in the past but not currently	0.00 (0.00%) 0.00 (0.00%)	516.00 (57.14%) 21,792,025.47 (52.62%)	400.00 (56.50%) 12,142,121.30 (41.91%)			
employed now and in the past	0.00 (0.00%) 0.00 (0.00%)	387.00 (42.86%) 19,623,160.67 (47.38%)	308.00 (43.50%) 16,827,369.47 (58.09%)			

Table 27
Bivariate Analyses of Selected Independent Variable Pairs Among Childbearing-Aged Participants (unweighted and weighted data 1999-2004)

Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30					χ^2 *p value unweighted*	χ^2 *p value weighted*
	Employment Status (emp3cat)					
Highest Education (educ2)	employed	not employed[R]				
high school diploma, GED or higher[R]	1,428.00 (77.06%) 79,230,888.20 (85.68%)	609.00 (46.24%) 27,676,273.14 (65.97%)			318.45 *<0.0001*	32.44 *0.0000*
less than high school diploma	425.00 (22.94%) 13,237,911.77 (14.32%)	708.00 (53.76%) 14,278,602.02 (34.03%)				
	Reason for Unemployment (unem2cat)					
Highest Education (educ2)	working[R]	voluntary unemployment	involuntary unemployment	missing		
high school diploma, GED or higher[R]	1,428.00 (77.06%) 79,230,888.20 (85.68%)	378.00 (40.91%) 17,810,827.03 (63.01%)	165.00 (56.12%) 6,863,476.19 (67.32%)	66.00 (65.35%) 3,001,969.92 (85.65%)	359.99 *<0.0001*	14.28 *0.0000*
less than high school diploma	425.00 (22.94%) 13,237,911.77 (14.32%)	546.00 (59.09%) 10,454,194.64 (36.99%)	129.00 (43.88%) 3,332,499.03 (32.68%)	35.00 (34.65%) 503,127.87 (14.35%)		
	Reason for Unemployment (unem2cat)					
Employment Status (emp3cat)	working[R]	voluntary unemployment	involuntary unemployment	missing		
employed	1,853.00 (100.00%) 92,468,799.96 (100.00%)	0.00 (0.00%) 0.00 (0.00%)	0.00 (0.00%) 0.00 (0.00%)	0.00 (0.00%) 0.00 (0.00%)	3,171.00 *<0.0001*	69.89 *0.0000*
not employed[R]	0.00 (0.00%) 0.00 (0.00%)	924.00 (100.00%) 28,265,021.67 (100.00%)	295.00 (100.00%) 10,263,114.01 (100.00%)	99.00 (100.00%) 3,493,878.27 (100.00%)		
	Marital Status (marr3cat)					
U.S. Poverty Threshold (pov2cat)	married or living with partner	widowed, divorced or separated	never married[R]	missing		
more than 1.00[R]	962.00 (80.30%) 53,319,206.48 (86.28%)	152.00 (58.24%) 9,842,029.42 (68.57%)	1,049.00 (64.08%) 36,871,187.98 (68.93%)	64.00 (83.12%) 3,921,199.31 (80.77%)	129.06 *<0.0001*	3.33 *0.0086*
1.00 or less	162.00 (13.52%) 5,800,026.31 (9.39%)	91.00 (34.87%) 3,840,416.45 (26.76%)	471.00 (28.77%) 12,750,736.01 (23.84%)	* (0.00%) 196,018.69 (4.04%)		
missing	74.00 (6.18%) 2,681,415.46 (4.34%)	* (0.00%) 671,506.51 (4.68%)	117.00 (7.15%) 3,871,027.49 (7.24%)	7.00 (9.09%) 737,263.30 (15.19%)		

Table 27
Bivariate Analyses of Selected Independent Variable Pairs Among Childbearing-Aged Participants (unweighted and weighted data 1999-2004)

Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30					χ^2 *p value unweighted*	χ^2 *p value weighted*
	Highest Education (educ2)					
U.S. Poverty Threshold (pov2cat)	high school diploma, GED or higher^{R}	less than high school diploma				
more than 1.00^{R}	1,562.00 (76.68%) 87,575,534.18 (81.92%)	664.00 (58.50%) 16,310,950.22 (59.25%)			115.63 *<0.0001*	3.08 *0.056*
1.00 or less	362.00 (17.77%) 13,492,269.81 (12.62%)	368.00 (32.42%) 9,094,927.65 (33.04%)				
missing	113.00 (5.55%) 5,839,357.35 (5.46%)	103.00 (9.07%) 2,121,855.42 (7.71%)				
	Current Occupation (cocc2cat)					
U.S. Poverty Threshold (pov2cat)	not working^{R}	management, professional & sales	services & goods			
more than 1.00^{R}	754.00 (56.95%) 27,435,957.82 (65.06%)	1,061.00 (85.36%) 59,864,335.85 (88.35%)	412.00 (67.99%) 16,653,329.52 (67.78%)		255.96 *<0.0001*	12.27 *0.0000*
1.00 or less	455.00 (34.37%) 11,977,284.37 (28.40%)	125.00 (10.06%) 4,212,539.78 (6.22%)	150.00 (24.75%) 6,397,373.32 (26.04%)			
missing	115.00 (8.69%) 2,759,715.38 (6.54%)	57.00 (4.59%) 3,682,016.11 (5.43%)	44.00 (7.26%) 1,519,481.28 (6.18%)			
	Household Size (hsize)					
U.S. Poverty Threshold (pov2cat)	four persons or less^{R}	more than four persons				
more than 1.00^{R}	143.00 (6.55%) 6,871,248.02 (6.45%)	73.00 (7.37%) 1,089,964.75 (3.89%)			58.17 *<0.0001*	2.77 *0.074*
1.00 or less	1618.00 (74.15%) 83,822,831.95 (78.74%)	609.00 (61.45%) 20,130,791.24 (71.77%)				
missing	421.00 (19.29%) 15,759,948.42 (14.80%)	309.00 (31.18%) 6827249.05 (24.34%)				
	Reason for Unemployment (unem2cat)					
U.S. Poverty Threshold (pov2cat)	working^{R}	voluntary unemployment	involuntary unemployment	missing		
more than 1.00^{R}	1,476.00 (79.65%) 76,635,520.79 (82.88%)	536.00 (58.01%) 18,956,878.10 (67.07%)	147.00 (49.83%) 5,920,332.22 (57.69%)	68.00 (67.33%) 2,440,892.08 (69.64%)	211.91 *<0.0001*	3.40 *0.007*
1.00 or less	275.00 (14.84%) 10,609,913.10 (11.47%)	312.00 (33.77%) 7,475,095.63 (26.45%)	114.00 (38.64%) 3,526,666.48 (34.36%)	* (0.00%) 975,522.25 (27.83%)		
missing	102.00 (5.50%) 5,223,366.07 (5.65%)	76.00 (8.23%) 1,833,047.95 (6.49%)	34.00 (11.53%) 816,115.30 (7.95%)	4.00 (3.96%) 88,683.45 (2.53%)		

Table 27
Bivariate Analyses of Selected Independent Variable Pairs Among Childbearing-Aged Participants (unweighted and weighted data 1999-2004)

Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30					χ^2 *p value unweighted*	χ^2 *p value weighted*
	Highest Education (educ2)					
Race-Ethnicity (race5cat)	high school diploma, GED or higher^{R}	less than high school diploma				
Non-Hispanic White^{R}	1,173.00 (57.58%) 81,668,719.25 (76.39%)	320.00 (28.19%) 16,218,824.91 (58.92%)				
Non-Hispanic Black	349.00 (17.13%) 9,102,591.03 (8.51%)	274.00 (24.14%) 3,644,587.34 (13.24%)			327.27 *<0.0001*	8.13 *0.0001*
Mexican American	316.00 (15.51%) 4,617,923.82 (4.32%)	429.00 (37.80%) 4,052,651.98 (14.72%)				
Other Hispanic	94.00 (4.61%) 5,003,783.20 (4.68%)	83.00 (7.31%) 2,455,070.23 (8.92%)				
Asian, Native American, Pacific Islander & Multi-Racial	105.00 (5.15%) 6.514.144.04 (6.09%)	* (0.00%) 1,156,598.84 (4.20%)				
	Reason for Unemployment (unem2cat)					
Race-Ethnicity (race5cat)	working^{R}	voluntary unemployment	involuntary unemployment	missing		
Non-Hispanic White^{R}	990.00 (53.43%) 69,427,978.22 (75.08%)	341.00 (36.90%) 19,176,171.18 (67.84%)	125.00 (42.37%) 6,764,445.01 (65.91%)	37.00 (36.63%) 2,518,949.76 (71.87%)		
Non-Hispanic Black	319.00 (17.22%) 8,156,696.99 (8.82%)	176.00 (19.05%) 2,047,744.12 (7.24%)	94.00 (31.86%) 1,966,882.49 (19.16%)	34.00 (33.66%) 575,854.77 (16.43%)	152.76 *<0.0001*	6.17 *0.0000*
Mexican American	367.00 (19.81%) 4,764,599.82 (5.15%)	308.00 (33.33%) 3,138,379.77 (11.10%)	51.00 (17.29%) 495,733.47 (4.83%)	* (0.00%) 271,862.74 (7.76%)		
Other Hispanic	83.00 (4.48%) 4,176,772.64 (4.52%)	69.00 (7.47%) 2,648,536.68 (9.37%)	* (0.00%) 639,990.34 (6.24%)	* (0.00%) 60,692.55 (1.73%)		
Asian, Native American, Pacific Islander & Multi-Racial	94.00 (5.07%) 5,942,752.30 (6.43%)	30.00 (3.25%) 1,254,189.93 (4.44%)	* (0.00%) 396,062.70 (3.86%)	* (0.00%) 77,737.96 (2.22%)		

Table 27
Bivariate Analyses of Selected Independent Variable Pairs Among Childbearing-Aged Participants (unweighted and weighted data 1999-2004)

Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30					χ^2 *p value* *unweighted*	χ^2 *p value* *weighted*
	U.S. Poverty Threshold (pov2cat)					
Race-Ethnicity (race5cat)	more than 1.00^{R}	1.00 or less	missing			
Non-Hispanic White^{R}	1,179.00 (52.94%) 78,846,347.78 (75.85%)	231.00 (31.64%) 13,908,178.21 (61.58%)	83.00 (38.43%) 5,133,018.17 (64.48%)			
Non-Hispanic Black	363.00 (16.30%) 8,204,067.35 (7.89%)	205.00 (28.08%) 3,541,288.29 (15.68%)	55.00 (25.46%) 1,001,822.73 (12.58%)		117.68 *<0.0001*	2.26 *0.040*
Mexican American	481.00 (21.60%) 5,845,858.36 (5.62%)	211.00 (28.90%) 2,204,434.75 (9.76%)	53.00 (24.54%) 620,282.70 (7.79%)			
Other Hispanic	117.00 (5.25%) 5,034,661.04 (4.84%)	44.00 (6.03%) 1,630,523.78 (7.22%)	* (0.00%) 860,807.40 (10.81%)			
Asian, Native American, Pacific Islander & Multi-Racial	87.00 (3.91%) 6,022,688.67 (5.79%)	39.00 (5.34%) 1,302,772.44 (5.77%)	* (0.00%) 345,281.77 (4.34%)			
	Age (age4cat)					
Race-Ethnicity/Hispanic Grouping (race4cat)	16-19^{R}	20-29	30-39	40-49		
Non-Hispanic White^{R}	367.00 (33.82%) 2,396,253.18 (66.94%)	514.00 (58.14%) 5825897.52 (74.76%)	364.00 (51.85%) 2,950,223.53 (69.17%)	248.00 (49.40%) 3,574,174.26 (77.13%)		
Non-Hispanic Black	247.00 (22.76%) 1,714,116.00 (9.26%)	145.00 (16.40%) 3,746,815.73 (8.26%)	119.00 (16.95%) 3,760,796.13 (10.34%)	112.00 (22.31%) 3,525,450.50 (10.28%)	154.45 *<0.0001*	2.14 *0.0454*
Hispanic	430.00 (39.63%) 3,378,004.42 (18.25%)	193.00 (21.83%) 5,626,135.77 (12.41%)	176.00 (25.07%) 4,650,483.61 (12.79%)	124.00 (24.70%) 2,541,944.23 (7.41%)		
Asian, Native American, Pacific Islander & Multi-Racial	41.00 (3.78%) 1,027,208.52 (5.55%)	32.00 (3.62%) 2,071,845.43 (4.57%)	43.00 (6.13%) 2,796,130.81 (7.69%)	* (0.00%) 1,775,558.12 (5.18%)		
	Health Insurance (hi2cat)					
Race-Ethnicity/Hispanic Grouping (race4cat)	private^{R}	public	none	missing		
Non-Hispanic White^{R}	1,178.00 (57.69%) 78,720,618.81 (78.62%)	107.00 (24.37%) 4,273,726.43 (43.65%)	171.00 (27.63%) 12,766,445.38 (58.66%)	37.00 (50.68%) 2,126,753.55 (75.55%)		
Non-Hispanic Black	341.00 (16.70%) 7,770,696.56 (7.76%)	157.00 (35.76%) 2,391,338.58 (24.42%)	109.00 (17.61%) 2,356,978.10 (10.83%)	* (0.00%) 228,165.13 (8.12%)	379.04 *<0.0001*	14.58 *0.0000*
Hispanic	445.00 (21.79%) 8,321,761.47 (8.31%)	143.00 (32.57%) 2,264,254.21 (23.13%)	320.00 (51.70%) 5,257,453.41 (24.16%)	* (0.00%) 353,098.93 (12.54%)		
Asian, Native American, Pacific Islander & Multi-Racial	78.00 (3.82%) 5,319,701.64 (5.31%)	32.00 (7.30%) 862,100.86 (8.81%)	* (0.00%) 1,381,927.47 (6.35%)	* (0.00%) 107,012.90 (3.80%)		

Table 27
Bivariate Analyses of Selected Independent Variable Pairs Among Childbearing-Aged Participants (unweighted and weighted data 1999-2004)

Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30					χ^2 *p value* *unweighted*	χ^2 *p value* *weighted*
	Food Security (food2cat)					
Race-Ethnicity/Hispanic Grouping (race4cat)	food secure[R]	food insecure	missing			
Non-Hispanic White[R]	1,301.00 (50.58%) 85,394,002.15 (74.88%)	121.00 (26.36%) 8,336,128.60 (58.56%)	71.00 (50.00%) 4,157,413.41 (66.80%)			
Non-Hispanic Black	479.00 (18.62%) 10,346,994.84 (9.07%)	124.00 (27.02%) 2,041,509.69 (14.34%)	* (0.00%) 358,673.84 (5.76%)		124.63 *<0.0001*	5.36 *0.0003*
Hispanic	684.00 (26.59%) 11,941,499.37 (10.47%)	203.00 (44.22%) 3,529,387.56 (24.79%)	36.00 (25.36%) 725,681.09 (11.66%)			
Asian, Native American, Pacific Islander & Multi-Racial	108.00 (4.21%) 6,361,416.29 (5.58%)	* (0.00%) 327,496.20 (2.30%)	* (0.00%) 981,830.38 (15.77%)			
	Body Mass Index (bmi30cat)					
Race-Ethnicity/Hispanic Grouping (race4cat)	<30.0[R] underweight normal overweight	30.0+ obese	missing			
Non-Hispanic White[R]	1,205.00 (51.13%) 78,241,008.88 (76.08%)	271.00 (34.88%) 18,701,542.56 (61.89%)	* (0.00%) 944,992.72 (65.55%)			
Non-Hispanic Black	374.00 (15.87%) 6,845,949.44 (6.66%)	238.00 (30.63%) 5,681,300.24 (%)	* (0.00%) 219,928.68 (15.26%)		109.52 *<0.0001*	6.29 *0.0001*
Hispanic	665.00 (28.21%) 11,319,384.11 (11.01%)	247.00 (31.79%) 4,600,426.61 (15.23%)	* (0.00%) 276,757.30 (19.19%)			
Asian, Native American, Pacific Islander & Multi-Racial	113.00 (4.79%) 6,437,554.49 (6.26%)	* (0.000%) 1,233,188.39 (4.08%)	0.00 (0.00%) 0.00 (0.00%)			
	Serum Cotinine (cot3cat)					
Race-Ethnicity/Hispanic Grouping (race4cat)	< 1.0 ng/ml[R]	1.0 - 10.0 ng/ml	> 10.0 ng/ml			
Non-Hispanic White[R]	1,094.00 (46.20%) 72,627,347.15 (73.46%)	51.00 (26.84%) 2,624,966.84 (49.99%)	343.00 (57.17%) 22,168,404.25 (74.52%)			
Non-Hispanic Black	413.00 (17.44%) 8,121,811.18 (8.21%)	74.00 (38.95%) 1,040,809.38 (%)	129.00 (21.50%) 3,435,662.74 (%)		121.11 *<0.0001*	5.07 *0.0005*
Hispanic	767.00 (32.39%) 13,036,768.80 (13.19%)	48.00 (25.26%) 885,077.90 (16.86%)	105.00 (17.50%) 2,260,525.28 (7.59%)			
Asian, Native American, Pacific Islander & Multi-Racial	94.00 (3.97%) 5,085,546.44 (5.14%)	* (0.00%) 699,447.48 (13.32%)	* (0.00%) 1,885,748.96 (6.34%)			
missing = 15						

Table 27
Bivariate Analyses of Selected Independent Variable Pairs Among Childbearing-Aged Participants (unweighted and weighted data 1999-2004)

Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30					χ^2 *p value unweighted*	χ^2 *p value weighted*
	Type of Residence (res3cat)					
Race-Ethnicity/Hispanic Grouping (race4cat)	attached or detached house^{R}	mobile home or trailer	all other types *including missing/unknown*			
Non-Hispanic White^{R}	1,016.00 (49.03%) 67,876,940.99 (76.00%)	102.00 (50.50%) 6,356,324.95 (75.65%)	375.00 (41.71%) 23654278.21 (64.29%)			
Non-Hispanic Black	404.00 (19.50%) 7,961,266.03 (8.92%)	* (0.00%) 562,549.01 (6.69%)	194.00 (21.58%) 4,223,363.33 (11.48%)		33.37 *<0.0001*	1.35 *0.257*
Hispanic	585.00 (28.23%) 9,340,713.89 (10.46%)	67.00 (33.17%) 1,024,914.57 (12.19%)	271.00 (30.14%) 5,830,939.55 (%)			
Asian, Native American, Pacific Islander & Multi-Racial	67.00 (3.23%) 4,127,049.64 (4.62%)	* (0.00%) 457,989.26 (5.45%)	59.00 (6.56%) 3,085,703.98 (8.39%)			
	Age of Residence (resb60cat)					
Race-Ethnicity/Hispanic Grouping (race4cat)	1960 or newer^{R}	older than 1960	missing or unknown			
Non-Hispanic White^{R}	885.00 (55.48%) 59,496,716.52 (76.23%)	407.00 (53.13%) 25,276,967.64 (78.76%)	201.00 (24.75%) 13,113,860.01 (53.82%)			
Non-Hispanic Black	256.00 (16.05%) 5,812,871.98 (7.45%)	128.00 (16.71%) 2,230,733.61 (6.95%)	239.00 (29.43%) 4,703,572.77 (19.30%)		246.39 *<0.0001*	5.96 *0.0001*
Hispanic	370.00 (23.20%) 7,654,076.36 (9.81%)	210.00 (27.42%) 3,681,581.81 (11.47%)	343.00 (42.25%) 4,860,909.85 (19.95%)			
Asian, Native American, Pacific Islander & Multi-Racial	84.00 (5.27%) 5,080,859.15 (6.51%)	* (0.00%) 902,917.05 (2.81%)	* (0.00%) 1,686,966.68 (6.92%)			
	Age of Residence (resb78cat)					
Race-Ethnicity/Hispanic Grouping (race4cat)	1978 or newer^{R}	older than 1978	missing or unknown			
Non-Hispanic White^{R}	630.00 (57.96%) 42,439,346.81 (76.62%)	662.00 (51.96%) 42,334,337.34 (77.32%)	201.00 (24.75%) 13,113,860.01 (53.82%)			
Non-Hispanic Black	166.00 (15.27%) 4,043,746.25 (7.30%)	218.00 (17.11%) 3,999,859.33 (7.31%)	239.00 (29.43%) 4,703,572.77 (19.30%)		243.57 *<0.0001*	6.63 *0.0000*
Hispanic	243.00 (22.36%) 5,493,448.81 (9.92%)	337.00 (26.45%) 5,842,209.36 (10.67%)	343.00 (42.25%) 4,860,909.85 (19.95%)			
Asian, Native American, Pacific Islander & Multi-Racial	48.00 (4.42%) 3,411,506.97 (6.16%)	57.00 (4.47%) 2,572,269.22 (4.69%)	* (0.00%) 1,686,966.68 (6.92%)			

Table 27
Bivariate Analyses of Selected Independent Variable Pairs Among Childbearing-Aged Participants (unweighted and weighted data 1999-2004)

Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30					χ^2 *p value* *unweighted*	χ^2 *p value* *weighted*
	Resident Status (resd3cat)					
Race-Ethnicity/Hispanic Grouping (race4cat)	own^{R}	rent	other *including missing*			
Non-Hispanic White^{R}	927.00 (53.68%) 60,696,837.19 (78.57%)	464.00 (36.31%) 32,092,120.18 (62.72%)	102.00 (60.71%) 5,098,586.78 (83.75%)			
Non-Hispanic Black	262.00 (15.17%) 5,104,082.44 (6.61%)	341.00 (26.68%) 7,470,697.69 (14.60%)	* (0.00%) 172,398.23 (2.83%)		126.54 *<0.0001*	3.57 *0.006*
Hispanic	485.00 (28.08%) 8,125,715.77 (10.52%)	400.00 (31.30%) 7,461,604.12 (14.58%)	38.00 (22.63%) 609,248.13 (10.01%)			
Asian, Native American, Pacific Islander & Multi-Racial	53.00 (3.07%) 3,323,671.71 (4.30%)	73.00 (5.71%) 4,139,473.57 (8.09%)	* (0.00%) 207,597.59 (3.41%)			
	Total Hours Worked Prior Week (hrwk)					
Race-Ethnicity/Hispanic Grouping (race4cat)	not employed^{R}	less than 35 hours	35+ hours			
Non-Hispanic White^{R}	534.00 (38.67%) 30,936,387.14 (67.54%)	426.00 (57.88%) 27,636,151.10 (82.83%)	532.00 (50.47%) 39,191,833.99 (71.02%)			
Non-Hispanic Black	318.00 (23.03%) 5,066,780.38 (11.06%)	92.00 (12.50%) 1,595,025.75 (4.78%)	213.00 (20.21%) 6,085,372.23 (11.03%)			
Hispanic	482.00 (34.90%) 7,506,773.31 (16.39%)	188.00 (25.54%) 3,120,063.49 (9.35%)	252.00 (23.91%) 5,547,862.54 (10.05%)		103.63 *<0.0001*	5.76 *0.0002*
Asian, Native American, Pacific Islander & Multi-Racial	47.00 (35.07%) 2,298,097.19 (5.02%)	* (0.00%) 1,016,193.45 (3.04%)	57.00 (5.41%) 4,356,452.24 (7.89%)			
missing = 2						
	Marital Status (marr3cat)					
Race-Ethnicity/Hispanic Grouping (race4cat)	married or living with partner	widowed, divorced or	never married^{R}	missing		
Non-Hispanic White^{R}	685.00 (57.18%) 49,217,838.59 (79.64%)	103.00 (39.46%) 8,249,274.17 (57.47%)	671.00 (40.99%) 37,539,926.15 (70.18%)	34.00 (44.16%) 2,880,505.23 (59.34%)		
Non-Hispanic Black	130.00 (10.85%) 3,510,070.41 (5.68%)	72.00 (27.59%) 2,437,997.53 (16.98%)	408.00 (24.92%) 6,303,355.06 (11.78%)	* (0.00%) 495,755.36 (10.21%)	141.39 *<0.0001*	4.77 *0.0002*
Hispanic	337.00 (28.13%) 6,468,838.53 (10.47%)	67.00 (25.67%) 1,867,408.96 (13.01%)	498.00 (30.42%) 7,095,260.43 (13.26%)	* (0.00%) 765,060.08 (15.76%)		
Asian, Native American, Pacific Islander & Multi-Racial	46.00 (3.84%) 2,603,900.71 (4.21%)	* (0.00%) 1,799,271.71 (12.53%)	60.00 (3.67%) 2,554,409.84 (4.78%)	* (0.00%) 713,160.62 (14.69%)		

Table 27
Bivariate Analyses of Selected Independent Variable Pairs Among Childbearing-Aged Participants (unweighted and weighted data 1999-2004)

Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30					χ^2 *p value unweighted*	χ^2 *p value weighted*
	Highest Education (educ2)					
Race-Ethnicity/Hispanic Grouping (race4cat)	high school diploma, GED or higher[R]	less than high school diploma				
Non-Hispanic White[R]	1,173.00 (57.58%) 81,668,719.25 (76.39%)	320.00 (28.19%) 16,218,824.91 (58.92%)				
Non-Hispanic Black	349.00 (17.13%) 9,102,591.03 (8.51%)	274.00 (24.14%) 3,644,587.34 (13.24%)			320.16 *<0.0001*	7.34 *0.0004*
Hispanic	410.00 (20.13%) 9,621,707.02 (9.00%)	512.00 (45.11%) 6,507,722.21 (23.64%)				
Asian, Native American, Pacific Islander & Multi-Racial	105.00 (5.15%) 6.514.144.04 (6.09%)	* (0.00%) 1,156,598.84 (4.20%)				
	Reason for Unemployment (unem2cat)					
Race-Ethnicity/Hispanic Grouping (race4cat)	working[R]	voluntary unemployment	involuntary unemployment	missing		
Non-Hispanic White[R]	990.00 (53.43%) 69,427,978.22 (75.08%)	341.00 (36.90%) 19,176,171.18 (67.84%)	125.00 (42.37%) 6,764,445.01 (65.91%)	37.00 (36.63%) 2,518,949.76 (71.87%)		
Non-Hispanic Black	319.00 (17.22%) 8,156,696.99 (8.82%)	176.00 (19.05%) 2,047,744.12 (7.24%)	94.00 (31.86%) 1,966,882.49 (19.16%)	34.00 (33.66%) 575,854.77 (16.43%)	148.29 *<0.0001*	4.95 *0.0001*
Hispanic	450.00 (24.28%) 8,941,372.46 (9.67%)	377.00 (40.80%) 5,786,916.45 (20.47%)	68.00 (23.05%) 1,135,723.81 (11.07%)	28.00 (27.72%) 332,555.30 (9.49%)		
Asian, Native American, Pacific Islander & Multi-Racial	94.00 (5.07%) 5,942,752.30 (6.43%)	30.00 (3.25%) 1,254,189.93 (4.44%)	* (0.00%) 396,062.70 (3.86%)	* (0.00%) 77,737.96 (2.22%)		
	U.S. Poverty Threshold (pov2cat)					
Race-Ethnicity/Hispanic Grouping (race4cat)	more than 1.00[R]	1.00 or less	missing			
Non-Hispanic White[R]	1,179.00 (52.94%) 78,846,347.78 (75.85%)	231.00 (31.64%) 13,908,178.21 (61.58%)	83.00 (38.43%) 5,133,018.17 (64.48%)			
Non-Hispanic Black	363.00 (16.30%) 8,204,067.35 (7.89%)	205.00 (28.08%) 3,541,288.29 (15.68%)	55.00 (25.46%) 1,001,822.73 (12.58%)		115.59 *<0.0001*	2.63 *0.029*
Hispanic	598.00 (26.85%) 10,880,519.39 (10.47%)	255.00 (34.93%) 3,834,958.53 (16.98%)	70.00 (32.41%) 1,481,090.10 (18.60%)			
Asian, Native American, Pacific Islander & Multi-Racial	87.00 (3.91%) 6,022,688.67 (5.79%)	39.00 (5.34%) 1,302,772.44 (5.77%)	* (0.00%) 345,281.77 (4.34%)			

Table 28
Univariate Analyses of Xenobiotic Blood Levels Prior to Logarithmic Transformation (unweighted data[1] 1999 - 2004)

Childbearing-Aged Participants

Dependent Variable Components	Univariate Statistics (µg/dl)									Percentiles							
Specific Chemicals of Interest µg/dl sample (n)	Arithmetic Mean	Median	Mode	Arithmetic Standard Deviation	Skewness	Range	Minimum	Maximum	Variance	10th	25th	50th	75th	90th	95th	99th	>99th
Lead n = 3,173	1.13	0.90	0.70	1.01	7.18	23.70	0.20	23.90	1.03	0.5 527	0.6 841	0.9 1,649	1.3 2,476	1.9 2,877	2.6 3,021	4.6 3,144	3,173
Methylmercury n = 3,173	1.16	0.52	0.22	1.85	5.42	31.20	0.00	31.20	3.41	0.1 335	0.223 1,083	0.52 1,629	1.32 2,403	2.92 2,857	4.5 3,021	8.3 3,145	3,173
Sum of Lipid-Adjusted PCB Congeners n = 3,173	53.30	41.00	19.00	57.94	7.12	1,442.00	4.56	1,447.00	3,357.22	14.3 312	20.8 794	41 1,597	63 2,383	103.3 2,857	144.2 3,015	255.7 3,143	3,173

Pregnant Childbearing-Aged Participants

Dependent Variable Components	Univariate Statistics (µg/dl)									Percentiles							
Specific Chemicals of Interest µg/dl sample (n)	Arithmetic Mean	Median	Mode	Arithmetic Standard Deviation	Skewness	Range	Minimum	Maximum	Variance	10th	25th	50th	75th	90th	95th	99th	>99th
Lead	0.88	0.70	0.50	0.62	2.85	4.80	0.20	5.00	0.39	0.4 75	0.5 128	0.7 200	1.1 300	1.4 356	2 375	3.6 388	391
Methylmercury	1.06	0.42	0.22	1.71	3.38	11.80	0.00	11.80	2.92	0.1 41	0.2 106	0.42 206	1.1 295	2.82 356	4.7 373	9.2 388	391
Sum of Lipid-Adjusted PCB Congeners	39.96	31.80	47.80	34.58	4.35	372.00	4.60	377.20	1,196.00	13.2 47	20 99	31.8 200	47.8 296	70.06 352	93.2 373	222.8 388	391

[1] no missing data

Table 29
Prevalence Rates for Individual Xenobiotic Blood Levels for Childbearing-Aged Female Participants and Pregnant Childbearing-Aged Participants Prior to Logarithmic Transformation (unweighted data[1] 1999 - 2004)

	Childbearing-Aged Female Participants			Pregnant Childbearing-Aged Participants		
Specific Chemicals of Interest	Below Detection	At Detection[2]	Above Detection	Below Detection	At Detection[2]	Above Detection
Lead LBXBPB	1.9 per 100	2.2 per 100	95.9 per 100	5.1 per 100	3.1 per 100	91.8 per 100
Total Mercury LBXTHG	4.2 per 100	4.2 per 100	91.6 per 100	2.6 per 100	3.8 per 100	93.6 per 100
Inorganic Mercury LBXIHG	86.5 per 100	3.5 per 100	10.0 per 100	89.3 per 100	2.8 per 100	7.9 per 100
PCB 118 LBX118	39.7 per 100	NA	60.3 per 100	41.2 per 100	NA	58.8 per 100
PCB 138 LBX138	30.1 per 100	NA	69.9 per 100	32.7 per 100	NA	67.3 per 100
PCB 153 LBX153	26.6 per 100	NA	73.4 per 100	28.4 per 100	NA	71.6 per 100
PCB 180 LBX180	33.1 per 100	NA	66.9 per 100	37.6 per 100	NA	62.4 per 100
	Less Than Zero	Equal to Zero	Greater Than Zero	Less Than Zero	Equal to Zero	Greater Than Zero
MeHg (THg - IHg)	15 per 100	3.5 per 100	81.5 per 100	12.5 per 100	3.9 per 100	83.6 per 100

[1] with no missing data

[2] PCBs' Levels of Detection was sample-specific

NA = Not Available

Table 30
Univariate Analyses of Xenobiotic Blood Levels Post Logarithmic Transformation (unweighted and weighted data[1] 1999 - 2004)

Childbearing-Aged Females

Dependent Variable Components	Univariate Statistics (µg/dl)									Percentiles							
Sample unweighted weighted cumulative unweighted cumulative weighted	Geometric Mean	Median	Mode	Geometric Standard Deviation	Skewness	Range	Minimum	Maximum	Variance	10th	25th	50th	75th	90th	95th	99th	>99th
										-0.7	-0.5	-0.1	0.3	0.6	1.0	1.5	
										236	230	905	1,044	401	125	142	90
										8,677,332.81	8,808,667.99	37,256,412.64	50,936,423.22	16,271,954.52	5,231,194.99	4,232,471.45	3,087,575.82
										236	466	1,371	2,415	2,816	2,941	3,083	3,173
Lead	-0.08	-0.11	-0.36	0.60	0.36	4.78	-1.61	3.17	0.36	8,677,332.81	17,486,000.80	54,742,413.33	105,678,836.66	121,950,791.18	127,181,986.17	131,414,457.62	134,502,033.43
										0.1	0.2	0.4	0.8	1.4	1.7	2.2	
										219	568	842	774	449	161	132	28
										7,779,275.63	20,251,205.53	32,552,938.25	35,330,269.85	21,246,299.64	7,992,994.37	7,335,270.10	2,013,780.06
										219	787	1,629	2,403	2,852	3,013	3,145	3,173
Methylmercury	0.59	0.42	0.20	0.53	1.37	3.47	0.00	3.47	0.28	7,779,275.63	28,030,481.16	60,583,419.41	95,913,689.26	117,159,988.90	125,152,983.27	132,488,253.37	134,502,033.43
										2.7	3.0	3.7	4.1	4.6	5.0	5.5	
										322	449	813	799	470	162	126	32
										6,636,249.80	12,106,578.89	30,090,732.22	38,358,029.62	28,557,239.62	9,962,450.92	7,044,651.91	1,746,100.44
										322	771	1,584	2,383	2,853	3,015	3,141	3,173
Sum of Lipid-Adjusted PCB Congeners	3.65	3.71	2.98	0.78	0.23	5.76	1.52	7.28	0.60	6,636,249.80	18,742,828.69	48,833,560.91	87,191,590.53	115,748,830.15	125,711,281.07	132,755,932.98	134,502,033.43

Pregnant Childbearing-Aged Females

Dependent Variable Components	Univariate Statistics (µg/dl)									Percentiles							
Sample unweighted weighted cumulative unweighted cumulative weighted	Geometric Mean	Median	Mode	Geometric Standard Deviation	Skewness	Range	Minimum	Maximum	Variance	10th	25th	50th	75th	90th	95th	99th	>99th
										-0.9	-0.7	-0.4	0.1	0.3	0.7	1.3	
										12	43	84	141	41	34	13	
										87,634.48	390,879.19	1,079,815.88	1,937,996.05	360,736.20	405,690.39	145,829.38	23
										12	55	139	280	321	355	368	433,607.51
Lead	-0.31	-0.36	-0.69	0.60	0.10	3.22	-1.61	1.61	0.36	87,634.48	478,513.67	1,558,329.55	3,496,325.60	3,857,061.80	4,262,752.19	4,408,581.57	391
										0.1	0.2	0.4	0.7	1.3	1.7	2.3	
										29	77	81	104	58	21	18	3
										377,332.60	1,006,043.78	823,682.43	978,770.39	923,548.88	125,708.86	416,958.43	190,143.72
										29	106	187	291	349	370	388	391
Methylmercury	0.55	0.35	0.20	0.52	1.59	2.55	0.00	2.55	0.27	377,332.60	1,383,376.38	2,207,058.81	3,185,829.20	4,109,378.08	4,235,086.94	4,652,045.37	4,842,189.09
										2.6	3.0	3.5	3.9	4.2	4.5	5.4	
										47	52	101	96	56	21	15	3
										368,954.11	555,265.47	1,189,387.49	973,056.76	1,011,276.31	328,088.20	394,968.28	21,192.47
										47	99	200	296	352	373	388	391
Sum of Lipid-Adjusted PCB Congeners	3.46	3.46	3.87	0.66	0.17	4.41	1.53	5.93	0.44	368,954.11	924,219.58	2,113,607.07	3,086,663.83	4,097,940.14	4,426,028.34	4,820,996.62	4,842,189.09

[1] no missing data

Table 31
Prevalence Rates for Individual Xenobiotic Blood Levels for Childbearing-Aged Females and Pregnant Childbearing-Aged Females
(unweighted[1] and weighted data 1999 - 2004)

Sample Population (unweighted)

	Childbearing-Aged Female Participants			Pregnant Childbearing-Aged Participants		
Specific Chemicals of Interest	Below Geometric Mean		At or Above Geometric Mean	Below Geometric Mean		At or Above Geometric Mean
Lead LBXBPB	51.97 per 100		48.03 per 100	66.49 per 100		33.51 per 100
MeHg (THg - IHg)	60.23 per 100		39.77 per 100	67.01 per 100		32.99 per 100
PCBs (sumPCBla)	47.05 per 100		52.95 per 100	60.11 per 100		39.89 per 100

Study Population (weighted)

	Childbearing-Aged Females			Pregnant Childbearing-Aged Females		
Specific Chemicals of Interest	Below Geometric Mean		At or Above Geometric Mean	Below Geometric Mean		At or Above Geometric Mean
Lead LBXBPB	50.95 per 100		49.05 per 100	74.81 per 100		25.19 per 100
MeHg (THg - IHg)	52.55 per 100		47.45 per 100	59.42 per 100		32.99 per 100
PCBs (sumPCBla)	33.46 per 100		66.54 per 100	50.15 per 100		49.85 per 100

[1] with no missing data

Table 34
Exposure as Outcome in Four Categories: Number of Childbearing-Aged Females with Xenobiotic Blood Levels Below, At or Above the Geometric Mean
(unweighted and weighted data 1999-2004)

Dependent Variable Components Specific Chemicals of Interest Sample Frequency $_{unweighted}$ Col. Pct. Population Estimated Frequency $_{weighted}$ Col. Pct.	0	1	2	3	Total	χ^2 *p value* *unweighted*	χ^2 *p value* *weighted*
Lead							
Below Geometric Mean $_{unweighted}$	702.00 (100.00%)	600.00 (61.79%)	347.00 (34.53%)	0.00 (0.00%)	1,649.00 (51.97%)		
Below Geometric Mean $_{weighted}$	20,889,388.72 (100.00%)	25,299,379.85 (71.92%)	22,339,886.83 (43.63%)	0.00 (0.00%)	68,528,655.40 (50.95%)	1,344.41 *<0.0001*	49.30 *0.0000*
At or Above Geometric Mean $_{unweighted}$	0.00 (0.00%)	371.00 (38.21%)	658.00 (65.47%)	495.00 (100.00%)	1,524.00 (48.03%)		
At or Above Geometric Mean $_{weighted}$	0.00 (0.00%)	9,875,692.09 (28.08%)	28,865,899.17 (56.37%)	27,231,786.77 (100.00%)	65,973,378.03 (49.05%)		
Methylmercury							
Below Geometric Mean $_{unweighted}$	702.00 (100.00%)	742.00 (76.42%)	467.00 (46.47%)	0.00 (0.00%)	1,911.00 (60.23%)		
Below Geometric Mean $_{weighted}$	20,889,388.72 (100.00%)	27,957,025.03 (79.48%)	21,838,880.31 (42.65%)	0.00 (0.00%)	70,685,294.07 (52.55%)	1,398.82 <0.0001	75.63 *0.0000*
At or Above Geometric Mean $_{unweighted}$	0.00 (0.00%)	229.00 (23.58%)	538.00 (53.53%)	495.00 (100.00%)	1,262.00 (39.77%)		
At or Above Geometric Mean $_{weighted}$	0.00 (0.00%)	7,218,046.91 (20.52%)	29,366,905.69 (57.35%)	27,231,786.77 (100.00%)	63,816,739.36 (47.45%)		
Sum of PCBs							
Below Geometric Mean $_{unweighted}$	702.00 (100.00%)	600.00 (61.79%)	191.00 (19.00%)	0.00 (0.00%)	1,493.00 (47.05%)		
Below Geometric Mean $_{weighted}$	20,889,388.72 (100.00%)	17,093739.00 (48.60%)	7,027,018.85 (13.72%)	0.00 (0.00%)	45,010,146.57 (33.46%)	1,631.85 *<0.0001*	35.44 *0.0000*
At or Above Geometric Mean $_{unweighted}$	0.00 (0.00%)	371.00 (38.21%)	814.00 (81.00%)	495.00 (100.00%)	1,680.00 (52.95%)		
At or Above Geometric Mean $_{weighted}$	0.00 (0.00%)	18,081,332.94 (51.40%)	44,178,767.15 (86.28%)	27,231,786.77 (100.00%)	89,491,886.86 (66.54%)		

0 = all blood levels of lead, methylmercury and PCBs are below geometric mean (GM)
1 = one blood level is at or above geometric mean and two are below the geometric mean
2 = two blood levels are at or above geometric mean and one blood level is below geometric mean
3 = all blood levels of lead, methylmercury and PCBs are at or above geometric mean

Table 35

Exposure as Outcome in Four Categories: Number of Pregnant Childbearing-Aged Females with Xenobiotic Blood Levels Below, At or Above the Geometric Mean (unweighted and weighted data 1999-2004)

Total Number of Chemicals At or Above Geometric Mean Sample Frequency $_{unweighted}$ Col. Pct. Population Estimated Frequency $_{weighted}$ Col. Pct. * = cell size < 30	0	1	2	3	Total	χ^2 *p value unweighted*	χ^2 *p value weighted*
Lead							
Below Geometric Mean $_{unweighted}$	137.00	67.00	56.00	0.00	260.00		
	(100.00%)	(56.78%)	(50.91%)	(0.00%)	(66.49%)		
Below Geometric Mean $_{weighted}$	1,575,513.12	753,583.23	1,292,921.09	0.00	3,622,017.44		
	(100.00%)	(61.21%)	(74.38%)	(0.00%)	(74.80%)	137.63	10.27
At or Above Geometric Mean $_{unweighted}$	0.00	51.00	54.00	*	131.00	*<0.0001*	*0.0000*
	(0.00%)	(43.22%)	(49.09%)	(100.00%)	(33.51%)		
At or Above Geometric Mean $_{weighted}$	0.00	477,587.57	445,452.07	297,132.00	1,220,171.64		
	(0.00%)	(38.79%)	(25.62%)	(100.00%)	(25.20%)		
Methylmercury							
Below Geometric Mean $_{unweighted}$	137.00	85.00	40.00	0.00	262.00		
	(100.00%)	(72.03%)	(36.36%)	(0.00%)	(67.00%)		
Below Geometric Mean $_{weighted}$	1,575,513.12	991,992.10	309,519.74	0.00	2,877,024.96		
	(100.00%)	(80.57%)	(17.81%)	(0.00%)	(59.42%)	168.33	10.66
At or Above Geometric Mean $_{unweighted}$	0.00	33.00	70.00	*	129.00	*<0.0001*	*0.0000*
	(0.00%)	(27.97%)	(63.64%)	(100.00%)	(33.00%)		
At or Above Geometric Mean $_{weighted}$	0.00	239,178.70	1,428,853.42	297,132.00	1,965,164.12		
	(0.00%)	(19.43%)	(82.19%)	(100.00%)	(40.58%)		
Sum of PCBs							
Below Geometric Mean $_{unweighted}$	137.00	84.00	*	0.00	235.00		
	(100.00%)	(71.19%)	(0.00%)	(0.00%)	(60.10%)		
Below Geometric Mean $_{weighted}$	1,575,513.12	716,766.28	135,932.33	0.00	2,428,211.73		
	(100.00%)	(58.22%)	(7.82%)	(0.00%)	(50.15%)	239.11	13.53
At or Above Geometric Mean $_{unweighted}$	0.00	34.00	96.00	*	156.00	*<0.0001*	*0.0000*
	(0.00%)	(28.81%)	(87.27%)	(100.00%)	(39.90%)		
At or Above Geometric Mean $_{weighted}$	0.00	514,404.53	1,602,440.84	297,132.00	2,413,977.37		
	(0.00%)	(41.78%)	(92.18%)	(100.00%)	(49.85%)		

0 or 1 = all blood levels of lead, methylmercury and PCBs are below geometric mean OR one blood level is at or above geometric mean and two blood levels are below geometric mean

2 or 3 = two blood levels are at or above geometric mean and one blood level is below geometric mean OR all blood levels of lead, methylmercury and PCBs are at or above geometric mean

Table 36

Exposure as Outcome in Two Categories: Number of Childbearing-Aged Females with Xenobiotic Blood Levels Below, At or Above the Geometric Mean

(unweighted and weighted data 1999-2004)

Dependent Variable Components Specific Chemicals of Interest Sample Frequency $_{unweighted}$ Col. Pct. Population Estimated Frequency $_{weighted}$ Col. Pct.	0 and 1		2 and 3		Total	χ^2 *p value* *unweighted*	χ^2 *p value* *weighted*
Lead							
Below Geometric Mean $_{unweighted}$	1,302.00 (77.82%)		347.00 (23.13%)		1,649.00 (51.97%)	947.72 *<0.0001*	101.39 *0.0000*
Below Geometric Mean $_{weighted}$	46,188,768.57 (82.39%)		22,339,886.83 (28.48%)		68,528,655.40 (50.95%)		
At or Above Geometric Mean $_{unweighted}$	371.00 (22.18%)		1,153.00 (76.87%)		1,524.00 (48.03%)		
At or Above Geometric Mean $_{weighted}$	9,875,692.09 (17.61%)		56,097,685.94 (71.52%)		65,973,378.03 (49.05%)		
Methylmercury							
Below Geometric Mean $_{unweighted}$	1,444.00 (86.31%)		467.00 (31.13%)		1,911.00 (60.23%)	1,005.26 *<0.0001*	120.71 *0.0000*
Below Geometric Mean $_{weighted}$	48,846,413.75 (87.13%)		21,838,880.31 (27.84%)		70,685,294.07 (52.55%)		
At or Above Geometric Mean $_{unweighted}$	229.00 (13.69%)		1,033.00 (68.87%)		1,262.00 (39.77%)		
At or Above Geometric Mean $_{weighted}$	7,218,046.91 (12.87%)		56,598,692.46 (72.16%)		63,816,739.36 (47.45%)		
Sum of PCBs							
Below Geometric Mean $_{unweighted}$	1,302.00 (77.82%)		191.00 (12.73%)		1,493.00 (47.05%)	1,345.02 *<0.0001*	79.07 *0.0000*
Below Geometric Mean $_{weighted}$	37,983,127.71 (67.75%)		7,027,018.85 (8.96%)		45,010,146.57 (33.46%)		
At or Above Geometric Mean $_{unweighted}$	371.00 (22.18%)		1,309.00 (87.27%)		1,680.00 (52.95%)		
At or Above Geometric Mean $_{weighted}$	18,081,332.94 (32.25%)		71,410,553.92 (91.04%)		89,491,886.86 (66.54%)		

0 or 1 = all blood levels of lead, methylmercury and PCBs are below geometric mean OR
one blood level is at or above geometric mean and two blood levels are below geometric mean

2 or 3 = two blood levels are at or above geometric mean and one blood level is below geometric mean OR
all blood levels of lead, methylmercury and PCBs are at or above geometric mean

Table 37

Exposure as Outcome in Two Categories: Number of Pregnant Childbearing-Aged Females with Xenobiotic Blood Levels Below, At or Above the Geometric Mean (unweighted and weighted data 1999-2004)

Total Number of Chemicals At or Above Geometric Mean Sample Frequency $_{unweighted}$ Col. Pct. Population Estimated Frequency $_{weighted}$ Col. Pct. * = cell size < 30	0 and 1		2 and 3		Total	χ^2 *p value unweighted*	χ^2 *p value weighted*
Lead							
Below Geometric Mean $_{unweighted}$	204.00		56.00		260.00		
	(80.00%)		(41.18%)		(66.49%)		
Below Geometric Mean $_{weighted}$	2,329,096.35		1,292,921.09		3,622,017.44		
	(%)		(%)		(74.80%)		
At or Above Geometric Mean $_{unweighted}$	51.00		80.00		131.00	60.01	4.86
	(20.00%)		(58.82%)		(33.51%)	*<0.0001*	*0.033*
At or Above Geometric Mean $_{weighted}$	477,587.57		742,584.07		1,220,171.64		
	(9%)		(%)		(25.20%)		
Methylmercury							
Below Geometric Mean $_{unweighted}$	222.00		40.00		262.00		
	(87.06%)		(29.41%)		(67.00%)		
Below Geometric Mean $_{weighted}$	256,505.22		309,519.74		2,877,024.96		
	(51.75%)		(15.21%)		(59.42%)	133.33	24.07
At or Above Geometric Mean $_{unweighted}$	33.00		96.00		129.00	*<0.0001*	*0.0000*
	(12.94%)		(70.59%)		(33.00%)		
At or Above Geometric Mean $_{weighted}$	239,178.70		1,725,985.42		1,965,164.12		
	(48.25%)		(84.79%)		(40.58%)		
Sum of PCBs							
Below Geometric Mean $_{unweighted}$	221.00		*		235.00		
	(86.67%)		(0.00%)		(60.10%)		
Below Geometric Mean $_{weighted}$	2,292,279.40		135,932.33		2,428,211.73		
	(81.67%)		(6.68%)		(50.15%)	215.74	30.62
At or Above Geometric Mean $_{unweighted}$	34.00		122.00		156.00	*<0.0001*	*0.0000*
	(13.33%)		(89.71%)		(39.90%)		
At or Above Geometric Mean $_{weighted}$	514,404.53		1,899,572.84		2,413,977.37		
	(18.33%)		(93.32%)		(49.85%)		

0 or 1 = all blood levels of lead, methylmercury and PCBs are below geometric mean OR one blood level is at or above geometric mean and two blood levels are below geometric mean

2 or 3 = two blood levels are at or above geometric mean and one blood level is below geometric mean OR all blood levels of lead, methylmercury and PCBs are at or above geometric mean

Table 38
Estimated Prevalence Rates of Xenobiotic Blood Levels
Among Childbearing-Aged Female Participants (unweighted 1999 - 2004)

Individual Chemicals: Xenobiotic Blood Levels Below, At or Above Geometric Mean	Prevalence Rates Below Geometric Mean	Prevalence Rates At or Above Geometric Mean
Lead	51.97 per 100	48.03 per 100
Methylmercury	60.23 per 100	39.77 per 100
PCBs	47.05 per 100	52.95 per 100
Combinations of Chemicals: Both Concurrently At or Above Geometric Mean		Prevalence Rates
Lead and Methylmercury		54.36 per 100
Methylmercury and PCBs		50.12 per 100
PCBs and Lead		57.26 per 100
Exposure as Four Categories: Number of Chemicals Concurrently At or Above Geometric Mean		Prevalence Rates
0		22.12 per 100
1		30.60 per 100
2		31.67 per 100
3		15.60 per 100
Exposure as Two Categories: Number of Chemicals Concurrently At or Above Geometric Mean		Prevalence Rates
0 or 1		52.73 per 100
2 or 3		47.27 per 100

n = 3,173

Table 39
Estimated Prevalence Rates of Xenobiotic Blood Levels
Among Childbearing-Aged Females in U.S. (weighted 1999 - 2004)

Individual Chemicals: Xenobiotic Blood Levels Below, At or Above Geometric Mean	Prevalence Rates Below Geometric Mean	Prevalence Rates At or Above Geometric Mean
Lead	50.95 per 100	49.05 per 100
Methylmercury	52.55 per 100	47.45 per 100
PCBs	33.46 per 100	66.54 per 100
Combinations of Chemicals: Both Concurrently At or Above Geometric Mean		Prevalence Rates
Lead and Methylmercury		53.68 per 100
Methylmercury and PCBs		55.39 per 100
PCBs and Lead		74.38 per 100
Exposure as Four Categories: Number of Chemicals Concurrently At or Above Geometric Mean		Prevalence Rates
0		15.53 per 100
1		26.15 per 100
2		38.07 per 100
3		20.25 per 100
Exposure as Two Categories: Number of Chemicals Concurrently At or Above Geometric Mean		Prevalence Rates
0 or 1		41.68 per 100
2 or 3		58.32 per 100

n = 134,502,033.43

Table 40
Estimated Prevalence Rates of Xenobiotic Blood Levels
Among Pregnant Childbearing-Aged Participants (unweighted 1999 - 2004)

Individual Chemicals: Xenobiotic Blood Levels Below, At or Above Geometric Mean	Prevalence Rates Below Geometric Mean	Prevalence Rates At or Above Geometric Mean
Lead	66.49 per 100	33.51 per 100
Methylmercury	67.01 per 100	32.99 per 100
PCBs	60.11 per 100	39.89 per 100
Combinations of Chemicals: Both Concurrently At or Above Geometric Mean		Prevalence Rates
Lead and Methylmercury		31.01 per 100
Methylmercury and PCBs		52.56 per 100
PCBs and Lead		50.38 per 100
Exposure as Four Categories: Number of Chemicals Concurrently At or Above Geometric Mean		Prevalence Rates
0		19.52 per 100
1		12.15 per 100
2		10.95 per 100
3		*
Exposure as Two Categories: Number of Chemicals Concurrently At or Above Geometric Mean		Prevalence Rates
0 or 1		15.24 per 100
2 or 3		5.01 per 100

n = 391

Table 41
Estimated Prevalence Rates of Xenobiotic Blood Levels
Among Pregnant Childbearing-Aged Females in U.S. (weighted 1999 - 2004)

Individual Chemicals: Xenobiotic Blood Levels Below, At or Above Geometric Mean	Prevalence Rates Below Geometric Mean	Prevalence Rates At or Above Geometric Mean
Lead	74.81 per 100	25.19 per 100
Methylmercury	59.42 per 100	32.99 per 100
PCBs	50.15 per 100	49.85 per 100
Combinations of Chemicals: Both Concurrently At or Above Geometric Mean		Prevalence Rates
Lead and Methylmercury		22.04 per 100
Methylmercury and PCBs		65.87 per 100
PCBs and Lead		49.72 per 100
Exposure as Four Categories: Number of Chemicals Concurrently At or Above Geometric Mean		Prevalence Rates
0		7.54 per 100
1		3.50 per 100
2		3.39 per 100
3		1.09 per 100
Exposure as Two Categories: Number of Chemicals Concurrently At or Above Geometric Mean		Prevalence Rates
0 or 1		9.07 per 100
2 or 3		2.60 per 100

n = 4,842,189.09

Table 42
Bivariate Analyses of Independent Variables on Exposure as Outcome with Two Categories Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables with Exposure Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30	0 and 1		2 and 3		χ^2 *p value unweighted*	χ^2 *p value weighted*
Susceptibility-Related Attributes						
Age (age4cat)						
16-19^{R}	781.00 (46.68%) 14,660,569.05 (26.15%)		304.00 (20.27%) 3,849,899.68 (4.91%)			
20-29	520.00 (31.08%) 23,750,605.62 (42.36%)		364.00 (24.27%) 21,596,909.29 (27.53%)		446.40 *<0.0001*	18.38 *0.0000*
30-39	275.00 (16.44%) 12,303,495.51 (21.95%)		427.00 (28.47%) 24,054,340.99 (30.67%)			
40-49	97.00 (5.80) 5,349,790.48 (9.54%)		405.00 (27.00%) 28,936,422.82 (36.89%)			
Health Status						
Perceived Health Status (huq2cat)						
excellent, very good, good^{R}	1,523.00 (91.03%) 51,900,967.08 (92.57%)		1,317.00 (87.86%) 72,104,278.01 (91.96%)		8.51 *0.0035*	0.15 *0.696*
fair, poor	150.00 (8.97%) 4,163,493.57 (7.43%)		182.00 (12.14%) 6,302,386.02 (8.04%)			
Charleson Co-Morbidity Scale (CCMS3cat)						
none^{R}	1,480.00 (88.46%) 48,153,165.73 (85.89%)		1,334.00 (88.93%) 70,103,855.70 (89.38%)			
one co-morbidity	173.00 (10.34%) 6,875,028.46 (12.26%)		130.00 (8.67%) 6,271,704.85 (8.00%)		8.84 *0.012*	1.08 *0.347*
more than one co-morbidity	* (0.00%) 1,036,266.46 (1.85%)		36.00 (2.40%) 2,062,012.23 (2.63%)			
Iron Deficiency (FeD2cat)						
within normal limits^{R}	1,421.00 (84.94%) 51,036,315.36 (91.03%)		1,303.00 (86.87%) 71,800,443.27 (91.54%)		2.42 *0.119*	0.07 *0.791*
iron deficient	252.00 (15.06%) 5,028,145.30 (8.97%)		197.00 (13.13%) 6,637,129.51 (8.46%)			
Treatment for Iron Deficiency past 3 mo (FeTx2cat)						
yes	93.00 (5.56%) 2,101,128.65 (3.75%)		78.00 (5.20%) 3,045,167.32 (3.88%)		0.19 *0.658*	0.005 *0.942*
no^{R}	1,580.00 (94.44%) 53,963,332.01 (96.25%)		1,421.00 (94.80%) 75,378,826.38 (96.12%)			

Table 42
Bivariate Analyses of Independent Variables on Exposure as Outcome with Two Categories Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables with Exposure Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30	0 and 1		2 and 3		χ^2 *p value* *unweighted*	χ^2 *p value* *weighted*
Iron Deficiency and Treatment (FeDTx)						
	1,357.00		1,251.00			
	(81.11%)		(83.46%)			
	49,651,905.68		69,790,793.00			
normal/no treatment^{R}	(88.56%)		(88.99%)			
	64.00		51.00			
	(3.83%)		(3.40%)			
	1,384,409.68		1,996,071.19		3.46	0.54
normal w/treatment	(2.47%)		(2.55%)		*0.326*	*0.983*
	*		*			
	(0.00%)		(0.00%)			
	716,718.97		1,049,096.13			
deficient w/treatment	(1.28%)		(1.34%)			
	223.00		170.00			
	(13.33%)		(11.34%)			
	4,311,426.33		5,588,033.38			
deficient/no treatment	(7.69%)		(7.13%)			
Health Insurance (hi2cat)						
	1,053.00		989.00			
	(62.94%)		(65.93%)			
	41,460,120.89		58,672,657.58			
private^{R}	(73.95%)		(74.80%)			
	267.00		172.00			
	(15.96%)		(11.47%)			
	4,425,605.00		5,365,815.08		14.85	0.19
public	(7.89%)		(6.84%)		*0.002*	*0.901*
	311.00		308.00			
	(18.59%)		(20.53%)			
	8,880,950.82		12,881,853.55			
none	(15.84%)		(16.42%)			
	42.00		31.00			
	(2.51%)		(2.07%)			
	1,297,783.95		1,517,246.57			
missing	(2.31%)		(1.93%)			
Regular Source of Healthcare (hp2cat)						
	1,407.00		1,266.00			
	(84.10%)		(84.40%)			
	47,714,928.87		67,743,353.45			
yes^{R}	(85.11%)		(86.37%)		0.053	0.07
	266.00		234.00		*0.817*	*0.797*
	(15.90%)		(15.60%)			
	8,349,531.79		10,694,219.32			
no	(14.89%)		(13.63%)			
Source of Healthcare (hcsre)						
	908.00		899.00			
	(54.27%)		(59.93%)			
	33,642,652.17		51,519,187.82			
healthcare provider^{R}	(60.01%)		(65.58%)			
	399.00		277.00			
	(23.85%)		(18.47%)			
	11,321,776.58		12,446,320.30		15.44	0.82
clinic	(20.19%)		(15.87%)		*0.0015*	*0.489*
	339.00		303.00			
	(20.26%)		(20.20%)			
	9,623,922.51		13,338,734.24			
ER or none	(17.17%)		(17.01%)			
	27.00		21.00			
	(1.61%)		(1.40%)			
	1,476,109.39		1,133,330.41			
missing	(2.63%)		(1.44%)			

Table 42
Bivariate Analyses of Independent Variables on Exposure as Outcome with Two Categories Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables with Exposure Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30	0 and 1		2 and 3		χ^2 *p value* *unweighted*	χ^2 *p value* *weighted*
Nutritional Status						
Food Security (food2cat)						
	1,333.00		1,239.00			
	(79.68%)		(82.60%)			
	46,653,491.95		67,390,420.71			
food secure^{R}	(83.21%)		(85.92%)			
	271.00		188.00		9.15	4.49
	(16.20%)		(12.53%)		*0.010*	*0.017*
	7,205,478.55		7,029,043.50			
food insecure	(12.85%)		(8.96%)			
	69.00		73.00			
	(4.12%)		(4.87%)			
	2,205,490.16		4,018,108.56			
missing	(3.93%)		(5.12%)			
Body Mass Index (bmi30cat)						
<30.0^{R}	1,217.00		1,140.00			
underweight	(72.74%)		(76.00%)			
normal	41,844,407.61		60,999,489.31			
overweight	(74.64%)		(77.77%)			
	430.00		347.00		6.30	0.69
	(25.70%)		(23.13%)		*0.430*	*0.507*
30.0+	13,438,670.22		16,777,787.58			
obese	(23.97%)		(21.39%)			
	26.00		13.00			
	(1.55%)		(0.87%)			
	781,382.83		660,295.88			
missing	(1.39%)		(0.84%)			
Fat Intake/AMDR (fat3cat)						
	1,020.00		878.00			
	(60.97%)		(58.53%)			
	33,215,112.27		47,929,229.77			
recommended or less^{R}	(59.24%)		(61.10%)		1.95	0.16
	653.00		622.00		*0.1625*	*0.690*
	(39.03%)		(41.47%)			
	22,849,348.38		30,508,343.01			
more than recommended	(40.76%)		(38.90%)			
Protein Intake/AMDR (prot3cat)						
	1,440.00		1,272.00			
	(86.07%)		(84.80%)			
	49,359,245.64		69,404,519.64			
recommended or more^{R}	(88.04%)		(88.48%)		1.03	0.03
	233.00		228.00		*0.309*	*0.857*
	(13.93%)		(15.20%)			
	6,705,215.02		9,033,053.14			
less than recommended	(11.96%)		(11.52%)			
Iron Intake/RDA (iron2cat)						
	507.00		422.00			
	(30.30%)		(28.13%)			
	14,985,849.52		18,523,830.10			
recommended or more^{R}	(26.73%)		(23.62%)		1.80	0.56
	1,166.00		1,078.00		*0.1796*	*0.460*
	(69.70%)		(71.87%)			
	41,078,611.14		59,913,742.68			
less than recommended	(73.27%)		(76.38%)			
Calcium Intake/RDA (calc2cat)						
	449.00		396.00			
	(26.84%)		(26.40%)			
	15,850,605.09		26,711,938.83			
recommended or more^{R}	(28.27%)		(34.06%)		0.08	0.94
	1,224.00		1,104.00		*0.780*	*0.337*
	(73.16%)		(73.60%)			
	40,213,855.57		51,725,633.94			
less than recommended	(71.73%)		(65.94%)			

Table 42
Bivariate Analyses of Independent Variables on Exposure as Outcome with Two Categories Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables with Exposure Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30	0 and 1	2 and 3	χ^2 *p value* *unweighted*	χ^2 *p value* *weighted*
Selenium Intake/RDA (sele2cat)				
recommended or more^{R}	1,326.00 (79.26%) 44,641,673.61 (79.63%)	1,233.00 (82.20%) 68,036,809.25 (86.74%)	4.38 *0.036*	4.61 *0.040*
less than recommended	347.00 (20.74%) 11,422,787.05 (20.37%)	267.00 (17.80%) 10,400,763.53 (13.26%)		
Reproductive Status				
Current Pregnancy (pregnant)				
pregnant	255.00 (15.24%) 2,806,683.92 (5.01%)	136.00 (9.07%) 2,035,505.17 (2.60%)		
not pregnant^{R}	1,351.00 (80.5%) 51,426,908.09 (91.73%)	1,290.00 (86.00%) 74,949,610.84 (95.55%)	28.63 *<0.0001*	4.09 *0.023*
missing	67.00 (4.00%) 1,830,868.64 (3.27%)	74.00 (4.93%) 1,452,456.76 (1.85%)		
Trimester of Pregnancy (tripcorr)				
not pregnant^{R}	1,418.00 (84.76%) 53,257,776.74 (94.99%)	1,364.00 (90.93%) 76,402,067.61 (97.40%)		
1st trimester	77.00 4.60%) 1,114,818.61 (1.99%)	72.00 (4.80%) 876,747.50 (1.12%)	46.19 *<0.0001*	2.35 *0.085*
2nd trimester	100.00 (5.98%) 973,564.59 (1.74%)	32.00 (2.13%) 549,930.94 (0.70%)		
3rd trimester	78.00 (4.66%) 718,300.72 (1.28%)	32.00 (2.13%) 608,826.72 (0.78%)		
Ever Pregnant (tprg2cat)				
never pregnant^{R}	936.00 (55.95%) 30,643,248.04 (54.66%)	599.00 (39.93%) 28,921,848.94 (36.87%)	81.21 *<0.0001*	13.36 *0.0007*
one or more pregnancies	737.00 (44.05%) 25,421,212.62 (45.34%)	901.00 (60.07%) 49,515,723.83 (63.13%)		
Live Births (live)				
no live births^{R}	1,095.00 (65.45%) 34,042,014.38 (60.72%)	725.00 (48.33%) 33,378,223.83 (42.55%)	94.75 *<0.0001*	15.34 *0.0003*
one or more live births	578.00 (34.55%) 22,022,446.27 (39.28%)	775.00 (51.67%) 45,059,348.94 (57.45%)		

Table 42
Bivariate Analyses of Independent Variables on Exposure as Outcome with Two Categories Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables with Exposure Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30	0 and 1		2 and 3		χ^2 *p value unweighted*	χ^2 *p value weighted*
Ever Breastfed (brstfda)						
never breastfed^{R}	1,276.00 (76.27%) 40,009,667.89 (71.36%)		1,047.00 (69.80%) 51,944,654.41 (66.22%)		16.88 *<0.0001*	2.02 *0.162*
breastfed more than one month and/or currently	397.00 (23.73%) 16,054,792.77 (28.54%)		453.00 (30.20%) 26,492,918.36 (33.78%)			
Exposure-Related Attributes						
Acculturation						
Birthplace (born2cat)						
U.S.^{R}	1,421.00 (84.94%) 49,686,661.17 (88.62%)		1,252.00 (83.47%) 70,617,035.63 (90.03%)		1.28 *0.256*	0.37 *0.545*
outside U.S.	252.00 (15.06%) 6,377,799.49 (11.38%)		248.00 (16.53%) 7,820,537.14 (9.97%)			
Years in U.S. (yrus5)						
born in U.S.^{R}	1,421.00 (85.04%) 49,686,661.17 (88.67%)		1,252.00 (83.69%) 70,617,035.63 (90.19%)			
five or more years	170.00 (10.17%) 4,699,016.12 (8.39%)		182.00 (12.17%) 6,374,503.09 (8.14%)		3.72 *0.156*	1.26 *0.292*
less than five years	80.00 (4.79%) 1,652,073.94 (2.95%)		62.00 (4.14%) 1,303,892.97 (1.67%)			
Language Spoken at Home (lang2cat)						
English^{R}	1,510.00 (90.26%) 52,407,413.27 (93.48%)		1,334.00 (89.05%) 74,353,781.09 (95.07%)		1.24 *0.265*	1.57 *0.217*
Other	163.00 (9.74%) 3,657,047.38 (6.52%)		164.00 (10.95%) 3,856,724.33 (4.93%)			
U.S. Citizenship (usczn2cat)						
U.S. citizen^{R}	1,490.00 (89.06%) 52,431,440.49 (93.52%)		1,324.00 (88.33%) 74,393,831.42 (94.87%)		0.43 *0.513*	0.99 *0.324*
non-U.S. citizen	183.00 (10.94%) 3,633,020.17 (6.48%)		175.00 (11.67%) 4,021,872.67 (5.13%)			
Diet						
Seafood Eaten in Past 30 Days (smpw2cat)						
none^{R}	483.00 (28.87%) 14,572,597.13 (25.99%)		203.00 (13.53%) 8,298,243.66 (10.58%)		109.78 *<0.0001*	22.36 *0.0000*
any	1,190.00 (71.13%) 41,491,863.53 (74.01%)		1,297.00 (86.47%) 70,139,329.12 (89.42%)			

Table 42

Bivariate Analyses of Independent Variables on Exposure as Outcome with Two Categories Among Childbearing-Aged Females

(unweighted and weighted data 1999-2004)

Independent Variables with Exposure Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30	0 and 1		2 and 3		χ^2 *p value unweighted*	χ^2 *p value weighted*
Fish Eaten in Past 30 Days (fish2cat)						
none[R]	715.00 (42.74%) 22,681,779.88 (40.46%)		325.00 (21.67%) 14,127,959.80 (18.01%)		159.37 *<0.0001*	40.16 *0.0000*
any	958.00 (57.26%) 33,382,680.78 (59.54%)		1,175.00 (78.33%) 6,4309,612.98 (81.99%)			
Shellfish Eaten in Past 30 Days (shell2cat)						
none[R]	941.00 (56.25%) 30,638,660.38 (54.65%)		616.00 (41.07%) 32,379,978.80 (41.28%)		72.92 *<0.0001*	7.60 *0.0085*
any	732.00 (43.75%) 25,425,800.28 (45.35%)		884.00 (58.93%) 46,057,593.97 (58.72%)			
Tap Water Consumed Prior 24h (tap2kct)						
none[R]	654.00 (39.09%) 19,341,590.85 (34.50%)		475.00 (31.67%) 21,163,237.39 (26.98%)			
< 2,000 ml	775.00 (46.32%) 27,284,851.16 (48.67%)		763.00 (50.87%) 43,760,634.26 (55.79%)		33.78 *<0.0001*	2.03 *0.122*
2,000+ ml	163.00 (9.74%) 6,637,991.60 (11.84%)		132.00 (8.80%) 8,891,560.90 (11.34%)			
missing=1	81.00 (4.84%) 2,800,027.04 (4.99%)		130.00 (8.67%) 4,622,140.22 (5.89%)			
Alcohol Consumption						
Alcohol Consumption (retohuse)						
never, seldom drinker[R] *including 16-19 y/o*	1,076.00 (64.32%) 26,791,446.13 (47.79%)		667.00 (44.47%) 25,429,069.23 (32.42%)			
drinker	349.00 (20.86%) 16,087,057.48 (28.69%)		381.00 (25.40%) 24,583,022.09 (31.34%)		148.84 *<0.0001*	6.50 *0.0010*
heavy drinker	202.00 (12.07%) 11,799,383.44 (21.05%)		353.00 (23.53%) 23,965,996.00 (30.55%)			
missing	46.00 (2.75%) 1,386,573.60 (2.47%)		99.00 (6.60%) 4,459,485.45 (5.69%)			
Tobacco Use						
Serum Cotinine (cot3cat)						
< 1.0 ng/ml[R]	1,310.00 (78.77%) 42,870,593.92 (76.89%)		1,058.00 (70.77%) 56,000,879.65 (71.69%)			
1.0 - 10.0 ng/ml	103.00 (6.19%) 2,911,068.62 (5.22%)		87.00 (5.82%) 2,339,233.00 (2.99%)		35.99 <0.0001	4.30 *0.0197*
> 10.0 ng/ml	250.00 (15.03%) 9,977,616.00 (17.89%)		350.00 (23.41%) 19,772,725.24 (25.31%)			

Table 42
Bivariate Analyses of Independent Variables on Exposure as Outcome with Two Categories Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables with Exposure Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30	0 and 1		2 and 3		χ^2 *p value* *unweighted*	χ^2 *p value* *weighted*
ETS (ETS)						
no ETS^{R}	1,273.00 (76.09%) 42,613,280.22 (76.01%)		1,144.00 (76.27%) 59,184,091.36 (75.45%)			
ETS at home or work	347.00 (20.74%) 11,530,060.37 (20.57%)		303.00 (20.20%) 15,176,809.23 (19.35%)		0.432 *0.805*	0.41 *0.666*
ETS at home and work	53.00 (3.17%) 1,921,120.07 (3.43%)		53.00 (3.53%) 4,076,672.18 (5.20%)			
Residence						
Tap Water Source (h2os2cat)						
public^{R}	1,470.00 (87.87%) 48,669,218.11 (86.81%)		1,356.00 (90.40%) 68,066,690.05 (86.78%)			
private	160.00 (9.56%) 6,135,157.79 (10.94%)		106.00 (7.07%) 8,356,377.23 (10.65%)		6.46 *0.039*	0.03 *0.968*
missing	43.00 (2.57%) 1,260,084.76 (2.25%)		38.00 (2.53%) 2,014,505.49 (2.57%)			
Residential Tap Water Treatment (h2ox2cat)						
yes	470.00 (28.09%) 18,237,088.22 (32.58%)		393.00 (26.20%) 27,071,146.45 (34.51%)			
no^{R}	1,167.00 (69.75%) 36,752,210.02 (65.55%)		1,072.00 (71.47%) 49,793,127.34 (63.48%)		1.49 *0.475*	0.08 *0.925*
missing	36.00 (2.15%) 1,075,162.42 (1.92%)		35.00 (2.33%) 1,573,298.98 (2.01%)			
Type of Residence (res3cat)						
attached or detached house^{R}	1,066.00 (63.72%) 35,113,061.69 (62.63%)		1,006.00 (67.07%) 54,192,908.87 (69.09%)			
mobile home or trailer	107.00 (6.40%) 3,865,488.99 (6.89%)		95.00 (6.33%) 4,536,288.81 (5.78%)		4.38 *0.112*	1.29 *0.286*
all other types *including missing/unknown*	500.00 (29.89%) 17,085,909.98 (30.48%)		399.00 (26.60%) 19,708,375.09 (25.13%)			

Table 42
Bivariate Analyses of Independent Variables on Exposure as Outcome with Two Categories Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables with Exposure Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30	0 and 1	2 and 3	χ^2 *p value unweighted*	χ^2 *p value weighted*
Age of Residence (resb60cat)				
1960 or newer[R]	841.00 (50.27%) 32,355,341.90 (57.71%)	754.00 (50.27%) 45,689,182.12 (58.25%)		
older than 1960	404.00 (24.15%) 12,895,677.19 (23.00%)	362.00 (24.16%) 19,196,522.91 (24.47%)	0.00 *0.999*	0.20 *0.815*
missing/unknown	428.00 (25.58%) 10,813,441.57 (19.29%)	384.00 (25.60%) 13,551,867.74 (17.28%)		
Age of Residence (resb78cat)				
1978 or newer[R]	547.00 (32.70%) 21,828,679.09 (38.93%)	540.00 (36.00%) 33,559,369.76 (42.78%)		
older than 1978	698.00 (41.72%) 23,422,340.00 (41.78%)	576.00 (38.40%) 31,326,335.27 (39.94%)	4.69 *0.096*	0.36 *0.699*
missing/unknown	428.00 (25.58%) 10,813,441.57 (19.29%)	384.00 (25.60%) 13,551,867.74 (17.28%)		
Resident Status (resd3cat)				
own[R]	887.00 (53.02%) 31,914,972.43 (56.93%)	840.00 (56.00%) 45,335,334.69 (57.80%)		
rent	676.00 (40.41%) 20,737,390.37 (36.99%)	602.00 (40.13%) 30,426,505.20 (38.79%)	12.26 *0.002*	0.77 *0.466*
other *including missing*	110.00 (6.58%) 3,412,097.85 (6.09%)	58.00 (3.87%) 2,675,732.88 (3.41%)		
Years at Current Residence (re5yrcat)				
more than five years[R]	555.00 (33.17%) 18,106,390.76 (32.30%)	558.00 (37.20%) 27,787,927.92 (35.43%)		
five years or less	1,095.00 (65.45%) 37,302,721.50 (66.54%)	912.00 (60.80%) 49,152,891.44 (62.66%)	8.21 *0.016*	0.46 *0.634*
missing	23.00 (1.37%) 655,348.40 (1.17%)	30.00 (2.00%) 1,496,753.41 (1.91%)		
Household Size (hsize)				
four persons or less[R]	1,091.00 (65.21%) 41,630,345.58 (74.25%)	1,091.00 (72.73%) 64,823,682.81 (82.64%)		
more than four persons	582.00 (34.79%) 14,434,115.08 (25.75%)	409.00 (27.27%) 13,613,889.97 (17.36%)	20.83 *<0.0001*	5.43 *0.024*

Table 42
Bivariate Analyses of Independent Variables on Exposure as Outcome with Two Categories Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

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Rooms in Residence (rm3cat)						
7+ rooms^{R}	610.00 (36.46%) 22,384,013.42 (39.93%)		538.00 (35.87%) 30,232,499.39 (38.54%)			
4-6 rooms	892.00 (53.32%) 28,129,923.41 (50.17%)		799.00 (53.27%) 40,970,209.18 (52.23%)		0.89 *0.827*	0.05 *0.982*
1-3 rooms	132.00 (7.89%) 4,412,246.48 (7.87%)		131.00 (8.73%) 5,762,926.63 (7.35%)			
missing	39.00 (2.33%) 1,138,277.35 (2.03%)		32.00 (2.13%) 1,471,937.57 (1.88%)			
Occupation						
Current Occupation (cocc2cat)						
not working^{R}	757.00 (45.25%) 18,579,398.21 (33.14%)		567.00 (37.80%) 23,593,559.36 (30.08%)			
management, professional & sales	598.00 (35.74%) 25,983,638.35 (46.35%)		645.00 (43.00%) 41,775,253.39 (53.26%)		21.16 *<0.0001*	1.01 *0.374*
services & goods	318.00 (19.01%) 11,501,424.09 (20.51%)		288.00 (19.20%) 13,068,760.03 (16.66%)			
Time in Current Employment (cjt)						
not working^{R}	757.00 (45.25%) 18,579,398.21 (33.14%)		567.00 (37.80%) 23,593,559.36 (30.08%)			
less than five years	781.00 (46.68%) 30,471,853.31 (54.36%)		653.00 (43.53%) 36,769,786.41 (46.88%)		80.16 *<0.0001*	3.86 *0.028*
five or more years	135.00 (8.07%) 7,013,209.13 (12.51%)		280.00 (18.67%) 18,074,227.00 (23.04%)			
Total Hours Worked Prior Week (hrwk)						
not employed^{R}	786.00 (47.01%) 19,977,686.48 (35.71%)		595.00 (39.69%) 25,830,351.54 (32.94%)			
less than 35 hours	418.00 (25.00%) 13,916,897.10 (24.88%)		318.00 (21.21%) 19,450,536.69 (24.80%)		43.91 *<0.0001*	0.15 *0.859*
35+ hours	468.00 (27.99%) 22,046,705.15 (39.41%)		586.00 (39.09%) 33,134,815.85 (42.26%)			

Table 42
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(unweighted and weighted data 1999-2004)

Independent Variables with Exposure Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30	0 and 1		2 and 3		χ^2 *p value* *unweighted*	χ^2 *p value* *weighted*
Longest Held Occupation (locc2cat)						
not applicable^{R}	836.00 (49.97%) 28,223,195.93 (50.34%)		726.00 (48.40%) 35,894,160.58 (45.76%)			
management, professional & sales	436.00 (26.06%) 15,629,691.43 (27.88%)		467.00 (31.13%) 25,785,494.72 (32.87%)		11.89 *0.0026*	0.79 *0.460*
services & goods	401.00 (23.97%) 12,211,573.30 (21.78%)		307.00 (20.47%) 16,757,917.47 (21.36%)			
Time in Longest Employment (ljt)						
not applicable^{R}	836.00 (49.97%) 28,223,195.93 (50.34%)		726.00 (48.40%) 35,894,160.58 (45.76%)			
less than five years	629.00 (37.60%) 19,448,228.46 (34.69%)		368.00 (24.53%) 15,094,040.39 (19.24%)		130.88 *<0.0001*	5.74 0.0061
five or more years	208.00 (12.43%) 8,393,036.27 (14.97%)		406.00 (27.07%) 27,449,371.80 (35.00%)			
Socioeconomic Factors						
Education						
Highest Education (educ2)						
high school diploma, GED or higher^{R}	954.00 (57.02%) 41,908,990.51 (74.75%)		1,083.00 (72.25%) 64,998,170.83 (82.94%)			
less than high school diploma	719.00 (42.98%) 14,155,470.15 (25.25%)		416.00 (27.75%) 13,372,263.15 (17.06%)		79.75 *<0.0001*	5.20 *0.027*
Employment						
Employment Status (emp3cat)						
employed	917.00 (54.88%) 37,506,551.23 (66.91%)		936.00 (62.40%) 54,962,248.73 (70.07%)			
not employed^{R}	754.00 (45.12%) 18,546,689.91 (33.09%)		564.00 (37.60%) 23,475,324.04 (29.93%)		18.42 *<0.0001*	0.25 *0.619*
Reason for Unemployment (unem2cat)						
working^{R}	917.00 (54.81%) 37,506,551.23 (66.90%)		936.00 (62.40%) 54,962,248.73 (70.07%)			
voluntary unemployment	533.00 (31.86%) 12,975,338.65 (23.14%)		391.00 (26.07%) 15,289,683.02 (19.49%)			
involuntary unemployment	144.00 (8.61%) 3,625,315.15 (6.47%)		151.00 (10.07%) 6,637,798.86 (8.46%)		45.05 *<0.0001*	0.77 *0.517*
missing	79.00 (4.72%) 1,957,255.62 (3.49%)		22.00 (1.47%) 1,547,842.16 (1.97%)			

Table 42

Bivariate Analyses of Independent Variables on Exposure as Outcome with Two Categories Among Childbearing-Aged Females

(unweighted and weighted data 1999-2004)

Independent Variables with Exposure Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30	0 and 1		2 and 3		χ^2 *p value* *unweighted*	χ^2 *p value* *weighted*
Work History (wkcp)						
never employed^{R}	259.00 (15.48%) 4,501,108.85 (8.03%)		149.00 (9.93%) 3,737,701.95 (4.77%)			
currently employed	577.00 (34.49%) 23,722,087.08 (42.31%)		577.00 (38.47%) 32,156,458.63 (41.00%)		27.71 <0.0001	1.49 *0.229*
employed in the past but not currently	498.00 (29.77%) 14,078,289.37 (25.11%)		418.00 (27.87%) 19,855,857.41 (25.31%)			
employed now and in the past	339.00 (20.6%) 13,762,975.36 (24.55%)		356.00 (23.73%) 22,687,554.78 (28.92%)			
Income						
U.S. Poverty Threshold (pov2cat)						
more than 1.00^{R}	1,159.00 (69.28%) 42,663,631.82 (76.10%)		1,068.00 (71.20%) 61,289,991.37 (78.14%)			
1.00 or less	416.00 (24.87%) 9,918,920.81 (17.69%)		314.00 (20.93%) 12,668,276.66 (16.15%)		10.42 *0.005*	0.085 *0.919*
missing	98.00 (5.86%) 3,481,908.03 (6.21%)		118.00 (7.87%) 4,479,304.74 (5.71%)			
Marital Status						
Marital Status (marr3cat)						
married or living with partner	546.00 (32.64%) 22,312,508.65 (39.80%)		652.00 (43.47%) 39,488,139.60 (50.34%)			
widowed, divorced or separated	81.00 (4.84%) 3,129,387.79 (5.58%)		180.00 (12.00%) 11,224,564.59 (14.31%)		158.07 *<0.0001*	11.95 *0.0000*
never married^{R}	1,025.00 (61.27%) 29,803,777.82 (53.16%)		612.00 (40.80%) 23,689,173.67 (30.20%)			
missing	21.00 (1.26%) 818,786.41 (1.46%)		56.00 (3.73%) 4,035,694.90 (5.15%)			

Table 42
Bivariate Analyses of Independent Variables on Exposure as Outcome with Two Categories Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables with Exposure Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30	0 and 1		2 and 3		χ^2 *p value* *unweighted*	χ^2 *p value* *weighted*
Race-Ethnicity						
Race-Ethnicity (race5cat)						
Non-Hispanic White^{R}	762.00 (45.55%) 39,164,389.13 (69.86%)		731.00 (48.73%) 58,723,155.03 (74.87%)			
Non-Hispanic Black	295.00 (17.63%) 4,574,953.56 (8.16%)		328.00 (21.87%) 8,172,224.81 (10.42%)			
Mexican American	440.00 (26.30%) 5,007,799.19 (8.93%)		305.00 (20.33%) 3,662,776.62 (4.67%)		23.29 *0.0001*	2.67 *0.044*
Other Hispanic	104.00 (6.22%) 3,953,960.50 (7.05%)		74.00 (4.93%) 3,572,031.72 (4.55%)			
Asian, Native American, Pacific Islander & Multi-Racial	72.00 (4.30%) 3,363,358.28 (6.00%)		62.00 (4.13%) 4,307,384.60 (5.49%)			
Race-Ethnicity/Hispanic Grouping (race4cat)						
Non-Hispanic White^{R}	762.00 (45.55%) 39,164,389.13 (69.86%)		731.00 (48.73%) 58,723,155.03 (74.87%)			
Non-Hispanic Black	295.00 (17.63%) 4,574,953.56 (8.16%)		328.00 (21.87%) 8,172,224.81 (10.42%)		23.27 *<0.0001*	3.28 *0.029*
Hispanic	544.00 (32.52%) 8,961,759.69 (15.98%)		379.00 (25.27%) 7,234,808.34 (9.22%)			
Asian, Native American, Pacific Islander & Multi-Racial	72.00 (4.30%) 3,363,358.28 (6.00%)		62.00 (4.13%) 4,307,384.60 (5.49%)			

Table 43

Summary of Chi-Square (χ^2) and p Values of Weighted Independent Variables on Exposure as Outcome with Two Categories (1999-2004)

Variables $p < 0.20$	p value	χ^2 *weighted*	Variables $p > 0.20$	p value	χ^2 *weighted*
Fish in Past 30 Days (fish2cat)	*0.000*	40.16	Language Spoken at Home (lang2cat)	*0.217*	1.57
Seafood in Past 30 Days [1] (smpw2cat)	*0.000*	22.36	Work History (wkcp)	*0.229*	1.49
Age (age4cat)	*0.000*	18.38	Type of Residence (res3cat)	*0.286*	1.29
Marital Status (marr3cat)	*0.000*	11.95	Years in U.S. (yrus5)	*0.292*	1.26
Live Births (live)	*0.000*	15.34	U.S. Citizenship (usczn2cat)	*0.324*	0.99
Ever Pregnant[1] (tprg2cat)	*0.001*	13.36	Calcium Intake/RDA (calc2cat)	*0.337*	0.94
Alcohol Consumption (retohuse)	*0.001*	6.50	Charleson Co-Morbidity Index (CCMS3cat)	*0.347*	1.08
Time in Longest Employment (ljt)	*0.006*	5.74	Current Occupation (cocc2cat)	*0.374*	1.01
Shellfish in Past 30 Days (shell2cat)	*0.009*	7.60	Longest Held Occupation (locc2cat)	*0.460*	0.79
Food Security (food2cat)	*0.017*	4.49	Iron Intake/RDA (iron2cat)	*0.460*	0.56
Serum Cotinine (cot3cat)	*0.019*	4.30	Resident Status (resd3cat)	*0.466*	0.77
Current Pregnancy (pregnant)	*0.023*	4.09	Source of Healthcare (hcsre)	*0.489*	0.82
Household Size (hsize)	*0.024*	5.43	Body Mass Index (bmi30cat)	*0.507*	0.69
Highest Education (educ2)	*0.027*	5.20	Reason for Unemployment (unem2cat)	*0.517*	0.77
Time in Current Employment (cjt)	*0.028*	3.86	Birthplace (born2cat)	*0.545*	0.37
Race-Ethnicity/Hispanic Grouping (race4cat)	*0.029*	3.28	Employment Status (emp3cat)	*0.619*	0.25
Selenium Intake/RDA (sele2cat)	*0.040*	4.61	Years at Current Residence (re5yrcat)	*0.634*	0.46
Race-Ethnicity[1] (race5cat)	*0.044*	2.67	Environmental Tobacco Smoke (ETS)	*0.666*	0.41
Trimester of Pregnancy[1] (tripcorr)	*0.085*	2.35	Perceived Health Status (huq2cat)	*0.696*	0.15
Tap Water Consumed 24h (tap2kct)	*0.122*	2.03	Age of Residence (resb78cat)	*0.699*	0.36
Ever Breastfed (brstfda)	*0.162*	2.02	Fat Intake/AMDR (fat3cat)	*0.732*	0.12
			Iron Deficiency (FeD2cat)	*0.791*	0.07
			Regular Source of Healthcare (hp2cat)	*0.797*	0.07
			Age of Residence (resb60cat)	*0.815*	0.20
			Protein Intake/AMDR (prot3cat)	*0.857*	0.03
			Total Hours Worked Prior Week (hrwk)	*0.859*	0.15
			Health Insurance (hi2cat)	*0.901*	0.19
			U.S. Poverty Threshold (pov2cat)	*0.919*	0.08
			Residential Tap Water Treatment (h2ox2cat)	*0.925*	0.08
			Treatment for Iron Deficiency past 3 mo (FeTx2cat)	*0.942*	0.00
			Tap Water Source (h2os2cat)	*0.968*	0.03
			Rooms in Residence (rm3cat)	*0.982*	0.05
			Iron Deficiency and Treatment (FeDTx)	*0.983*	0.54

[1]variable dropped due to low cell size or too similar to other variables

Table 44
Stepwise Logistic Regression Analyses of Exposure as Outcome with Two Categories
(1999 - 2004)

Variable Name *p < 0.20*	df	-2LL Wald F	Difference	df	*p* value
Initial Regression	32	1,018.12			
1 Time in Current Employment (cjt)	30	1,014.32	3.80	2	*>0.10* *drop*
2 Tap Water Consumed 24h (tap2kct)	27	1,009.88	1.63	3	*>0.20* *drop*
3 Live Births (live)	26	1,008.06	1.82	1	*>0.10* *drop*
4 Race-Ethnicity/Hispanic Grouping (race4cat)	23	992.69	15.37	3	*<0.01* *keep*
5 Current Pregnancy (pregnant)	24	1,002.16	5.90	2	*>0.05* *drop*
6 Serum Cotinine (cot3cat)	22	993.50	8.66	2	*<0.02* *keep*
7 Household Size (hsize)	23	993.78	8.38	1	*<0.01* *keep*
8 Time in Longest Employment (ljt)	22	974.08	28.08	2	*<0.001* *keep*
9 Highest Education (educ2)	23	973.50	28.66	1	*<0.001* *keep*
10 Marital Status (marr3cat)	21	960.42	41.74	3	*<0.001* *keep*
11 Age (age4cat)	21	686.92	315.24	3	*<0.001* *keep*
12 Food Security (food2cat)	22	955.84	46.32	2	*<0.001* *keep*
13 Selenium Intake/RDA (sele2cat)	23	990.31	11.85	1	*<0.001* *keep*
14 Ever Breastfed (brstfda)	23	977.17	24.99	1	*<0.001* *keep*
15 Fish in Past 30 Days (fish2cat)	23	877.76	124.40	1	*<0.001* *keep*
16 Shellfish in Past 30 Days (shell2cat)	23	980.03	22.13	1	*<0.001* *keep*
17 Alcohol Consumption (retohuse)	21	970.95	31.21	3	*<0.001* *keep*

Table 45
Best-Fit Logistic Regression Exposure Model *with no interactions*
(1999 - 2004)

Variable Name *in ascending order by p value*	df	-2LL Wald F	R^2		*p* value
Best Fit Regression	24	1,002.16	*0.2719*		
Fish in Past 30 Days (fish2cat)	1	26.26			*0.0000*
Age (age4cat)	3	11.92			*0.0000*
Food Security (food2cat)	2	5.94			*0.0052*
Ever Breastfed (brstfda)	1	5.32			*0.0258*
Highest Education (educ2)	1	3.81			*0.0572*
Shellfish in Past 30 Days (shell2cat)	1	3.73			*0.0598*
Marital Status (marr3cat)	3	2.13			*0.1106*
Selenium Intake/RDA (sele2cat)	1	2.44			*0.1255*
Time in Longest Employment (ljt)	2	1.68			*0.1976*
Alcohol Consumption (retohuse)	3	1.60			*0.2020*
Household Size (hsize)	1	1.55			*0.2193*
Serum Cotinine (cot3cat)	2	1.37			*0.2641*
Race-Ethnicity/Hispanic Grouping (race4cat)	3	0.96			*0.4210*

Table 46
Variance Inflation Factor Test for Collinearity Among Independent Variables
using the Best-Fit Logistic Regression Exposure Model (1999-2004)

	df	sum of squares	mean square	F value	pr>F	
Best-Fit Exposure Model *with no interactions*	13	258.99	19.930	113.13	*<0.001*	

Variable Name	df	parameter estimate	std error	t value	pr>t	VIF
Intercept	1	-0.499	0.044	-11.26	*<0.0001*	0.0000
Age (age4cat)	1	0.121	0.005	23.01	*<0.0001*	1.9536
Food Security (food2cat)	1	-0.00009	0.008	-0.01	*0.9907*	1.0376
Selenium Intake/RDA (sele2cat)	1	-0.002	0.011	-0.20	*0.8433*	1.0318
Ever Breastfed (brstfda)	1	-0.032	0.011	-2.94	*0.0033*	1.3960
Fish in Past 30 Days (fish2cat)	1	0.124	0.009	13.22	*<0.0001*	1.1280
Shellfish in Past 30 Days (shell2cat)	1	0.092	0.009	10.46	*<0.0001*	1.1003
Alcohol Consumption (retohuse)	1	0.010	0.006	1.74	*0.0813*	1.2960
Serum Cotinine (cot3cat)	1	0.034	0.005	6.31	*<0.0001*	1.0774
Household Size (hsize)	1	0.001	0.010	0.10	*0.9241*	1.4153
Time in Longest Employment (ljt)	1	0.006	0.006	1.04	*0.2965*	1.0729
Highest Education (educ2)	1	0.008	0.010	0.76	*0.4459*	1.3966
Marital Status (marr3cat)	1	0.025	0.006	4.46	*<0.0001*	1.7699
Race-Ethnicity/Hispanic Grouping (race4cat)	1	0.021	0.005	4.37	*<0.0001*	1.1474

Table 47
Statistical Significance of Interactions Between Independent Variables using the Best-Fit Logistic Regression Exposure Model (1999-2004)

Independent Variables	Age (age4cat)	Food Security (food2cat)	Selenium Intake/RDA (sele2cat)	Ever Breastfed (brstfda)	Fish in Past 30 Days (fish2cat)	Shellfish in Past 30 Days (shell2cat)	Alcohol Consumption (retohuse)	Serum Cotinine (cot3cat)	Household Size (hsize)	Time in Longest Employment (ljt)	Highest Education (educ2)	Marital Status (marr3cat)	Race-Ethnicity Hispanic Grouping (race4cat)
Age (age4cat)													
Food Security (food2cat)	*op*												
Selenium Intake/RDA (sele2cat)	*<0.001*	*ns*											
Ever Breastfed (brstfda)	*ns*	*<0.01*	*<0.01*										
Fish in Past 30 Days (fish2cat)	*<0.001*	*<0.05*	*ns*	*<0.01*									
Shellfish in Past 30 Days (shell2cat)	*<0.001*	*<0.001*	*ns*	*ns*	*ns*								
Alcohol Consumption (retohuse)	*op*	*<0.001*	*<0.05*	*op*	*<0.01*	*<0.001*							
Serum Cotinine (cot3cat)	*<0.001*	*ns*	*<0.001*	*<0.02*	*<0.001*	*<0.05*	*<0.001*						
Household Size (hsize)	*ns*	*<0.01*	*ns*	*<0.001*	*ns*	*<0.001*	*<0.001*	*<0.001*					
Time in Longest Employment (ljt)	*<0.001*	*<0.01*	*ns*	*<0.001*	*<0.001*	*ns*	*<0.001*	*<0.01*	*<0.001*				
Highest Education (educ2)	*<0.001*	*<0.05*	*ns*	*<0.02*	*ns*	*ns*	*<0.01*	*ns*	*<0.05*	*ns*			
Marital Status (marr3cat)	*op*	*op*	*ns*	*<0.001*	*<0.001*	*<0.001*	*<0.001*	*op*	*ns*	*<0.001*	*<0.01*		
Race-Ethnicity/Hispanic Grouping (race4cat)	*<0.001*	*op*	*<0.05*	*<0.01*	*<0.001*	*<0.01*	*op*	*op*	*ns*	*<0.001*	*<0.01*	*op*	

op = overparameterized unable to calculate
ns = not statistically significant p>0.05

Table 48
Odds Ratios and Confidence Intervals for Best-Fit Logistic Regression Exposure Model *with no interactions* (1999-2004)

Variable Names	df	-2LL Wald F	*p* value	Odds Ratios	Confidence Intervals
Susceptibility-Related Attributes					
Age (age4cat)	3	11.92	*0.0000*		
16-19[R]				1.00	*ns*
20-29				3.50	1.56 - 7.85
30-39				8.48	3.16 - 22.74
40-49				30.20	8.36 - 109.15
Nutritional Status					
Food Security (food2cat)	2	5.94	*0.0052*		
food secure[R]				1.00	*ns*
food insecure				0.61	0.35 - 1.06
missing				2.38	1.18 - 4.82
Selenium Intake/RDA (sele2cat)	1	2.44	*0.1255*		
recommended or more[R]				1.00	*ns*
less than recommended				0.66	0.39 - 1.13
Reproductive Status					
Ever Breastfed (brstfda)	1	5.32	*0.0258*		
never breastfed[R]				1.00	*ns*
breastfed more than one month or currently				0.56	0.34 - 0.93
Exposure-Related Attributes					
Diet					
Fish Eaten in Past 30 Days (fish2cat)	1	26.26	*0.0000*		
none[R]				1.00	*ns*
any				3.11	1.99 - 4.86
Shellfish Eaten in Past 30 Days (shell2cat)	1	3.73	*0.0598*		
none[R]				1.00	*ns*
any				1.53	0.98 - 2.38
Alcohol Consumption					
Alcohol Consumption (retohuse)	3	1.60	*0.2020*		
never, seldom drinker[R]				1.00	*ns*
drinker				0.66	0.37 - 1.17
heavy drinker				1.20	0.70 - 2.07
missing				1.30	0.57 - 2.99
Tobacco Use					
Serum Cotinine (cot3cat)	2	1.37	*0.2641*		
< 1.0 ng/ml[R]				1.00	*ns*
1.0 - 10.0 ng/ml				0.73	0.26 - 2.09
> 10.0 ng/ml				1.38	0.88 - 2.16
Residence					
Household Size (hsize)	1	1.55	*0.2193*		
four persons or less[R]				1.00	*ns*
more than four persons				0.71	0.41 - 1.23
Occupation					
Time in Longest Employment (ljt)	2	1.68	*0.1976*		
not applicable[R]				1.00	*ns*
less than five years				0.87	0.53 - 1.42
five or more years				1.68	0.90 - 3.13

[R] = referent group
ns = not significant

Table 48
Odds Ratios and Confidence Intervals for Best-Fit Logistic Regression Exposure Model *with no interactions*
(1999 - 2004)

Variable Names	df	-2LL Wald F	*p* value	Odds Ratios	Confidence Intervals
Socioeconomic Factors					
Education					
Highest Education (educ2)	1	3.81	*0.0572*		
high school diploma, GED or higher[R]				1.00	*ns*
less than high school diploma				1.96	0.98 - 3.93
Marital Status					
Marital Status (marr3cat)	3	2.13	*0.1106*		
married or living with partner				0.93	0.57 - 1.53
widowed, divorced or separated				1.17	0.58 - 2.34
never married[R]				1.00	*ns*
missing				5.11	1.18 - 22.26
Race-Ethnicity					
Race-Ethnicity/Hispanic Grouping (race4cat)	3	0.96	*0.4210*		
Non-Hispanic White[R]				1.00	*ns*
Non-Hispanic Black				1.08	0.56 - 2.11
Hispanic				0.67	0.39 - 1.15
Asian, Native American, Pacific Islander & Multi-Racial				0.59	0.15 - 2.32

[R] = referent group
ns = not significant

Table 49
Bivariate Analyses of Independent Variables on Lead Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30	Below Geometric Mean		At or Above Geometric Mean		χ^2 *p value unweighted*	χ^2 *p value weighted*
Susceptibility-Related Attributes						
Age (age4cat)						
16-19[R]	654.00 (39.66%) 12,999,734.19 (18.97%)		431.00 (28.28%) 5,510,734.53 (8.35%)			
20-29	558.00 (33.84%) 29,716,727.03 (43.36%)		326.00 (21.39%) 15,630,787.88 (23.69%)		229.84 *<0.0001*	9.12 *0.0001*
30-39	307.00 (18.62%) 16,135,631.99 (23.55%)		395.00 (25.92%) 20,222,204.52 (30.65%)			
40-49	130.00 (7.88%) 9,676,562.19 (14.12%)		372.00 (24.41%) 24,609,651.11 (37.30%)			
Health Status						
Perceived Health Status (huq2cat)						
excellent, very good, good[R]	1,537.00 (93.21%) 65,500,225.53 (95.58%)		1,303.00 (85.55%) 58,505,019.56 (88.72%)		49.49 *<0.0001*	15.58 *0.0003*
fair, poor	112.00 (6.79%) 3,028,429.87 (4.42%)		220.00 (14.45%) 7,437,449.73 (11.28%)			
Charleson Co-Morbidity Scale (CCMS3cat)						
none[R]	1,466.00 (88.90%) 60,632,812.02 (88.48%)		1,348.00 (88.45%) 57,624,209.41 (87.34%)			
one co-morbidity	164.00 (9.95%) 6,883,909.15 (10.05%)		139.00 (9.12%) 6,262,824.16 (9.49%)		7.88 *0.019*	0.89 *0.415*
more than one co-morbidity	* (0.00%) 1,011,934.23 (1.48%)		37.00 (2.43%) 2,086,344.46 (3.16%)			
Iron Deficiency (FeD2cat)						
within normal limits[R]	1,444.00 (87.57%) 63,102,828.80 (92.08%)		1,280.00 (83.99%) 59,733,929.83 (90.54%)		8.35 *0.004*	0.57 *0.453*
iron deficient	205.00 (12.43%) 5,425,826.61 (7.92%)		244.00 (16.01%) 6,239,448.20 (9.46%)			
Treatment for Iron Deficiency past 3 mo (FeTx2cat)						
yes	87.00 (5.28%) 2,760,932.98 (4.03%)		84.00 (5.52%) 2,385,362.99 (3.62%)		0.09 *0.765*	0.13 *0.719*
no[R]	1,562.00 (94.72%) 65,767,722.42 (95.97%)		1,439.00 (94.48%) 63,574,435.97 (96.38%)			

Table 49
Bivariate Analyses of Independent Variables on Lead Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30	Below Geometric Mean		At or Above Geometric Mean		χ^2 *p value unweighted*	χ^2 *p value weighted*
Iron Deficiency and Treatment (FeDTx)						
normal/no treatment[R]	1,384.00 (83.93%) 61,488,181.97 (89.73%)		1,224.00 (80.37%) 57,954,516.72 (87.86%)			
normal w/treatment	60.00 (3.64%) 1,614,646.83 (2.36%)		55.00 (3.61%) 1,765,834.04 (2.68%)		8.59 *0.035*	1.11 *0.355*
deficient w/treatment	* (0.00%) 1,146,286.15 (1.67%)		* (0.00%) 619,528.95 (0.94%)			
deficient/no treatment	178.00 (10.79%) 4,279,540.45 (6.24%)		215.00 (14.12%) 5,619,919.25 (8.52%)			
Health Insurance (hi2cat)						
private[R]	1,190.00 (72.16%) 55,923,021.64 (81.61%)		852.00 (55.91%) 44,209,756.83 (67.01%)			
public	202.00 (12.25%) 3,486,900.48 (5.09%)		237.00 (15.55%) 6,304,519.60 (9.56%)		104.60 *<0.0001*	5.47 *0.0028*
none	221.00 (13.40%) 7,704,122.50 (11.24%)		398.00 (26.12%) 14,058,681.86 (21.31%)			
missing	36.00 (2.18%) 1,414,610.78 (2.06%		37.00 (2.43%) 1,400,419.73 (2.12%)			
Regular Source of Healthcare (hp2cat)						
yes[R]	1,393.00 (84.48%) 57,510,193.88 (83.92%)		1,280.00 (83.99%) 57,948,088.45 (87.84%)		0.14 *0.707*	0.57 *0.454*
no	256.00 (15.52%) 11,018,461.53 (16.08%)		244.00 (16.01%) 8,025,289.58 (12.16%)			
Source of Healthcare (hcsre)						
healthcare provider[R]	981.00 (59.49%) 42,334,589.32 (61.78%)		826.00 (54.20%) 42,827,250.68 (64.92%)			
clinic	319.00 (19.35%) 11,550,404.67 (16.85%)		357.00 (23.43%) 12,217,692.20 (18.52%)		10.88 *0.012*	0.25 *0.859*
ER or none	323.00 (19.59%) 13,205,152.16 (19.27%)		319.00 (20.93%) 9,757,504.60 (14.79%)			
missing	26.00 (1.58%) 1,438,509.25 (2.10%)		22.00 (1.44%) 1,170,930.55 (1.77%)			

Table 49
Bivariate Analyses of Independent Variables on Lead Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30	Below Geometric Mean		At or Above Geometric Mean		χ^2 *p value unweighted*	χ^2 *p value weighted*
Nutritional Status						
Food Security (food2cat)						
	1,373.00		1,199.00			
	(83.26%)		(78.67%)			
	59,441,670.08		54,602,242.59			
food secure[R]	(86.74%)		(82.76%)			
	211.00		248.00			
	(12.80%)		(16.27%)		10.86	0.94
	6,584,798.88		7,649,723.16		*0.004*	*0.398*
food insecure	(9.61%)		(11.60%)			
	65.00		77.00			
	(3.94%)		(5.05%)			
	2,502,186.44		3,721,412.28			
missing	(3.65%)		(5.64%)			
Body Mass Index (bmi30cat)						
	1,229.00		1,128.00			
<30.0[R]	(74.53%)		(74.02%)			
underweight	52,465,286.32		50,378,610.60			
normal	(76.56%)		(76.36%)			
overweight	394.00		383.00			
	(23.89%)		(25.13%)		3.89	0.05
	15,255,751.26		14,960,706.54		*0.142*	*0.954*
30.0+	(22.26%)		(22.68%)			
obese	26.00		13.00			
	(1.58%)		(0.85%)			
	807,617.82		634,060.89			
missing	(1.18%)		(0.96%)			
Fat Intake/AMDR (fat3cat)						
	1,041.00		1,007.00			
	(63.13%)		(66.25%)			
	41,953,958.29		44,725,757.50			
recommended or less[R]	(61.22%)		(67.93%)		3.37	3.46
	608.00		513.00		*0.066*	*0.069*
	(36.87%)		(33.75%)			
	26,574,697.11		21,117,948.89			
more than recommended	(38.78%)		(32.07%)			
Protein Intake/AMDR (prot3cat)						
	1,456.00		1,256.00			
	(88.30%)		(82.41%)			
	61,807,745.82		56,956,019.45			
recommended or more[R]	(90.19%)		(86.33%)		22.06	2.41
	193.00		268.00		*<0.0001*	*0.127*
	(11.70%)		(17.59%)			
	6,720,909.58		9,017,358.58			
less than recommended	(9.81%)		(13.67%)			
Iron Intake/RDA (iron2cat)						
	1,362.00		1,152.00			
	(82.60%)		(75.59%)			
	56,771,475.34		50,670,842.64			
recommended or more[R]	(82.84%)		(76.80%)		23.62	3.14
	287.00		372.00		*<0.0001*	*0.083*
	(17.40%)		(24.41%)			
	11,757,180.06		15,302,535.39			
less than recommended	(17.16%)		(23.20%)			
Calcium Intake/RDA (calc2cat)						
	390.00		250.00			
	(23.65%)		(16.40%)			
	14,461,237.96		11,615,071.24			
recommended or more[R]	(21.10%)		(17.61%)		25.83	0.66
	1,259.00		1,274.00		*<0.0001*	*0.420*
	(76.35%)		(83.60%)			
	54,067,417.44		54,358,306.79			
less than recommended	(78.90%)		(82.39%)			

Table 49
Bivariate Analyses of Independent Variables on Lead Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30	Below Geometric Mean		At or Above Geometric Mean		χ^2 *p value unweighted*	χ^2 *p value weighted*
Selenium Intake/RDA (sele2cat)						
recommended or more[R]	1,372.00 (83.20%) 58,048,368.20 (84.71%)		1,185.00 (77.76%) 54,597,023.61 (82.76%)		15.01 *0.0001*	0.33 *0.567*
less than recommended	277.00 (16.80%) 10,480,287.20 (15.29%)		339.00 (22.24%) 11,376,354.42 (17.24%)			
Reproductive Status						
Current Pregnancy (pregnant)						
pregnant	260.00 (15.77%) 3,622,017.44 (5.29%)		131.00 (8.60%) 1,220,171.65 (1.85%)			
not pregnant[R]	1,314.00 (79.68%) 62,923,534.24 (91.82%)		1,327.00 (87.07%) 63,452,984.69 (96.18%)		38.33 *<0.0001*	6.49 *0.003*
missing	75.00 (4.55%) 1,983,103.72 (2.89%)		66.00 (4.33%) 1,300,221.69 (1.97%)			
Trimester of Pregnancy (tripcorr)						
not pregnant[R]	1,389.00 (84.23%) 64,906,637.96 (94.71%)		1,393.00 (91.40%) 64,753,206.38 (98.15%)			
1st trimester	88.00 (5.34%) 1,429,778.42 (2.09%)		61.00 (4.00%) 561787.69 (0.85%)		43.35 *<0.0001*	4.18 *0.0108*
2nd trimester	96.00 (5.82%) 1,183,077.47 (1.73%)		36.00 (2.36%) 340,418.06 (0.52%)			
3rd trimester	76.00 (4.61%) 1,009,161.55 (1.47%)		34.00 (2.23%) 317,965.89 (0.48%)			
Ever Pregnant (tprg2cat)						
never pregnant[R]	926.00 (56.16%) 38,526,860.71 (56.22%)		609.00 (39.96%) 21,038,236.27 (31.89%)		83.17 *<0.0001*	12.20 *0.0011*
one or more pregnancies	723.00 (43.84%) 30,001,794.69 (43.78%)		915.00 (60.04%) 44,935,141.76 (68.11%)			
Live Births (live)						
no live births[R]	1,079.00 (65.43%) 42,276,920.40 (61.69%)		741.00 (48.62%) 25,143,317.82 (38.11%)		91.52 *<0.0001*	11.67 *0.0014*
one or more live births	570.00 (34.57%) 26,251,735.00 (38.31%)		783.00 (51.38%) 40,830,060.21 (61.89%)			
Ever Breastfed (brstfda)						
never breastfed[R]	1,275.00 (77.32%) 50,391,371.75 (73.53%)		1,048.00 (68.77%) 41,562,950.55 (63.00%)		29.54 *<0.0001*	4.89 *0.032*
breastfed more than one month or currently	374.00 (22.68%) 18,137,283.65 (26.47%)		476.00 (31.23%) 24,410,427.48 (37.00%)			

Table 49
Bivariate Analyses of Independent Variables on Lead Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30	Below Geometric Mean		At or Above Geometric Mean		χ^2 *p value unweighted*	χ^2 *p value weighted*
Exposure-Related Attributes						
Acculturation						
Birthplace (born2cat)						
U.S.[R]	1,513.00 (91.75%) 63,840,566.10 (93.16%)		1,160.00 (76.12%) 56,463,130.70 (85.58%)		145.89 *<0.0001*	7.61 *0.0084*
outside U.S.	136.00 (8.25%) 4,688,089.30 (6.84%)		364.00 (23.88%) 9,510,247.33 (14.42%)			
Years in U.S. (yrus5)						
born in U.S.[R]	1,513.00 (91.75%) 63,840,566.10 (93.16%)		1,160.00 (76.42%) 56,463,130.70 (85.80%)			
five or more years	111.00 (6.73%) 4,165,110.26 (6.08%)		241.00 (15.88%) 6,908,408.95 (10.50%)		149.07 *<0.0001*	8.05 *0.001*
less than five years	* (0.00%) 522,979.04 (0.76%)		117.00 (7.71%) 2,432,987.87 (3.70%)			
Language Spoken at Home (lang2cat)						
English[R]	1,575.00 (95.63%) 66,327,561.13 (97.11%)		1,269.00 (83.27%) 60,433,633.23 (91.60%)		130.76 *<0.0001*	15.45 *0.0003*
Other	72.00 (4.37%) 1,974,026.91 (2.89%)		255.00 (16.73%) 5,539,744.80 (8.40%)			
U.S. Citizenship (usczn2cat)						
U.S. citizen[R]	1,581.00 (95.88%) 67,122,982.83 (97.95%)		1,233.00 (80.96%) 59,702,289.08 (90.52%)		175.97 *<0.0001*	20.42 *0.0000*
non-U.S. citizen	68.00 (4.12%) 1,405,672.57 (2.05%)		290.00 (19.04%) 6,249,220.26 (9.48%)			
Diet						
Seafood Eaten in Past 30 Days (smpw2cat)						
none[R]	384.00 (23.29%) 12,454,300.40 (18.17%)		302.00 (19.82%) 10,416,540.38 (15.79%)		5.63 *0.018*	0.68 *0.414*
any	1,265.00 (76.71%) 56,074,355.00 (81.83%)		1,222.00 (80.18%) 55,556,837.65 (84.21%)			
Fish Eaten in Past 30 Days (fish2cat)						
none[R]	560.00 (33.96%) 19,647,726.77 (28.67%)		480.00 (31.50%) 17,162,012.91 (26.01%)		2.18 *0.139*	0.71 *0.404*
any	1,089.00 (66.04%) 48,880,928.64 (71.33%)		1,044.00 (68.50%) 48,811,365.12 (73.99%)			

Table 49
Bivariate Analyses of Independent Variables on Lead Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30	Below Geometric Mean		At or Above Geometric Mean		χ^2 *p value unweighted*	χ^2 *p value weighted*
Shellfish Eaten in Past 30 Days (shell2cat)						
none[R]	858.00 (52.03%) 34,676,194.15 (50.60%)		699.00 (45.87%) 28,342,445.03 (42.96%)		12.05 *0.0005*	1.72 *0.197*
any	791.00 (47.97%) 33,852,461.25 (49.40%)		825.00 (54.13%) 37,630,933.00 (57.04%)			
Tap Water Consumed Prior 24h (tap2kct)						
none[R]	614.00 (37.23%) 21,450,765.92 (31.30%)		515.00 (33.79%) 1,9054,062.33 (28.88%)			
< 2,000 ml	804.00 (48.76%) 37,062,060.55 (54.08%)		734.00 (48.16%) 33,983,424.87 (51.51%)		18.43 *0.0004*	0.87 *0.465*
2,000+ ml	150.00 (9.10%) 7,062,800.32 (10.31%)		145.00 (9.51%) 8,466,752.18 (12.83%)			
missing	81.00 (4.91%) 2,953,028.61 (4.31%)		130.00 (8.53%) 4,469,138.66 (6.77%)			
Alcohol Consumption						
Alcohol Consumption (retohuse)						
never, seldom drinker[R] *including 16-19 y/o*	954.00 (57.85%) 30,947,002.42 (45.16%)		789.00 (51.77%) 21,273,512.95 (32.25%)			
drinker	393.00 (23.83%) 20,442,176.43 (29.83%)		337.00 (22.11%) 20,227,903.14 (30.66%)		32.76 *<0.0001*	2.17 *0.104*
heavy drinker	228.00 (13.83%) 14,365,946.82 (20.96%)		327.00 (21.46%) 21,399,432.62 (32.44%)			
missing	74.00 (4.49%) 2,773,529.73 (4.05%)		71.00 (4.66%) 3,072,529.32 (4.66%)			
Tobacco Use						
Serum Cotinine (cot3cat)						
< 1.0 ng/ml[R]	1,373.00 (83.72%) 57,274,884.18 (83.94%)		995.00 (65.55%) 41,596,589.39 (63.37%)			
1.0 - 10.0 ng/ml	79.00 (4.82%) 2,665,982.02 (3.91%)		111.00 (7.31%). 2,584,319.60 (3.94%)		144.86 *<0.0001*	9.52 *0.0004*
> 10.0 ng/ml	188.00 (11.46%) 8,289,862.88 (12.15%)		412.00 (27.14%) 21,460,478.36 (32.69%)			

Table 49
Bivariate Analyses of Independent Variables on Lead Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30	Below Geometric Mean		At or Above Geometric Mean		χ^2 *p value unweighted*	χ^2 *p value weighted*
ETS (ETS)						
	1,303.00		1,114.00			
	(79.02%)		(73.10%)			
	55,492,798.64		46,304,572.94			
no ETS[R]	(80.98%)		(70.19%)			
	296.00		354.00		15.39	3.19
	(17.95%)		(23.23%)		*0.0005*	*0.051*
	10,918,510.24		15,788,359.36			
ETS at home or work	(15.93%)		(23.93%)			
	50.00		56.00			
	(3.03%)		(3.67%)			
	2,117,346.52		3,880,445.73			
ETS at home and work	(3.09%)		(5.88%)			
Residence						
Tap Water Source (h2os2cat)						
	1,444.00		1,382.00			
	(87.57%)		(90.68%)			
	59,518,100.62		57,217,807.54			
public[R]	(86.85%)		(86.73%)			
	170.00		96.00		18.55	0.19
	(10.31%)		(6.30%)		*<0.0001*	*0.827*
	7,567,628.68		6,923,906.34			
private	(11.04%)		(10.50%)			
	35.00		46.00			
	(2.12%)		(3.02%)			
	1,442,926.10		1,831,664.14			
missing	(2.11%)		(2.78%)			
Residential Tap Water Treatment (h2ox2cat)						
	519.00		344.00			
	(31.47%)		(22.57%)			
	24,424,707.57		20,883,527.10			
yes	(35.64%)		(31.65%)			
	1,096.00		1,143.00		31.73	0.25
	(66.46%)		(75.00%)		*<0.0001*	*0.779*
	42,669,608.13		43,875,729.23			
no[R]	(62.27%)		(66.51%)			
	34.00		37.00			
	(2.06%)		(2.43%)			
	1,434,339.70		1,214,121.70			
missing	(2.09%)		(1.84%)			
Type of Residence (res3cat)						
	1,090.00		982.00			
	(66.10%)		(64.44%)			
	44,740,021.12		44,565,949.44			
attached or detached house[R]	(65.29)		(67.55%)			
	82.00		120.00		11.24	3.72
	(4.97%)		(7.87%)		*0.003*	*0.032*
	2,426,410.42		5,975,367.38			
mobile home or trailer	(3.54%)		(9.06%)			
	477.00		422.00			
	(28.93%)		(27.69%)			
all other types	21,362,223.86		15,432,061.21			
including missing/unknown	(31.17%)		(23.39%)			
Age of Residence (resb60cat)						
	958.00		637.00			
	(58.10%)		(41.80%)			
	43,391,502.90		34,653,021.12			
1960 or newer[R]	(63.32%)		(52.53%)			
	382.00		384.00		106.19	3.34
	(23.17%)		(25.20%)		*<0.0001*	*0.045*
	15,705,459.67		16,386,740.43			
older than 1960	(22.92%)		(24.84%)			
	309.00		503.00			
	(18.74%)		(33.01%)			
	9,431,692.83		14,933,616.49			
missing/unknown	(13.76%)		(22.64%)			

Table 49
Bivariate Analyses of Independent Variables on Lead Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30	Below Geometric Mean		At or Above Geometric Mean		χ^2 *p value unweighted*	χ^2 *p value weighted*
Age of Residence (resb78cat)						
1978 or newer[R]	639.00 (38.75%) 30,166,573.45 (44.02%)		448.00 (29.40%) 25,221,475.40 (38.23%)			
older than 1978	701.00 (42.51%) 28,930,389.12 (42.22%)		573.00 (37.60%) 25,818,286.14 (39.13%)		87.98 *<0.0001*	2.24 *0.118*
missing/unknown	309.00 (18.74%) 9,431,692.83 (13.76%)		503.00 (33.01%) 14,933,616.49 (22.64%)			
Resident Status (resd3cat)						
own[R]	936.00 (56.76%) 37,822,046.75 (55.19%)		791.00 (51.90%) 39,428,260.38 (59.76%)			
rent	619.00 (37.54%) 27,252,592.24 (39.77%)		659.00 (43.24%) 23,911,303.33 (36.24%)		10.89 *0.004*	0.50 *0.608*
other *including missing*	94.00 (5.70%) 3,454,016.42 (5.04%)		74.00 (4.86%) 2,633,814.32 (3.99%)			
Years at Current Residence (re5yrcat)						
more than five years[R]	560.00 (33.96%) 21,445,224.57 (31.29%)		553.00 (36.29%) 24,449,094.12 (37.06%)			
five years or less	1,067.00 (64.71%) 46,043,199.30 (67.19%)		940.00 (61.68%) 40,412,413.64 (61.26%)		4.69 *0.090*	0.92 *0.407*
missing	22.00 (1.33%) 1,040,231.53 (1.52%)		31.00 (2.03%) 1,111,870.27 (1.69%)			
Household Size (hsize)						
four persons or less[R]	1,170.00 (70.95%) 54,111,047.44 (78.96%)		1,012.00 (66.40%) 52,342,980.95 (79.34%)			
more than four persons	479.00 (29.05%) 14,417,607.96 (21.04%)		512.00 (33.60%) 13,630,397.08 (20.66%)		7.63 *0.005*	0.01 *0.917*
Rooms in Residence (rm3cat)						
7+ rooms[R]	658.00 (39.90%) 27,588,606.15 (40.26%)		490.00 (32.15%) 25,027,906.66 (37.94%)			
4-6 rooms	823.00 (49.91%) 33,258,195.04 (48.53%)		868.00 (56.96%) 35,841,937.55 (54.33%)		20.91 *0.0001*	0.78 *0.508*
1-3 rooms	132.00 (8.00%) 6,188,856.51 (9.03%)		131.00 (8.60%) 3,986,316.60 (6.04%)			
missing	36.00 (2.18%) 1492997.69 (2.18%)		35.00 (2.30%) 1,117,217.23 (1.69%)			

Table 49
Bivariate Analyses of Independent Variables on Lead Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30	Below Geometric Mean		At or Above Geometric Mean		χ^2 *p value unweighted*	χ^2 *p value weighted*
Occupation						
Current Occupation (cocc2cat)						
not working[R]	662.00 (40.15%) 19,925,730.66 (29.08%)		662.00 (43.44%) 22,247,226.92 (33.72%)			
management, professional & sales	699.00 (42.39%) 34,344,959.83 (50.12%)		544.00 (35.70%) 33,413,931.91 (50.65%)		15.91 *0.0004*	1.11 *0.339*
services & goods	288.00 (17.47%) 14,257,964.91 (20.81%)		318.00 (20.87%) 10,312,219.21 (15.63%)			
Time in Current Employment (cjt)						
not working[R]	662.00 (40.15%) 1,992,730.66 (29.08%)		662.00 (43.44%) 22,247,226.92 (33.72%)			
less than five years	819.00 (49.67%) 39,135,012.39 (57.11%)		615.00 (40.35%) 28,106,627.34 (42.60%)		39.19 *<0.0001*	3.39 *0.043*
five or more years	168.00 (10.19%) 9,467,912.35 (13.82%)		247.00 (16.21%) 15,619,523.77 (23.68%)			
Total Hours Worked Prior Week (hrwk)						
not employed[R]	687.00 (41.69%) 21,100,851.66 (30.85%)		694.00 (45.57%) 24,707,186.37 (37.46%)			
less than 35 hours	424.00 (25.73%) 19,069,618.35 (27.88%)		312.00 (20.49%) 14,297,815.45 (21.68%)		12.55 *0.019*	2.47 *0.096*
35+ hours	537.00 (32.58%) 28,235,013.48 (41.28%)		517.00 (33.95%) 26,946,507.53 (40.86%)			
Longest Held Occupation (locc2cat)						
not applicable[R]	789.00 (47.85%) 31,338,831.82 (45.73%)		773.00 (50.72%) 32,778,524.69 (49.68%)			
management, professional & sales	464.00 (28.14%) 20,407,781.11 (29.78%)		439.00 (28.81%) 21,007,405.04 (31.84%)		5.91 *0.05*	0.48 *0.623*
services & goods	396.00 (24.01%) 16,782,042.47 (24.49%)		312.00 (20.47%) 12,187,448.30 (18.47%)			
Time in Longest Employment (ljt)						
not applicable[R]	789.00 (47.85%) 31,338,831.82 (45.73%)		773.00 (50.72%) 32,778,524.69 (49.68%)			
less than five years	565.00 (34.26%) 19,352,964.01 (28.24%)		432.00 (28.35%) 15,189,304.84 (23.02%)		13.94 *0.0009*	0.67 *0.517*
five or more years	295.00 (17.89%) 17,836,859.57 (26.03%)		319.00 (20.93%) 18,005,548.50 (27.29%)			

Table 49
Bivariate Analyses of Independent Variables on Lead Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30	Below Geometric Mean		At or Above Geometric Mean		χ^2 *p value unweighted*	χ^2 *p value weighted*
Socioeconomic Factors						
Education						
Highest Education (educ2)						
high school diploma, GED or higher[R]	1,096.00 (66.46%) 54,561,650.03 (79.62%)		941.00 (61.79%) 52,345,511.31 (79.42%)		7.54 *0.006*	0.001 *0.973*
less than high school diploma	553.00 (33.54%) 13,967,005.37 (20.38%)		582.00 (38.21%) 13,560,727.93 (20.58%)			
Employment						
Employment Status (emp3cat)						
employed	988.00 (59.99%) 48,624,413.53 (70.97%)		865.00 (56.76%) 43,844,386.43 (66.46%)		3.39 *0.065*	1.39 *0.244*
not employed[R]	659.00 (40.01%) 19,893,022.35 (29.03%)		659.00 (43.24%) 22,128,991.60 (33.54%)			
Reason for Unemployment (unem2cat)						
working[R]	988.00 (59.92%) 48,624,413.53 (70.95%)		865.00 (56.76%) 43,844,386.43 (66.46%)		29.80 *<0.0001*	2.65 *0.060*
voluntary unemployment	466.00 (28.26%) 14,209,493.25 (20.74%)		458.00 (30.05%) 14,055,528.43 (21.30%)			
involuntary unemployment	123.00 (7.46%) 3,277,577.66 (4.78%)		172.00 (11.29%) 6,985,536.35 (10.59%)			
missing	72.00 (4.37%) 2,417,170.97 (3.53%)		29.00 (1.90%) 1,087,926.82 (1.65%)			
Work History (wkcp)						
never employed[R]	190.00 (11.52%) 3,194,843.15 (4.66%)		218.00 (14.30%) 5,043,967.65 (7.65%)			
currently employed	599.00 (36.33%) 28,143,988.67 (41.07%)		555.00 (36.42%) 27,734,557.04 (42.04%)		8.98 *0.029*	1.13 *0.348*
employed in the past but not currently	472.00 (28.62%) 16,730,887.51 (24.41%)		444.00 (29.13%) 17,203,259.27 (26.08%)			
employed now and in the past	388.00 (23.53%) 20,458,936.07 (29.85%)		307.00 (20.14%) 15,991,594.07 (24.24%)			

Table 49
Bivariate Analyses of Independent Variables on Lead Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30	Below Geometric Mean		At or Above Geometric Mean		χ^2 *p value unweighted*	χ^2 *p value weighted*
Income						
U.S. Poverty Threshold (pov2cat)						
more than 1.00[R]	1,237.00 (75.02%) 52,645,984.77 (76.82%)		990.00 (64.96%) 51,307,638.42 (77.77%)		43.72 *<0.0001*	0.45 *0.641*
1.00 or less	333.00 (20.19%) 12,438,247.06 (18.15%)		397.00 (26.05%) 10,148,950.41 (15.38%)			
missing	79.00 (4.79%) 3,444,423.57 (5.03%)		137.00 (8.99%) 4,516,789.20 (6.85%)			
Marital Status						
Marital Status (marr3cat)						
married or living with partner	567.00 (34.38%) 26,626,378.49 (38.85%)		631.00 (41.40%) 35,174,269.76 (53.32%)		82.89 *<0.0001*	9.23 *0.0001*
widowed, divorced or separated	86.00 (5.22%) 4,130,631.35 (6.03%)		175.00 (11.48%) 10,223,321.03 (15.50%)			
never married[R]	964.00 (58.46%) 36,238,595.44 (52.88%)		673.00 (44.16%) 17,254,356.05 (26.15%)			
missing	32.00 (1.94%) 1,533,050.12 (2.24%)		45.00 (2.95%) 3,321,431.19 (5.03%)			
Race-Ethnicity						
Race-Ethnicity (race5cat)						
Non-Hispanic White[R]	899.00 (54.52%) 52,707,454.40 (76.91%)		594.00 (38.98%) 45,180,089.76 (68.48%)		84.47 *<0.0001*	5.12 *0.0018*
Non-Hispanic Black	258.00 (15.65%) 4,619,603.97 (6.74%)		365.00 (23.95%) 8,127,574.39 (12.32%)			
Mexican American	333.00 (20.19%) 3,455,944.04 (5.04%)		412.00 (27.03%) 5,214,631.76 (7.90%)			
Other Hispanic	92.00 (5.58%) 4,332,333.00 (6.32%)		86.00 (5.64%) 3,193,659.22 (4.84%)			
Asian, Native American, Pacific Islander & Multi-Racial	67.00 (4.06%) 3,413,319.99 (4.98%)		67.00 (4.40%) 4,257,422.89 (6.45%)			

Table 49
Bivariate Analyses of Independent Variables on Lead Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30	Below Geometric Mean		At or Above Geometric Mean		χ^2 *p value unweighted*	χ^2 *p value weighted*
Race-Ethnicity/Hispanic Grouping (race4cat)						
Non-Hispanic White^{R}	899.00 (54.52%) 52,707,454.40 (76.91%)		594.00 (38.98%) 45,180,089.76 (68.48%)			
Non-Hispanic Black	258.00 (15.65%) 4,619,603.97 (6.74%)		365.00 (23.95%) 8,127,574.39 (12.32%)		81.66 *<0.0001*	5.08 *0.004*
Hispanic	425.00 (25.77%) 7,788,277.04 (11.36%)		498.00 (32.68%) 8,408,290.98 (12.74%)			
Asian, Native American, Pacific Islander & Multi-Racial	67.00 (4.06%) 3,413,319.99 (4.98%)		67.00 (4.40%) 4,257,422.89 (6.45%)			

Table 50

Summary of Chi-Square (χ^2) and p Values of Weighted Independent Variables on Lead (1999-2004)

Variables *p < 0.20*	*p* value	χ^2 *weighted*	Variables *p > 0.20*	*p* value	χ^2 *weighted*
U.S. Citizenship (usczn2cat)	*0.0000*	20.42	Employment Status (emp3cat)	*0.244*	1.39
Marital Status (marr3cat)	*0.0001*	9.23	Current Occupation (cocc2cat)	*0.339*	1.11
Age (age4cat)	*0.0001*	9.12	Work History (wkcp)	*0.348*	1.13
Perceived Health Status (huq2cat)	*0.0003*	15.58	Iron Deficiency and Treatment (FeDTx)	*0.355*	1.11
Language Spoken at Home (lang2cat)	*0.0003*	15.45	Food Security (food2cat)	*0.398*	0.94
Serum Cotinine (cot3cat)	*0.0004*	9.52	Fish in Past 30 Days (fish2cat)	*0.404*	0.71
Years in U.S. (yrus5)	*0.001*	8.05	Years at Current Residence (re5yrcat)	*0.407*	0.92
Ever Pregnant[1] (tprg2cat)	*0.0011*	12.20	Seafood in Past 30 Days [1] (smpw2cat)	*0.414*	0.68
Live Births (live)	*0.0014*	11.67	Calcium Intake/RDA (calc2cat)	*0.420*	0.66
Race-Ethnicity[1] (race5cat)	*0.0018*	5.12	Iron Deficiency (FeD2cat)	*0.453*	0.57
Health Insurance (hi2cat)	*0.0028*	5.47	Regular Source of Healthcare (hp2cat)	*0.454*	0.57
Current Pregnancy (pregnant)	*0.003*	6.49	Tap Water Consumed 24h (tap2kct)	*0.465*	0.87
Race-Ethnicity/Hispanic Grouping (race4cat)	*0.004*	5.08	Rooms in Residence (rm3cat)	*0.508*	0.78
Birthplace[1] (born2cat)	*0.0084*	7.61	Time in Longest Employment (ljt)	*0.517*	0.67
Trimester of Pregnancy[1] (tripcorr)	*0.0108*	4.18	Selenium Intake/RDA (sele2cat)	*0.567*	0.33
Ever Breastfed (brstfda)	*0.032*	4.89	Resident Status (resd3cat)	*0.608*	0.50
Type of Residence (res3cat)	*0.032*	3.72	Longest Held Occupation (locc2cat)	*0.623*	0.48
Charleson Co-Morbidity Scale (CCMS3cat)	*0.0415*	0.89	U.S. Poverty Threshold (pov2cat)	*0.641*	0.45
Time in Current Employment (cjt)	*0.043*	3.39	Treatment for Iron Deficiency past 3 mo (FeTx2cat)	*0.719*	0.13
Age of Residence 1960 (resb60cat)	*0.045*	3.34	Residential Tap Water Treatment (h2ox2cat)	*0.779*	0.25
Environmental Tobacco Smoke (ETS)	*0.051*	3.19	Tap Water Source (h2os2cat)	*0.827*	0.19
Reason for Unemployment (unem2cat)	*0.060*	2.65	Source of Healthcare (hscre)	*0.859*	0.25
Fat Intake/AMDR[1] (fat3cat)	*0.069*	3.46	Household Size (hsize)	*0.917*	0.01
Iron Intake/RDA (iron2cat)	*0.083*	3.14	Body Mass Index (bmi30cat)	*0.954*	0.05
Total Hours Worked Prior Week (hrwk)	*0.096*	2.47	Highest Education (educ2)	*0.973*	0.001
Alcohol Consumption (retohuse)	*0.104*	2.17			
Age of Residence (resb78cat)	*0.118*	2.24			
Protein Intake/AMDR[1] (prot3cat)	*0.127*	2.41			
Shellfish in Past 30 Days[1] (shell2cat)	*0.197*	1.72			

[1]variable dropped due to low cell size or too similar to other variables

Table 51
Stepwise Regression Analyses of Lead (1999-2004)

Variable Name *p <0.20*	df	-2LL Wald F	Difference	df	*p* value
Initial Regression	43	989.03			
1 Total Hours Worked Prior Week (hrwk)	41	982.83	6.20	2	*<0.05* *keep*
2 Reason for Unemployment (unem2cat)	40	982.23	6.80	3	*>0.05* *drop*
3 Language Spoken at Home (lang2cat)	39	979.45	2.88	1	*>0.05* *drop*
4 Ever Breastfed (brstfda)	38	978.68	0.77	1	*>0.20* *drop*
5 Charleson Co-Morbidity Scale (CCMS3cat)	36	969.95	8.73	2	*<0.02* *keep*
6 Environmental Tobacco Smoke (ETS)	36	974.50	4.18	2	*>0.05* *drop*
7 Perceived Health Status (huq2cat)	35	974.63	0.13	1	*>0.20* *drop*
8 Time in Current Employment (cjt)	33	961.77	12.86	2	*<0.01* *keep*
9 Iron Intake/RDA (iron2cat)	34	971.65	2.95	1	*>0.05* *drop*
10 Live Births (live)	33	968.97	2.68	1	*>0.10* *drop*
11 Years in U.S. (yrus5)	31	965.55	3.42	2	*>0.10* *drop*
12 Type of Residence (res3cat)	29	951.13	14.42	2	*<0.001* *keep*
13 Protein Intake/AMDR (prot3cat)	30	953.24	12.31	1	*<0.001* *keep*
14 Race-Ethnicity/Hispanic Grouping (race4cat)	28	935.33	30.22	3	*<0.001* *keep*
15 Marital Status (marr3cat)	28	927.02	38.53	3	*<0.001* *keep*
16 Age of Residence 1960 (resb60cat)	29	912.81	52.74	2	*<0.001* *keep*
17 Serum Cotinine (cot3cat)	29	906.75	58.80	2	*<0.001* *keep*
18 Alcohol Consumption (retohuse)	28	879.18	86.37	3	*<0.001* *keep*
19 U.S. Citizenship (usczn2cat)	30	879.17	86.38	1	*<0.001* *keep*
20 Current Pregnancy (pregnant)	29	940.52	25.03	2	*<0.001* *keep*
21 Health Insurance (hi2cat)	28	920.86	44.69	3	*<0.001* *keep*
22 Age (age4cat)	28	826.37	139.18	3	*<0.001* *keep*

Table 52
Best-Fit Logistic Regression Lead Model *with no interactions* (1999-2004)

Variable Name	df	-2LL Wald F	R^2		*p* value
Best Fit Regression	31	965.55	*0.2636*		
U.S. Citizenship (usczn2cat)	1	29.11			*0.0000*
Age (age4cat)	3	6.12			*0.0014*
Age of Residence 1960 (resb60cat)	2	6.26			*0.0041*
Serum Cotinine (cot3cat)	2	5.16			*0.0097*
Health Insurance (hi2cat)	3	4.18			*0.0109*
Current Pregnancy (pregnant)	2	4.96			*0.0114*
Alcohol Consumption (retohuse)	3	3.59			*0.0209*
Race-Ethnicity/Hispanic Grouping (race4cat)	3	3.36			*0.0269*
Marital Status (marr3cat)	3	2.29			*0.0917*
Protein Intake/AMDR (prot3cat)	1	1.92			*0.1727*
Type of Residence (res3cat)	2	1.62			*0.2089*
Time in Current Employment (cjt)	2	0.97			*0.3877*
Charleson Co-Morbidity Scale (CCMS3cat)	2	0.44			*0.6481*
Total Hours Worked Prior Week (hrwk)	2	0.20			*0.8160*

[1] in ascending order by p value

Table 53
Variance Inflation Factor Test for Collinearity Among Independent Variables using the Best-Fit Logistic Regression Lead Model (1999-2004)

	df	sum of squares	mean square	F value	pr>F	
Best-Fit Lead Model *with no interactions*	14	1121.85	80.132	442.98	*<0.001*	

Variable Name	df	parameter estimate	std error	t value	pr>t	VIF
Intercept	1	-0.758	0.031	*-24.36*	*<0.0001*	0.0000
Age (age4cat)	1	0.117	0.004	30.73	*<0.0001*	2.1871
Charleson Co-Morbidity Scale (CCMS3cat)	1	-0.017	*0.008*	-2.16	*0.0308*	1.0241
Health Insurance (hi2cat)	1	0.053	*0.004*	14.54	*<0.0001*	1.1648
Protein Intake/AMDR (prot3cat)	1	0.009	*0.008*	1.05	*0.2943*	1.0254
Current Pregnancy (pregnant)	1	0.218	*0.005*	42.67	*<0.0001*	1.1099
U.S. Citizenship (usczn2cat)	1	0.198	*0.010*	20.19	*<0.0001*	1.2777
Alcohol Consumption (retohuse)	1	0.021	*0.004*	5.39	*<0.0001*	1.4341
Serum Cotinine (cot3cat)	1	0.091	*0.004*	25.55	*<0.0001*	1.1252
Age of Residence 1960 (resb60cat)	1	0.012	*0.004*	2.70	*0.0070*	1.1350
Type of Residence (res3cat)	1	-0.002	*0.004*	-0.55	*<0.5832*	1.1372
Time in Current Employment (cjt)	1	0.002	*0.007*	0.29	*0.7714*	3.0955
Total Hours Worked Prior Week (hrwk)	1	-0.019	*0.006*	-3.41	*0.0006*	2.8840
Marital Status (marr3cat)	1	-0.0004	*0.004*	-0.09	*0.9243*	1.8221
Race-Ethnicity/Hispanic Grouping (race4cat)	1	0.027	*0.003*	7.96	*<0.0001*	1.2297

Table 54
Statistical Significance of Interactions Between Independent Variables using the Best-Fit Logistic Regression Lead Model (1999-2004)

Independent Variables	Age (age4cat)	Charleson Co-Morbidity Scale (CCMS3cat)	Health Insurance (hi2cat)	Protein Intake/AMDR (prot3cat)	Current Pregnancy (pregnant)	U.S. Citizenship (usczn2cat)	Alcohol Consumption (retohuse)	Serum Cotinine (cot3cat)	Age of Residence 1960 (resb60cat)	Type of Residence (res3cat)	Time in Current Employment (cjt)	Total Hours Worked Prior Week (hrwk)	Marital Status (marr3cat)	Race-Ethnicity Hispanic Grouping (race4cat)
Age (age4cat)														
Charleson Co-Morbidity Scale (CCMS3cat)	*op*													
Health Insurance (hi2cat)	*op*	*op*												
Protein Intake/AMDR (prot3cat)	*<0.001*	*<0.01*	*op*											
Current Pregnancy (pregnant)	*op*	*op*	*op*	*ns*										
U.S. Citizenship (usczn2cat)	*<0.001*	*op*	*ns*	*ns*	*ns*									
Alcohol Consumption (retohuse)	*op*	*op*	*op*	*<0.001*	*op*	*<0.05*								
Serum Cotinine (cot3cat)	*<0.001*	*op*	*ns*	*ns*	*ns*	*<0.01*	*<0.001*							
Age of Residence 1960 (resb60cat)	*<0.001*	*<0.001*	*ns*	*<0.001*	*ns*	*ns*	*<0.001*	*<0.001*						
Type of Residence (res3cat)	*<0.001*	*op*	*op*	*<0.01*	*<0.001*	*<0.05*	*ns*	*<0.01*	*op*					
Time in Current Employment (cjt)	*<0.001*	*<0.01*	*op*	*<0.01*	*ns*	*<0.05*	*<0.001*	*<0.001*	*<0.02*	*<0.01*				
Total Hours Worked Prior Week (hrwk)	*<0.001*	*<0.02*	*<0.01*	*<0.001*	*<0.01*	*ns*	*<0.001*	*<0.001*	*ns*	*<0.05*	*op*			
Marital Status (marr3cat)	*op*	*op*	*op*	*<0.05*	*op*	*ns*	*<0.001*	*op*	*<0.001*	*<0.001*	*<0.001*	*<0.001*		
Race-Ethnicity/Hispanic Grouping (race4cat)	*<0.001*	*op*	*op*	*ns*	*op*	*op*	*<0.001*	*ns*	*<0.001*	*<0.001*	*ns*	*<0.001*	*op*	

op = over parameterized unable to calculate
ns = not statistically significant p>0.05

Table 55. Odds Ratios and Confidence Intervals for Best-Fit Logistic Regression Lead Model *with no interactions* (1999-2004)

Variable Names	df	-2LL Wald F	*p* value	Odds Ratios	Confidence Intervals
Susceptibility-Related Attributes					
Age (age4cat)	3	6.12	*0.0014*		
16-19[R]				1.00	*ns*
20-29				0.87	0.47 - 1.63 %
30-39				1.60	0.70 - 3.68 %
40-49				4.31	1.93 - 9.62 %
Health Status					
Charleson Co-Morbidity Scale (CCMS3cat)	2	0.44	*0.6481*		
none[R]				1.00	*ns*
one co-morbidity				0.70	0.28 - 1.72 %
more than one co-morbidity				1.48	0.19 - 11.36 %
Health Insurance (hi2cat)	3	4.18	*0.0109*		
private[R]				1.00	*ns*
public				2.68	1.36 - 5.27 %
none				1.87	1.10 - 3.18 %
missing				1.81	0.25 - 13.33 %
Nutritional Status					
Protein Intake/AMDR (prot3cat)	1	1.92	*0.1727*		
recommended or more[R]				1.00	*ns*
less than recommended				1.64	0.80 - 3.37 %
Reproductive Status					
Current Pregnancy (pregnant)	2	4.96	*0.0114*		
pregnant				0.31	0.14 - 0.65 %
not pregnant[R]				1.00	*ns*
missing				1.23	0.45 - 3.36 %
Exposure-Related Attributes					
Acculturation					
U.S. Citizenship (usczn2cat)	1	29.11	*0.0000*		
U.S. citizen[R]				1.00	*ns*
non-U.S. citizen				7.64	3.57 - 16.32 %
Alcohol Consumption					
Alcohol Consumption (retohuse)	3	3.59	*0.0209*		
never, seldom drinker[R] *including 16-19 y/o*				1.00	*ns*
drinker				1.10	0.63 - 1.94 %
heavy drinker				2.83	1.42 - 5.65 %
missing				1.17	0.38 - 3.55 %
Tobacco Use					
Serum Cotinine (cot3cat)	2	5.16	*0.0097*		
< 1.0 ng/ml[R]				1.00	*ns*
1.0 - 10.0 ng/ml				0.99	0.47 - 2.09 %
> 10.0 ng/ml				2.42	1.39 - 4.21 %
Residence					
Type of Residence (res3cat)	2	1.62	*0.2089*		
attached or detached house[R]				1.00	*ns*
mobile home or trailer				1.72	0.80 - 3.69 %
all other types *including missing/unknown*				0.79	0.47 - 1.34 %
Age of Residence 1960 (resb60cat)	2	6.26	*0.0041*		
1960 or newer[R]				1.00	*ns*
older than 1960				1.92	1.27 - 2.88 %
missing/unknown				1.95	0.96 - 3.98 %
Occupation					
Time in Current Employment (cjt)	2	0.97	*0.3877*		
not working[R]				1.00	*ns*
less than five years				0.96	0.28 - 3.26 %
five or more years				1.49	0.40 - 5.49 %

[R] = referent group

ns = not significant

Table 55. Odds Ratios and Confidence Intervals for Best-Fit Logistic Regression Lead Model *with no interactions* (1999-2004)

Variable Names	df	-2LL Wald F	*p* value	Odds Ratios	Confidence Intervals
Total Hours Worked Prior Week (hrwk)	2	0.20	*0.8160*		
not employed[R]				1.00	*ns*
less than 35 hours				0.84	0.30 - 2.42 %
35+ hours				0.73	0.23 - 2.31 %
Socioeconomic Factors					
Marital Status					
Marital Status (marr3cat)	3	2.29	*0.0917*		
married or living with partner				1.83	0.93 - 3.62 %
widowed, divorced or separated				1.70	0.87 - 3.33 %
never married[R]				1.00	*ns*
missing				3.52	1.26 - 9.86 %
Race-Ethnicity					
Race-Ethnicity/Hispanic Grouping (race4cat)	3	3.36	*0.0269*		
Non-Hispanic White[R]				1.00	*ns*
Non-Hispanic Black				2.23	1.24 - 4.02 %
Hispanic				0.87	0.49 - 1.52 %
Asian, Native American, Pacific Islander & Multi-Racial				0.99	0.43 - 2.29 %

[R] = referent group

ns = not significant

Table 56
Bivariate Analyses of Independent Variables on Methylmercury Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30	Below Geometric Mean		At or Above Geometric Mean		χ^2 *p value unweighted*	χ^2 *p value weighted*
Susceptibility-Related Attributes						
Age (age4cat)						
16-19^{R} $n_{unweighted}$ = 1,085 $n_{weighted}$ = 18,510,468.72	810.00 (42.39%)%) 14,455,029.37 (20.45%)		275.00 (21.79%) 4,055,439.35 (6.35%)			
20-29 $n_{unweighted}$ = 884 $n_{weighted}$ = 45,347,514.91	489.00 (25.59%) 22,668,003.72 (32.07%)		395.00 (31.30%) 22,679,511.19 (35.54%)		147.60 *<0.0001*	7.11 *0.0005*
30-39 $n_{unweighted}$ =702 $n_{weighted}$ = 36,357,836.50	355.00 (18.58%) 17,129,874.37 (24.23%)		347.00 (27.50%) 19,227,962.13 (30.13%)			
40-49 $n_{unweighted}$ =502 $n_{weighted}$ = 34,286,213.30	257.00 (13.45%) 16,432,386.61 (23.25%)		245.00 (19.41%) 17,853,826.69 (27.98%)			
Health Status						
Perceived Health Status (huq2cat)						
excellent, very good, good^{R}	1,680.00 (87.96%) 63,450,323.11 (89.80%)		1,160.00 (91.92%) 60,554,921.99 (94.89%)		12.71 *0.0004*	9.87 *0.0030*
fair, poor	230.00 (12.04%) 7,204,062.21 (10.20%)		102.00 (8.08%) 3,261,817.38 (5.11%)			
Charleson Co-Morbidity Scale (CCMS3cat)						
none^{R}	1,686.00 (88.23%) 60,399,874.70 (85.45%)		1,128.00 (89.38%) 57,857,146.73 (90.66%)			
1 co-morbidity	191.00 (9.99%) 8,506,466.25 (12.03%)		112.00 (8.87%) 4,640,267.06 (7.27%)		1.12 *0.572*	1.66 *0.202*
>1 co-morbidity	34.00 (1.78%) 1,778,953.12 (2.52%)		* (0.00%) 1,319,325.57 (2.07%)			
Iron Deficiency (FeD2cat)						
within normal limits^{R}	1,608.00 (84.14%) 63,789,074.49 (90.24%)		1,116.00 (88.43%) 59,047,684.14 (92.53%)		11.49 *0.0007*	1.04 *0.314*
iron deficient $n_{unweighted}$ = 449 $n_{weighted}$ = 11,665,274.81	303.00 (15.86%) 6,896,219.58 (9.76%)		146.00 (11.57%) 4,769,055.23 (7.47%)			
Treatment for Iron Deficiency past 3 mo (FeTx2cat)						
yes	106.00 (5.55%) 2,391,018.45 (3.38%)		65.00 (5.15%) 2,755,277.52 (4.32%)		0.23 *0.632*	0.21 *0.644*
no^{R}	1,805.00 (94.45%) 68,294,275.61 (96.62%)		1,196.00 (94.85%) 61,047,882.77 (95.68%)			

Table 56
Bivariate Analyses of Independent Variables on Methylmercury Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30	Below Geometric Mean		At or Above Geometric Mean		χ^2 *p value unweighted*	χ^2 *p value weighted*
Iron Deficiency and Treatment (FeDTx)						
normal/no treatment[R]	1,534.00 (80.27%) 62,145,752.62 (87.92%)		1,074.00 (85.17%) 57,296,946.06 (89.80%)			
normal w/treatment	74.00 (3.87%) 1,643,321.87 (2.32%)		41.00 (3.25%) 1,737,159.00 (2.72%)		15.70 *0.001*	0.94 *0.431*
deficient w/treatment	32.00 (1.67%) 747,696.58 (1.06%)		* (0.00%) 1,018,118.52 (1.60%)			
deficient/no treatment	271.00 (14.18%) 6,148,523.00 (8.70%)		122.00 (9.67%) 3,750,936.71 (5.88%)			
Health Insurance (hi2cat)						
private[R]	1,141.00 (59.71%) 49,573,386.95 (70.13%)		901.00 (71.39%) 50,559,391.52 (79.23%)			
public	320.00 (16.75%) 6,926,919.83 (9.80%)		119.00 (9.43%) 2,864,500.25 (4.49%)		54.11 *<0.0001*	4.89 *0.005*
none	408.00 (21.35%) 12,652,508.42 (17.90%)		211.00 (16.72%) 9,110,295.94 (14.28%)			
missing	42.00 (2.20%) 1,532,478.86 (2.17%)		31.00 (2.46%) 1,282,551.66 (2.01%)			
Regular Source of Healthcare (hp2cat)						
yes[R]	1,593.00 (83.36%) 61,192,250.32 (86.57%)		1,080.00 (85.58%) 54,266,032.01 (85.03%)		2.82 *0.093*	0.08 *0.774*
no	318.00 (16.64%) 9,493,043.75 (13.43%)		182.00 (14.42%) 955,707.36 (14.97%)			
Source of Healthcare (hcsre)						
healthcare provider[R]	1,042.00 (54.53%) 44,913,152.13 (63.54%)		765.00 (60.62%) 40,248,687.86 (63.07%)			
clinic	442.00 (23.13%) 13,606,076.26 (19.25%)		234.00 (18.54%) 10,162,020.62 (15.92%)		22.05 *<0.0001*	0.446 *0.721*
ER or none	407.00 (21.30%) 11,199,956.05 (15.84%)		235.00 (18.62%) 11,762,700.70 (18.43%)			
missing	20.00 (1.05%) 966,109.62 (1.37%)		28.00 (2.22%) 1,643,330.19 (2.58%)			

Table 56
Bivariate Analyses of Independent Variables on Methylmercury Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30	Below Geometric Mean		At or Above Geometric Mean		χ^2 *p value unweighted*	χ^2 *p value weighted*
Nutritional Status						
Food Security (food2cat)						
food secure^{R}	1,523.00 (79.70%) 59,147,668.27 (83.68%)		1,049.00 (83.12%) 54,896,244.39 (86.02%)			
food insecure	306.00 (16.01%) 8,649,000.70 (12.24%)		153.00 (12.12%) 5,585,521.34 (8.75%)		9.41 *0.009*	1.80 *0.177*
missing	82.00 (4.29%) 2,888,625.09 (4.09%)		60.00 (4.75%) 3,334,973.63 (5.23%)			
Body Mass Index (bmi30cat)						
<30.0^{R} underweight normal overweight	1,392.00 (72.84%) 53,097,503.59 (75.12%)		965.00 (76.47%) 49,746,393.33 (77.95%)			
30.0+ obese	486.00 (25.43%) 16,312,928.21 (23.08%)		291.00 (23.06%) 13,903,529.59 (21.79%)		12.78 *0.002*	2.74 *0.075*
missing	33.00 (1.73%) 1,274,862.27 (1.80%)		6.00 (0.48%) 166,816.45 (0.26%)			
Fat Intake/AMDR (fat3cat)						
recommended or less^{R}	1,266.00 (66.28%) 45,640,465.88 (64.60%)		782.00 (62.11%) 41,039,249.92 (64.41%)		5.77 *0.016*	0.002 *0.965*
more than recommended	644.00 (33.72%) 25.015.694.25 (35.40%)		477.00 (37.89%) 22,676,951.75 (35.59%)			
Protein Intake/AMDR (prot3cat)						
recommended or more^{R}	1,602.00 (83.83%) 61,008,629.70 (86.31%)		1,110.00 (87.96%) 57,755,135.57 (90.50%)		10.41 *0.0012*	2.62 *0.113*
less than recommended	309.00 (16.17%) 9,676,664.37 (13.69%)		152.00 (12.04%) 6,061,603.79 (9.50%)			
Iron Intake/RDA (iron2cat)						
recommended or more^{R}	1,469.00 (76.87%) 53,617,171.06 (75.85%)		1,045.00 (82.81%) 53,825,146.92 (84.34%)		16.27 *<0.0001*	5.35 *0.025*
less than recommended	442.00 (23.13%) 17,068,123.00 (24.15%)		217.00 (17.19%) 9,991,592.45 (15.66%)			
Calcium Intake/RDA (calc2cat)						
recommended or more^{R}	393.00 (20.57%) 14,092,640.99 (19.94%)		247.00 (19.57%) 11,983,668.21 (18.78%)		0.46 *0.495*	0.09 *0.763*
less than recommended	1,518.00 (79.43%) 56,592,653.07 (80.06%)		1,015.00 (80.43%) 51,833,071.16 (81.22%)			

Table 56
Bivariate Analyses of Independent Variables on Methylmercury Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30	Below Geometric Mean		At or Above Geometric Mean		χ^2 *p value unweighted*	χ^2 *p value weighted*
Selenium Intake/RDA (sele2cat)						
recommended or more^{R}	1,470.00 (76.92%) 55,410,442.98 (78.39%)		1,087.00 (86.13%) 57,234,948.83 (89.69%)		41.21 *<0.0001*	15.05 *0.0003*
less than recommended	441.00 (23.08%) 15,274,851.09 (21.61%)		175.00 (13.87%) 6,581,790.53 (10.31%)			
Reproductive Status						
Current Pregnancy (pregnant)						
pregnant	262.00 (13.71%) 2,877,024.96 (4.07%)		129.00 (10.22%) 1,965,164.13 (3.08%)			
not pregnant^{R}	1,529.00 (80.01%) 64,879,866.53 (91.79%)		1,112.00 (88.11%) 61,496,652.40 (96.36%)		49.94 *<0.0001*	7.97 *0.001*
missing	120.00 (6.28%) 2,928,402.57 (4.14%)		21.00 (1.66%) 354,922.84 (0.56%)			
Trimester of Pregnancy (tripcorr)						
not pregnant^{R}	1,649.00 (86.29%) 67,808,269.11 (95.93%)		1,133.00 (89.78%) 61851575.24 (96.92%)			
1st trimester	91.00 (4.76%) 1,189,057.76 (1.68%)		58.00 (4.60%) 802508.35 (1.26%)		15.25 *0.0016*	0.64 *0.594*
2nd trimester	100.00 (5.23%) 999,781.18 (1.41%)		32.00 (2.54%) 523,714.35 (0.82%)			
3rd trimester	71.00 (3.72%) 688,186.02 (0.97%)		39.00 (3.09%) 638,941.42 (1.00%)			
Ever Pregnant (tprg2cat)						
never pregnant^{R}	981.00 (51.33%) 31,801,576.35 (44.99%)		554.00 (43.90%) 27,763,520.63 (43.51%)		16.83 *<0.0001*	0.09 *0.769*
one or more pregnancies	930.00 (48.67%) 38,883,717.72 (55.01%)		708.00 (56.10%) 36,053,218.73 (56.49%)			
Live Births (live)						
no live births^{R}	1,153.00 (60.33%) 35,540,111.79 (50.28%)		667.00 (52.85%) 31,880,126.42 (49.96%)		17.39 *<0.0001*	0.004 *0.947*
one or more live births	758.00 (39.67%) 35,145,182.28 (49.72%)		595.00 (47.15%) 31,936,612.94 (50.04%)			
Ever Breastfed (brstfda)						
never breastfed^{R}	1,444.00 (75.56%) 49,600,141.20 (70.17%)		879.00 (69.65%) 42,354,181.09 (66.37%)		13.54 *0.0002*	1.13 *0.293*
breastfed more than one month or currently	467.00 (24.44%) 21,085,152.86 (29.83%)		383.00 (30.35%) 21,462,558.27 (33.63%)			

Table 56
Bivariate Analyses of Independent Variables on Methylmercury Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30	Below Geometric Mean		At or Above Geometric Mean		χ^2 *p value unweighted*	χ^2 *p value weighted*
Exposure-Related Attributes						
Acculturation						
Birthplace (born2cat)						
U.S.R	1,627.00 (85.14%) 64,008,759.19 (90.55%)		1,046.00 (82.88%) 56,294,937.62 (88.21%)		2.91 *0.88*	0.94 *0.337*
outside U.S.	284.00 (14.86%) 6,676,534.88 (9.45%)		216.00 (17.12%) 7,521,801.75 (11.79%)			
Years in U.S. (yrus5)						
born in U.S.R	1627.00 (85.36%) 64,008,759.19 (90.70%)		1,046.00 (82.95%) 56,294,937.62 (88.29%)		12.39 *0.002*	2.74 *0.075*
five or more years	184.00 (9.65%) 4,664,393.86 (6.61%)		168.00 (13.32%) 6,409,125.35 (10.05%)			
less than five years	95.00 (4.98%) 1,896,206.83 (2.69%)		47.00 (3.73%) 1,059,760.09 (1.66%)			
Language Spoken at Home (lang2cat)						
EnglishR	1,706.00 (89.27%) 66,529,351.01 (94.12%)		1,138.00 (90.32%) 60,231,843.35 (94.72%)		0.89 0.344	0.21 *0.651*
Other	205.00 (10.73%) 4,155,943.05 (5.88%)		122.00 (9.68%) 3,357,828.66 (5.28%)			
U.S. Citizenship (usczn2cat)						
U.S. citizenR	1,685.00 (88.22%) 66,431,518.33 (94.01%)		1,129.00 (89.46%) 60,393,753.58 (94.64%)		1.17 *0.279*	0.22 *0.640*
non-U.S. citizen	225.00 (11.78%) 4,231,907.05 (5.99%)		133.00 (10.54%) 3,422,985.79 (5.36%)			
Diet						
Seafood Eaten in Past 30 Days (smpw2cat)						
noneR	608.00 (31.82%) 20,242,379.86 (28.64%)		78.00 (6.18%) 2,628,460.93 (4.12%)		294.76 *<0.0001*	67.08 *0.0000*
any	1,303.00 (68.18%) 50,442,914.21 (71.36%)		1,184.00 (93.82%) 61,188,278.44 (95.88%)			
Fish Eaten in Past 30 Days (fish2cat)						
noneR	877.00 (45.89%) 30,759,747.06 (43.52%)		163.00 (12.92%) 6,049,992.61 (9.48%)		375.12 *<0.0001*	118.37 *0.0000*
any	1,034.00 (54.11%) 39,925,547.00 (56.48%)		1,099.00 (87.08%) 57,766,746.76 (90.52%)			

Table 56
Bivariate Analyses of Independent Variables on Methylmercury Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30	Below Geometric Mean		At or Above Geometric Mean		χ^2 *p value unweighted*	χ^2 *p value weighted*
Shellfish Eaten in Past 30 Days (shell2cat)						
none[R]	1,113.00 (58.24%) 39,932,083.11 (56.49%)		444.00 (35.18%) 23,086,556.07 (36.18%)		161.72 *<0.0001*	17.18 *0.0002*
any	798.00 (41.76%) 30,753,210.95 (43.51%)		818.00 (64.82%) 40,730,183.30 (63.82%)			
Tap Water Consumed Prior 24h (tap2kct)						
none[R]	714.00 (37.36%) 23,745,482.27 (33.59%)		415.00 (32.88%) 16,759,345.97 (26.26%)			
< 2,000 ml	868.00 (45.42%) 33,285,324.77 (47.09%)		670.00 (53.09%) 37,760,160.66 (59.17%)		18.85 *0.0003*	5.15 *0.0039*
2,000+ ml	188.00 (9.84%) 8,452,430.26 (11.96%)		107.00 (8.48%) 7,077,122.24 (11.09%)			
missing	141.00 (7.38%) 5,202,056.77 (7.36%)		70.00 (5.55%) 2,220,110.49 (3.48%)			
Alcohol Consumption						
Alcohol Consumption (retohuse)						
never, seldom drinker[R] *including 16-19 y/o*	1,205.00 (63.06%) 32,093,577.92 (45.40%)		538.00 (42.63%) 20,126,937.45 (31.54%)			
drinker	386.00 (20.20%) 20.660.784.95 (29.23%)		344.00 (27.26%) 20,009,294.62 (31.35%)		135.79 <0.0001	3.48 *0.024*
heavy drinker	255.00 (13.34%) 15,637,342.25 (22.12%)		300.00 (23.77%) 20,128,037.19 (31.54%)			
missing	65.00 (3.40%) 2,293,588.95 (3.24%)		80.00 (6.34%) 3,552,470.10 (5.57%)			
Tobacco Use						
Serum Cotinine (cot3cat)						
< 1.0 ng/ml[R]	1,407.00 (74.05%) 49,609,568.34 (70.70%)		961.00 (76.39%) 49,261,905.23 (77.33%)			
1.0 - 10.0 ng/ml	135.00 (7.11%) 3,406,027.43 (4.85%)		55.00 (4.37%) 1,844,274.19 (2.90%)		10.01 *0.007*	2.55 *0.089*
> 10.0 ng/ml	358.00 (18.84%) 17,156,220.02 (24.45%)		242.00 (19.24%) 12,594,121.21 (19.77%)			

Table 56
Bivariate Analyses of Independent Variables on Methylmercury Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30	Below Geometric Mean	At or Above Geometric Mean	χ^2 *p value unweighted*	χ^2 *p value weighted*
ETS (ETS)				
no ETS^{R}	1,415.00 (74.05%) 51,322,375.31 (72.61%)	1,002.00 (79.40%) 50,474,996.27 (79.09%)		
ETS at home or work	442.00 (23.13%) 16,219,182.51 (22.95%)	208.00 (16.48%) 10,487,687.09 (16.43%)	23.07 *<0.0001*	1.84 *0.170*
ETS at home and work	54.00 (2.83%) 3,143,736.25 (4.45%)	52.00 (4.12%) 2,854,056.01 (4.47%)		
Residence				
Tap Water Source (h2os2cat)				
public^{R}	1665.00 (87.13%) 59,607,539.62 (84.33%)	1161.00 (92.00%) 57,128,368.55 (89.52%)		
private	199.00 (10.41%) 9,648,683.80 (13.65%)	67.00 (5.31%) 4,842,851.22 (7.59%)	25.81 *<0.0001*	2.71 *0.078*
missing	47.00 (2.46%) 1,429,070.65 (2.02%)	34.00 (2.69%) 1,845,519.59 (2.89%)		
Residential Tap Water Treatment (h2ox2cat)				
yes	490.00 (25.64%) 21,607,404.37 (30.57%)	373.00 (29.56%) 23,700,830.29 (37.14%)		
no^{R}	1,378.00 (72.11%) 47,657,844.04 (67.42%)	861.00 (68.23%) 38,887,493.32 (60.94%)	5.91 *0.053*	1.19 *0.315*
missing	43.00 (2.25%) 1,420,045.65 (2.01%)	28.00 (2.22%) 1,228,415.75 (1.92%)		
Type of Residence (res3cat)				
attached or detached house^{R}	1,222.00 (63.95%) 45,821,302.96 (64.82%)	850.00 (67.35%) 43,484,667.60 (68.14%)		
mobile home or trailer	147.00 (7.69%) 5,786,760.79 (8.19%)	55.00 (4.36%) 2,615,017.01 (4.10%)	14.62 *0.0007*	3.15 *0.053*
all other types *including missing/unknown*	542.00 (28.36%) 19,077,230.32 (26.99%)	357.00 (28.29%) 17,717,054.75 (27.76%)		
Age of Residence (resb60cat)				
1960 or newer^{R}	938.00 (49.08%) 40,533,763.38 (57.34%)	657.00 (52.06%) 37,510,760.64 (58.78%)		
older than 1960	472.00 (24.70%) 16,533,334.37 (23.39%)	294.00 (23.30%) 15,558,865.73 (24.38%)	2.69 *0.260*	0.24 *0.786*
missing/unknown	501.00 (26.22%) 13,618,196.32 (19.27%)	311.00 (24.64%) 10,747,112.99 (16.84%)		

Table 56
Bivariate Analyses of Independent Variables on Methylmercury Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30	Below Geometric Mean		At or Above Geometric Mean		χ^2 *p value unweighted*	χ^2 *p value weighted*
Age of Residence (resb78cat)						
1978 or newer[R]	613.00 (32.08%) 27,813,984.59 (39.35%)		474.00 (37.56%) 27,574,064.27 (43.21%)			
older than 1978	797.00 (41.71%) 29,253,113.15 (41.39%)		477.00 (37.80%) 25,495,562.11 (39.95%)		10.29 *0.006*	0.43 *0.650*
missing/unknown	501.00 (26.22%) 13,618,196.32 (19.27%)		311.00 (24.64%) 10,747,112.99 (16.84%)			
Resident Status (resd3cat)						
own[R]	1,022.00 (53.48%) 40,079,923.43 (56.70%)		705.00 (55.86%) 37,170,383.70 (58.25%)			
rent	767.00 (40.14%) 26,463,587.74 (37.44%)		511.00 (40.49%) 24,700,307.83 (38.71%)		11.59 *0.003*	1.34 *0.273*
other *including missing*	122.00 (6.38%) 4,141,782.90 (5.86%)		46.00 (3.65%) 1,946,047.83 (3.05%)			
Years at Current Residence (re5yrcat)						
more than five years[R]	680.00 (35.58%) 25,459,430.14 (36.02%)		433.00 (34.31%) 20,434,888.55 (32.02%)			
five years or less	1,207.00 (63.16%) 44,382,216.50 (62.79%)		800.00 (63.39%) 42,073,396.44 (65.93%)		5.29 *0.071*	0.69 *0.504*
missing	24.00 (1.26%) 843,647.42 (1.19%)		29.00 (2.30%) 1,308,454.38 (2.05%)			
Household Size (hsize)						
four persons or less[R]	1,232.00 (64.47%) 53,365,258.09 (75.50%)		950.00 (75.28%) 53,088,770.29 (83.19%)			
more than four persons	679.00 (35.53%) 17,320,035.97 (24.50%)		312.00 (24.72%) 10,727,969.07 (16.81%)		41.34 *<0.0001*	5.09 *0.029*
Rooms in Residence (rm3cat)						
7+ rooms[R]	649.00 (33.96%) 26,314,597.91 (37.23%)		499.00 (39.54%) 26,301,914.90 (41.21%)			
4-6 rooms	1,075.00 (56.25%) 38,280,251.55 (54.16%)		616.00 (48.81%) 30,819,881.04 (48.29%)		17.32 *0.0006*	0.38 *0.763*
1-3 rooms	145.00 (7.59%) 4,887,472.26 (6.91%)		118.00 (9.35%) 5,287,700.85 (8.29%)			
missing	42.00 (2.20%) 1,202,972.35 (1.70%)		29.00 (2.30%) 1,407,242.57 (2.21%)			

Table 56
Bivariate Analyses of Independent Variables on Methylmercury Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30	Below Geometric Mean		At or Above Geometric Mean		χ^2 *p value unweighted*	χ^2 *p value weighted*
Occupation						
Current Occupation (cocc2cat)						
not working[R]	876.00 (45.84%) 25,214,830.94 (35.67%)		448.00 (35.50%) 16,958,126.63 (26.57%)			
management, professional & sales	666.00 (34.85%) 32,727,760.81 (46.30%)		577.00 (45.72%) 35,031,130.92 (54.89%)		42.51 *<0.0001*	1.68 *0.198*
services & goods	369.00 (19.31%) 12,742,702.31 (18.03%)		237.00 (18.78%) 11,827,481.81 (18.53%)			
Time in Current Employment (cjt)						
not working[R]	876.00 (45.84%) 25,214,830.94 (35.67%)		448.00 (35.50%) 16,958,126.63 (26.57%)			
less than five years	857.00 (44.85%) 34,136,542.67 (48.29%)		577.00 (45.72%) 33,105,097.05 (51.88%)		71.67 *<0.0001*	1.96 *0.153*
more than five years	178.00 (9.31%) 11,333,920.45 (16.03%)		237.00 (18.78%) 13,753,515.68 (21.55%)			
Total Hours Worked Prior Week (hrwk)						
not employed[R]	910.00 (47.67%) 27,405,434.12 (38.85%)		471.00 (37.32%) 18,402,603.91 (28.84%)			
less than 35 hours	432.00 (22.63%) 14,877,373.97 (21.09%)		304.00 (24.09%) 18,490,059.83 (28.97%)		37.43 *<0.0001*	1.56 *0.222*
35+ hours	567.00 (29.70%) 28,257,445.38 (40.06%)		487.00 (38.59%) 26,924,075.63 (42.19%)			
Longest Held Occupation (locc2cat)						
not applicable[R]	937.00 (49.03%) 34,396,695.60 (48.66%)		625.00 (49.52%) 29,720,660.91 (46.57%)			
management, professional & sales	528.00 (27.63%) 21,722,891.47 (30.73%)		375.00 (29.71%) 19,692,294.68 (30.86%)		3.46 *0.177*	0.16 *0.852*
services & goods	446.00 (23.34%) 14,565,706.99 (20.61%)		262.00 (20.76%) 14,403,783.78 (22.57%)			
Time in Longest Employment (ljt)						
not applicable[R]	937.00 (49.03%) 34,396,695.60 (48.66%)		625.00 (49.52%) 29,720,660.91 (46.57%)			
less than five years	662.00 (34.64%) 21.201.670.86 (29.99%)		335.00 (26.55%) 13,340,597.99 (20.90%)		38.60 *<0.0001*	2.08 *0.136*
five or more years	312.00 (16.33%) 15,086,927.61 (21.34%)		302.00 (23.93%) 20,755,480.47 (32.52%)			

Table 56
Bivariate Analyses of Independent Variables on Methylmercury Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30	Below Geometric Mean		At or Above Geometric Mean		χ^2 *p value unweighted*	χ^2 *p value weighted*
Work History (wkcp)						
never employed[R]	297.00 (15.54%) 5,919,953.87 (8.38%)		111.00 (8.80%) 2,318,856.93 (3.63%)			
currently employed	640.00 (33.49%) 28,476,741.73 (40.29%)		514.00 (40.73%) 27,401,803.98 (42.94%)		44.59 *<0.0001*	2.21 *0.100*
employed in the past but not currently	579.00 (30.30%) 19,294,877.07 (27.30%)		337.00 (26.70%) 14,639,269.71 (22.94%)			
employed now and in the past	395.00 (20.67%) 16,993,721.39 (24.04%)		300.00 (23.77%) 19,456,808.75 (30.49%)			
Socioeconomic Factors						
Education						
Highest Education (educ2)						
high school diploma, GED or higher[R]	1,083.00 (56.67%) 52,397,693.39 (74.13%)		954.00 (75.65%) 54,509,467.95 (85.51%)		119.13 *<0.0001*	6.25 *0.02*
less than high school diploma	828.00 (43.33%) 18,287,600.67 (25.87%)		307.00 (24.35%) 9,240,132.63 (4.49%)			
Employment						
Employment Status (emp3cat)						
employed	1,039.00 (54.43%) 45,610,187.23 (64.54%)		814.00 (64.50%) 46,858,612.73 (73.43%)		31.75 *<0.0001*	2.27 *0.139*
not employed[R]	870.00 (45.57%) 25,063,887.32 (35.46%)		448.00 (35.50%) 16,958,126.63 (26.57%)			
Reason for Unemployment (unem2cat)						
working[R]	1,039.00 (54.37%) 45,610,187.23 (64.53%)		814.00 (64.50%) 46,858,612.73 (73.43%)			
voluntary unemployment	600.00 (31.40%) 16,867,871.13 (23.86%)		324.00 (25.67%) 11,397,150.54 (17.86%)		36.18 *<0.0001*	0.77 *0.516*
involuntary unemployment	196.00 (10.26%) 5,956,503.95 (8.43%)		99.00 (7.84%) 4,306,610.06 (6.75%)			
missing	76.00 (3.98%) 2,250,731.76 (3.18%)		25.00 (1.98%) 1,254,366.03 (1.97%)			
Income						
U.S. Poverty Threshold (pov2cat)						
more than 1.00[R]	1,287.00 (67.35%) 54,471,372.99 (77.06%)		940.00 (74.48%) 49,482,250.20 (77.54%)		26.16 *<0.0001*	1.28 *0.287*
1.00 or less	499.00 (26.11%) 13,125,723.04 (18.57%)		231.00 (18.30%) 9,461,474.43 (14.83%)			
missing	125.00 (6.54%) 3,088,198.03 (4.37%)		91.00 (7.21%) 4.873.014.74 (7.64%)			

Table 56
Bivariate Analyses of Independent Variables on Methylmercury Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30	Below Geometric Mean		At or Above Geometric Mean		χ^2 *p value unweighted*	χ^2 *p value weighted*
Marital Status						
Marital Status (marr3cat)						
married or living with partner	683.00 (35.74%) 33,354,082.47 (47.19%)		515.00 (40.81%) 28,446,565.78 (44.58%)			
widowed, divorced or separated	140.00 (7.33%) 6,588,677.95 (9.32%)		121.00 (9.59%) 7,765,274.43 (12.17%)		24.17 *<0.0001*	0.68 *0.569*
never married^{R}	1,050.00 (54.95%) 28,651,239.71 (40.53%)		587.00 (46.51%) 24,841,711.78 (38.93%)			
missing	38.00 (1.99%) 2,091,293.94 (2.96%)		39.00 (3.09%) 2,763,187.37 (4.33%)			
Race-Ethnicity						
Race-Ethnicity (race5cat)						
Non-Hispanic White^{R} $n_{unweighted}$ = 1493 $n_{weighted}$ = 97,887,544.16	881.00 (46.10%) 51,861,986.93 (73.37%)		612.00 (48.49%) 46,025,557.23 (72.12%)			
Non-Hispanic Black $n_{unweighted}$ = 623 $n_{weighted}$ = 12,747,178.37	353.00 (18.47%) 6,195,923.44 (8.77%)		270.00 (21.39%) 6,551,254.92 (10.27%)			
Mexican American $n_{unweighted}$ = 745 $n_{weighted}$ = 8,670,575.80	508.00 (26.58%) 5,459,501.32 (7.72%)		237.00 (18.78%) 3,211,074.49 (5.03%)		28.86 *<0.0001*	2.45 *0.059*
Other Hispanic $n_{unweighted}$ = 178 $n_{weighted}$ = 7,525,992.22	98.00 (5.13%) 3,709,893.41 (5.25%)		80.00 (96.34%) 3,816,098.81 (5.98%)			
Asian, Native American, Pacific Islander & Multi-Racial $n_{unweighted}$ = 134 $n_{weighted}$ = 7,670,742.88	71.00 (3.72%) 3,457,988.97 (4.89%)		63.00 (4.99%) 4,212,753.91 (6.60%)			
Race-Ethnicity/Hispanic Grouping (race4cat)						
Non-Hispanic White^{R} $n_{unweighted}$ = 1493 $n_{weighted}$ = 97,887,544.16	881.00 (46.10%) 51,861,986.93 (73.37%)		612.00 (48.49%) 46,025,557.23 (72.12%)			
Non-Hispanic Black $n_{unweighted}$ = 623 $n_{weighted}$ = 12,747,178.37	353.00 (18.47%) 6,195,923.44 (8.77%)		270.00 (21.39%) 6,551,254.92 (10.27%)		18.52 *0.0003*	0.55 *0.650*
Hispanic $n_{unweighted}$ = 923 $n_{weighted}$ = 16,196,568.02	606.00 (31.71%) 9,169,394.73 (12.97%)		317.00 (25.12%) 7,027,173.30 (11.01%)			
Asian, Native American, Pacific Islander & Multi-Racial $n_{unweighted}$ = 134 $n_{weighted}$ = 7,670,742.88	71.00 (3.72%) 3,457,988.97 (4.89%)		63.00 (4.99%) 4,212,753.91 (6.60%)			

Table 57
Summary of Chi-Square and *p* Values of Weighted Independent Variables on Methylmercury (1999-2004)

Variables *p < 0.20*	*p* value	χ^2 *weighted*	Variables *p > 0.20*	*p* value	χ^2 *weighted*
Fish in Past 30 Days (fish2cat)	*0.0000*	118.37	Charleson Co-Morbidity Scale (CCMS3cat)	*0.202*	1.66
Seafood in Past 30 Days [1] (smpw2cat)	*0.0000*	67.08	Total Hours Worked Prior Week (hrwk)	*0.222*	1.56
Shellfish in Past 30 Days (shell2cat)	*0.0002*	17.18	Resident Status (resd3cat)	*0.273*	1.34
Selenium Intake/RDA (sele2cat)	*0.0003*	15.05	U.S. Poverty Threshold (pov2cat)	*0.287*	1.28
Age (age4cat)	*0.0005*	7.11	Ever Breastfed (brstfda)	*0.293*	1.13
Current Pregnancy (pregnant)	*0.001*	7.97	Iron Deficiency (FeD2cat)	*0.314*	1.04
Perceived Health Status (huq2cat)	*0.003*	9.87	Residential Tap Water Treatment (h2ox2cat)	*0.315*	1.19
Tap Water Consumed 24h (tap2kct)	*0.0039*	5.15	Birthplace (born2cat)	*0.337*	0.94
Health Insurance (hi2cat)	*0.005*	4.89	Iron Deficiency and Treatment (FeDTx)	*0.431*	0.94
Highest Education (educ2)	*0.016*	6.25	Years at Current Residence (re5yrcat)	*0.504*	0.69
Alcohol Consumption (retohuse)	*0.024*	3.48	Reason for Unemployment (unem2cat)	*0.516*	0.77
Iron Intake/RDA [1] (iron2cat)	*0.025*	5.35	Marital Status (marr3cat)	*0.569*	0.68
Household Size (hsize)	*0.029*	5.09	Trimester of Pregnancy [1] (tripcorr)	*0.594*	0.64
Type of Residence (res3cat)	*0.053*	3.15	U.S. Citizenship (usczn2cat)	*0.64*	0.22
Race-Ethnicity [1] (race5cat)	*0.059*	2.45	Treatment for Iron Deficiency past 3 mo (FeTx2cat)	*0.644*	0.21
Body Mass Index (bmi30cat)	*0.075*	2.74	Race-Ethnicity/Hispanic Grouping (race4cat)	*0.650*	0.55
Years in U.S. (yrus5)	*0.075*	2.74	Age of Residence (resb78cat)	*0.65*	0.43
Tap Water Source [1] (h2os2cat)	*0.078*	2.71	Language Spoken at Home (lang2cat)	*0.651*	0.21
Serum Cotinine (cot3cat)	*0.089*	2.55	Source of Healthcare (hscre)	*0.721*	0.45
Work History [1] (wkcp)	*0.100*	2.21	Rooms in Residence (rm3cat)	*0.763*	0.38
Protein Intake/AMDR (prot3cat)	*0.113*	2.62	Calcium Intake/RDA (calc2cat)	*0.763*	0.09
Time in Longest Employment (ljt)	*0.136*	2.08	Ever Pregnant [1] (tprg2cat)	*0.769*	0.09
Employment Status (emp3cat)	*0.139*	2.27	Regular Source of Healthcare (hp2cat)	*0.774*	0.08
Time in Current Employment [1] (cjt)	*0.153*	1.96	Age of Residence (resb60cat)	*0.786*	0.24
Environmental Tobacco Smoke [1] (ETS)	*0.170*	1.84	Longest Held Occupation (locc2cat)	*0.852*	0.16
Food Security [1] (food2cat)	*0.177*	1.80	Live Births (live)	*0.947*	0.00
Current Occupation [1] (cocc2cat)	*0.198*	1.68	Fat Intake/AMDR (fat3cat)	*0.965*	0.00

[1] variable dropped due to low cell size or too similar to other variables

Table 58
Stepwise Logistic Regression Analyses of Methylmercury
(1999-2004)

Variable Name *p < 0.20*	df	-2LL Wald F	Difference	df	*p* value
Initial Regression	34	858.44			
1 Highest Education (educ2)	33	855.70	2.74	1	*>0.05* *drop*
2 Years in U.S. (yrus5)	31	855.14	0.56	2	*>0.20* *drop*
3 Protein Intake/AMDR (prot3cat)	30	854.69	0.45	1	*>0.20* *drop*
4 Race-Ethnicity/Hispanic Grouping (race4cat)	27	840.81	13.88	3	*<0.01* *keep*
5 Health Insurance (hi2cat)	27	849.95	4.74	3	*>0.10* *drop*
6 Serum Cotinine (cot3cat)	25	845.30	4.65	2	*>0.05* *drop*
7 Alcohol Consumption (retohuse)	22	819.83	25.47	3	*<0.001* *keep*
8 Body Mass Index (bmi30cat)	23	836.15	9.15	2	*<0.02* *keep*
9 Tap Water Consumed 24h (tap2kct)	22	820.64	24.66	3	*<0.001* *keep*
10 Type of Residence (res3cat)	23	823.17	22.13	2	*<0.001* *keep*
11 Household Size (hsize)	24	839.15	6.15	1	*<0.02* *keep*
12 Time in Longest Employment (ljt)	23	813.57	31.73	2	*<0.001* *keep*
13 Perceived Health Status (huq2cat)	24	833.05	12.25	1	*<0.001* *keep*
14 Age (age4cat)	22	819.52	25.78	3	*<0.001* *keep*
15 Current Pregnancy (pregnant)	23	827.90	17.40	2	*<0.001* *keep*
16 Selenium Intake/RDA (sele2cat)	24	827.26	180.04	1	*<0.001* *keep*
17 Shellfish in Past 30 Days (shell2cat)	24	803.69	41.61	1	*<0.001* *keep*
18 Fish in Past 30 Days (fish2cat)	24	483.99	361.31	1	*<0.001* *keep*

Table 59
Best-Fit Logistic Regression Methylmercury Model *with no interactions*
(1999-2004)

Variable Name	df	-2LL Wald F	R^2		*p* value
Best Fit Regression	25	845.30	*0.2339*		
Fish in Past 30 Days (fish2cat)	1	49.57			*0.0000*
Shellfish in Past 30 Days (shell2cat)	1	7.86			*0.0075*
Perceived Health Status (huq2cat)	1	5.64			*0.0219*
Type of Residence (res3cat)	2	3.49			*0.0392*
Age (age4cat)	3	3.03			*0.0393*
Current Pregnancy (pregnant)	2	2.41			*0.1011*
Time in Longest Employment (ljt)	2	2.41			*0.1012*
Selenium Intake/RDA (sele2cat)	1	2.51			*0.1201*
Tap Water Consumed 24h (tap2kct)	3	1.66			*0.1898*
Household Size (hsize)	1	1.65			*0.2063*
Body Mass Index (bmi30cat)	2	1.22			*0.3047*
Alcohol Consumption (retohuse)	3	1.23			*0.3090*
Race-Ethnicity/Hispanic Grouping (race4cat)	3	0.53			*0.6630*

[1] in ascending order by p value

Table 60
Variance Inflation Factor Test for Collinearity Among Independent Variables using the Best-Fit Logistic Regression Methylmercury Model (1999-2004)

	df	sum of squares	mean square	F value	pr>F	
Best Fit Methylmercury Model *with no interactions*	13	475.83	36.602	236.83	*<0.0001*	

Variable Name	df	parameter estimate	std error	t value	pr>t	VIF
Intercept	1	0.276	0.030	9.06	*<0.0001*	0.0000
Age (age4cat)	1	0.049	*0.003*	18.60	*<0.0001*	1.3457
Perceived Health Status (huq2cat)	1	-0.062	*0.009*	-7.15	*<0.0001*	1.0427
Body Mass Index (bmi30cat)	1	-0.022	*0.006*	-3.83	*0.0001*	1.0467
Selenium Intake/RDA (sele2cat)	1	0.005	*0.005*	0.99	*0.3220*	1.2023
Current Pregnancy (pregnant)	1	-0.120	*0.005*	-24.66	*<0.0001*	1.3146
Fish in Past 30 Days (fish2cat)	1	0.104	*0.007*	15.91	*<0.0001*	1.5611
Shellfish in Past 30 Days (shell2cat)	1	0.065	*0.007*	9.75	*<0.0001*	1.4184
Tap Water Consumed 24h (tap2kct)	1	0.004	*0.003*	1.2	*0.2311*	1.1557
Alcohol Consumption (retohuse)	1	0.003	*0.003*	0.83	*0.4059*	1.3577
Type of Residence (res3cat)	1	0.006	*0.003*	2.17	*0.0303*	1.0580
Household Size (hsize)	1	-0.026	*0.006*	-4.55	*<0.0001*	1.1123
Time in Longest Employment (ljt)	1	0.007	*0.003*	1.98	*0.0481*	1.0432
Race-Ethnicity/Hispanic Grouping (race4cat)	1	0.012	*0.0029*	4.16	*<0.0001*	1.1297

Table 61
Statistical Significance of Interactions Between Independent Variables using the Best-Fit Logistic Regression Methylmercury Model (1999-2004)

Independent Variables	Age (age4cat)	Perceived Health Status (huq2cat)	Body Mass Index (bmi30cat)	Selenium Intake/RDA (sele2cat)	Current Pregnancy (pregnant)	Fish in Past 30 Days (fish2cat)	Shellfish in Past 30 Days (shell2cat)	Tap Water Consumed 24h (tap2kct)	Alcohol Consumption (retohuse)	Type of Residence (res3cat)	Household Size (hsize)	Time in Longest Employment (ljt)	Race-Ethnicity Hispanic Grouping (race4cat)
Age (age4cat)													
Perceived Health Status (huq2cat)	*<0.001*												
Body Mass Index (bmi30cat)	*op*	*<0.05*											
Selenium Intake/RDA (sele2cat)	*ns*	*ns*	*ns*										
Current Pregnancy (pregnant)	*op*	*ns*	*op*	*<0.01*									
Fish in Past 30 Days (fish2cat)	*<0.001*	*ns*	*<0.001*	*<0.001*	*ns*								
Shellfish in Past 30 Days (shell2cat)	*<0.001*	*ns*	*op*	*<0.01*	*<0.05*	*ns*							
Tap Water Consumed 24h (tap2kct)	*<0.05*	*ns*	*op*	*<0.001*	*<0.05*	*<0.001*	*<0.01*						
Alcohol Consumption (retohuse)	*op*	*ns*	*op*	*ns*	*op*	*<0.01*	*<0.001*	*<0.001*					
Type of Residence (res3cat)	*ns*	*ns*	*op*	*<0.01*	*ns*	*<0.001*	*ns*	*<0.001*	*op*				
Household Size (hsize)	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*	*<0.001*	*<0.01*	*<0.01*	*ns*			
Time in Longest Employment (ljt)	*op*	*ns*	*op*	*ns*	*op*	*<0.001*	*<0.001*	*<0.001*	*<0.001*	*<0.01*	*<0.001*		
Race-Ethnicity/Hispanic Grouping (race4cat)	*<0.001*	*ns*	*op*	*ns*	*op*	*<0.001*	*<0.001*	*<0.001*	*op*	*<0.001*	*<0.05*	*<0.001*	

op = over parameterized unable to calculate
ns = not statistically significant p>0.05

Table 62
Odds Ratios and Confidence Intervals for Best-Fit Logistic Regression Methylmercury Model
with no interactions (1999-2004)

Variable Names	df	-2LL Wald F	*p* value	Odds Ratios	Confidence Intervals
Susceptibility-Related Attributes					
Age (age4cat)	3	3.03	*0.0393*		
16-19[R]				1.00	*ns*
20-29				2.32	1.30 - 4.14 %
30-39				2.14	1.17 - 3.93 %
40-49				2.11	1.06 - 4.22 %
Health Status					
Perceived Health Status (huq2cat)	1	5.64	*0.0219*		
excellent, very good, good[R]				1.00	*ns*
fair, poor				0.55	0.33 - 0.91 %
Nutritional Status					
Body Mass Index (bmi30cat)	2	1.22	*0.3047*		
<30.0[R] underweight, normal, overweight				1.00	*ns*
30.0+ obese				0.83	0.51 - 1.34 %
missing				0.24	0.03 - 1.97 %
Selenium Intake/RDA (sele2cat)	1	2.51	*0.1201*		
recommended or more[R]				1.00	*ns*
less than recommended				0.60	0.32 - 1.15 %
Reproductive Status					
Current Pregnancy (pregnant)	2	2.41	*0.1011*		
pregnant				0.65	0.37 - 1.11 %
not pregnant[R]				1.00	*ns*
missing				0.24	0.03 - 2.06 %
Exposure-Related Attributes					
Diet					
Fish in Past 30 Days (fish2cat)	1	49.57	*0.0000*		
none[R]				1.00	*ns*
any				6.63	3.86 - 11.38 %
Shellfish in Past 30 Days (shell2cat)	1	7.86	*0.0075*		
none[R]				1.00	*ns*
any				1.73	1.17 - 2.57 %
Tap Water Consumed 24h (tap2kct)	3	1.66	*0.1898*		
none[R]				1.00	*ns*
< 2,000 ml				1.35	0.89 - 2.05 %
2,000+ ml				0.86	0.37 - 1.97 %
missing				0.70	0.34 - 1.43 %
Alcohol Consumption					
Alcohol Consumption (retohuse)	3	1.23	*0.3090*		
never, seldom drinker[R] *including 16-19 y/o*				1.00	*ns*
drinker				0.81	0.47 - 1.39 %
heavy drinker				1.29	0.70 - 2.39 %
missing				1.76	0.77 - 4.02 %
Residence					
Type of Residence (res3cat)	2	3.49	*0.0392*		
attached or detached house[R]				1.00	*ns*
mobile home or trailer				0.44	0.22-0.86 %
all other types *including missing/unknown*				1.00	0.62 - 1.61 %
Household Size (hsize)	1	1.65	*0.2063*		
four persons or less[R]				1.00	*ns*
more than four persons				0.76	0.49 - 1.17 %
Occupation					
Time in Longest Employment (ljt)	2	2.41	*0.1012*		
not applicable[R]				1.00	*ns*
less than five years				0.62	0.38 - 1.03 %
five or more years				1.26	0.78 - 2.03 %

[R] = referent group
ns = not significant

Table 62
Odds Ratios and Confidence Intervals for Best-Fit Logistic Regression Methylmercury Model
with no interactions (1999-2004)

Variable Names	df	-2LL Wald F	*p* value	Odds Ratios	Confidence Intervals
Race-Ethnicity					
Race-Ethnicity/Hispanic Grouping (race4cat)	3	0.53	*0.6630*		
Non-Hispanic White[R]				1.00	*ns*
Non-Hispanic Black				1.42	0.74 - 2.72 %
Hispanic				1.26	0.77 - 2.08 %
Asian, Native American, Pacific Islander & Multi-Racial				1.44	0.41 - 4.97 %

[R] = referent group
ns = not significant

Table 63
Bivariate Analyses of Independent Variables on Sum of PCBs Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30	Below Geometric Mean		At or Above Geometric Mean		χ^2 *p value unweighted*	χ^2 *p value weighted*
Susceptibility-Related Attributes						
Age (age4cat)						
16-19^{R} $n_{unweighted}$ = 1,085 $n_{weighted}$ = 18,510,468.72	730.00 (48.89%) 12,591,232.42 (27.97%)		355.00 (21.13%) 5,919,236.31 (6.61%)			
20-29 $n_{unweighted}$ = 884 $n_{weighted}$ = 45,347,514.91	495.00 (33.15%) 21,467,627.54 (47.70%)		389.00 (23.15%) 23,879,887.37 (26.68%)		576.23 *<0.0001*	26.44 *0.0000*
30-39 $n_{unweighted}$ =702 $n_{weighted}$ = 36,357,836.50	229.00 (15.34%) 9,747,314.99 (21.66%)		473.00 (28.15%) 26,610,521.52 (29.74%)			
40-49 $n_{unweighted}$ =502 $n_{weighted}$ = 34,286,213.30	39.00 (2.61%) 1,203,971.63 (2.67%)		463.00 (27.56%) 33,082,241.67 (36.97%)			
Health Status						
Perceived Health Status (huq2cat)						
excellent, very good, good^{R}	1,351.00 (90.49%) 41,856,362.84 (92.99%)		1,489.00 (88.68%) 82,148,882.26 (91.83%)		2.75 *0.097*	0.59 *0.443*
fair, poor	142.00 (9.51%) 3,153,783.73 (7.01%)		190.00 (11.32%) 7,312,095.87 (8.17%)			
Charleson Co-Morbidity Scale (CCMS3cat)						
none^{R}	1,304.00 (87.34%) 38,384,929.48 (85.28%)		1,510.00 (89.88%) 79,872,091.95 (89.25%)			
one co-morbidity	167.00 (11.19%) 5,453,663.37 (12.12%)		136.00 (8.10%) 7,693,069.95 (8.60%)		9.84 *0.007*	0.47 *0.626*
more than one co-morbidity	* (0.00%) 1,171,553.72 (2.60%)		34.00 (2.02%) 1,926,724.96 (2.15%)			
Iron Deficiency (FeD2cat)						
within normal limits^{R}	1,245.00 (83.39%) 40,808,918.84 (90.67%)		1,479.00 (88.04%) 82,027,839.78 (91.66%)			
iron deficient $n_{unweighted}$ = 449 $n_{weighted}$ = 11,665,274.81	248.00 (16.61%) 4,201,227.73 (9.33%)		201.00 (11.96%) 7,464,047.08 (8.34%)		14.05 *0.0002*	0.43 *0.516*
Treatment for Iron Deficiency past 3 mo (FeTx2cat)						
yes	83.00 (5.56%) 1,244,972.93 (2.77%)		88.00 (5.24%) 3,901,323.04 (4.36%)		0.16 *0.692*	2.89 *0.096*
no^{R}	1,410.00 (94.44%) 43,765,173.64 (97.23%)		1,591.00 (94.76%) 85,576,984.75 (95.64%)			

Table 63
Bivariate Analyses of Independent Variables on Sum of PCBs Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30	Below Geometric Mean		At or Above Geometric Mean		χ^2 *p value unweighted*	χ^2 *p value weighted*
Iron Deficiency and Treatment (FeDTx)						
normal/no treatment^{R}	1,195.00 (80.04%) 40,135,834.13 (89.17%)		1,413.00 (84.16%) 79,306,864.55 (88.63%)			
normal w/treatment	50.00 (3.35%) 673,084.71 (1.50%)		65.00 (3.87%) 2,707,396.16 (3.03%)		14.59 *0.002*	1.49 *0.228*
deficient w/treatment	33.00 (2.21%) 571,888.22 (1.27%)		* (0.00%) 1,193,926.89 (1.33%)			
deficient/no treatment	215.00 (14.40%) 3,629,339.51 (8.06%)		178.00 (10.60%) 6,270,120.20 (7.01%)			
Health Insurance (hi2cat)						
private^{R}	870.00 (58.27%) 30,234,802.03 (67.17%)		1,172.00 (69.76%) 69,897,976.44 (78.11%)			
public	276.00 (18.49%) 3,894,084.20 (8.65%)		163.00 (9.70%) 5,897,335.88 (6.59%)		66.31 *<0.0001*	3.44 *0.025*
none	303.00 (20.29%) 9,622,947.81 (21.38%)		316.00 (18.81%) 12,139,856.55 (13.57%)			
missing	44.00 (2.95%) 1,258,312.52 (2.80%)		29.00 (1.73%) 1,556,717.99 (1.74%)			
Regular Source of Healthcare (hp2cat)						
yes^{R}	1,245.00 (83.39%) 37,055,253.83 (82.33%)		1,428.00 (85.00%) 78,403,028.49 (87.61%)		1.54 *0.214*	1.51 *0.226*
no	248.00 (16.61%) 7,954,892.73 (17.67%)		252.00 (15.00%) 11,088,858.37 (12.39%)			
Source of Healthcare (hcsre)						
healthcare provider^{R}	785.00 (52.58%) 25,696,440.66 (57.09%)		1,022.00 (60.83%) 59,465,399.34 (66.45%)			
clinic	357.00 (23.91%) 7,778,732.74 (17.28%)		319.00 (18.99%) 15,989,364.13 (17.87%)		36.72 *<0.0001*	2.09 *0.114*
ER or none	314.00 (21.03%) 9,179,047.38 (20.39%)		328.00 (19.52%) 13,783,609.38 (15.40%)			
missing	37.00 (2.48%) 2,355,925.79 (5.23%)		11.00 (0.65%) 253,514.02 (0.28%)			

Table 63
Bivariate Analyses of Independent Variables on Sum of PCBs Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30	Below Geometric Mean		At or Above Geometric Mean		χ^2 *p value unweighted*	χ^2 *p value weighted*
Nutritional Status						
Food Security (food2cat)						
food secure^{R}	1,155.00 (77.36%) 35,515,569.91 (78.91%)		1,417.00 (84.35%) 78,528,342.75 (87.75%)			
food insecure	261.00 (17.48%) 6,952,393.71 (15.45%)		198.00 (11.79%) 7,282,128.34 (8.14%)		25.42 *<0.0001*	3.39 *0.043*
missing	77.00 (5.16%) 2,542,182.95 (5.65%)		65.00 (3.87%) 3,681,415.78 (4.11%)			
Body Mass Index (bmi30cat)						
<30.0^{R} underweight normal overweight	1,099.00 (73.61%) 33,131,937.89 (73.61%)		1,258.00 (74.88%) 69,711,959.03 (77.90%)			
30.0+ obese	379.00 (25.39%) 11,702,893.48 (26.00%)		398.00 (23.69%) 18,513,564.32 (20.69%)		2.25 *0.324*	2.49 *0.094*
missing	15.00 (1.00%) 175,315.20 (0.39%)		24.00 (1.43%) 1,266,363.51 (1.42%)			
Fat Intake/AMDR (fat3cat)						
recommended or less^{R}	982.00 (65.82%) 28,732,213.69 (63.88%)		1,066.00 (63.57%) 57,947,502.11 (64.82%)			
more than recommended	510.00 (39.18%) 16,248,798.94 (36.12%)		611.00 (36.92%) 31,443,847.06 (35.18%)		1.75 *0.186*	0.03 *0.852*
Protein Intake/AMDR (prot3cat)						
recommended or more^{R}	1,256.00 (84.13%) 39,216,304.71 (87.13%)		1,456.00 (86.67%) 79,547,460.56 (88.89%)			
less than recommended	237.00 (15.87%) 5,793,841.86 (12.87%)		224.00 (13.33%) 9,944,426.30 (11.11%)		4.11 *0.043*	0.46 *0.502*
Iron Intake/RDA (iron2cat)						
recommended or more^{R}	1,167.00 (78.16%) 33,365,800.23 (74.13%)		1,347.00 (80.18%) 74,076,517.74 (82.77%)			
less than recommended	326.00 (21.84%) 11,644,346.33 (25.87%)		333.00 (19.82%) 15,415,369.12 (17.23%)		1.95 *0.163*	6.19 *0.017*
Calcium Intake/RDA (calc2cat)						
recommended or more^{R}	310.00 (20.76%) 9,569,989.07 (21.26%)		330.00 (19.64%) 16,506,320.13 (18.44%)			
less than recommended	1,183.00 (79.24%) 35,440,157.50 (78.74%)		1,350.00 (80.36%) 72,985,566.73 (81.56%)		0.62 *0.432*	0.57 *0.454*

Table 63
Bivariate Analyses of Independent Variables on Sum of PCBs Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30	Below Geometric Mean		At or Above Geometric Mean		χ^2 *p value unweighted*	χ^2 *p value weighted*
Selenium Intake/RDA (sele2cat)						
recommended or more^{R}	1,176.00 (78.77%) 35,992,279.96 (79.96%)		1,381.00 (82.20%) 76,653,111.85 (85.55%)		5.96 *0.015*	2.79 *0.102*
less than recommended	317.00 (21.23%) 9,017,866.61 (20.04%)		299.00 (17.80%) 12,838,775.01 (14.35%)			
Reproductive Status						
Current Pregnancy (pregnant)						
pregnant	235.00 (15.74%) 2,428,211.72 (5.39%)		156.00 (9.29%) 2,413,977.37 (2.70%)			
not pregnant^{R}	1,251.00 (83.79%) 42,277,463.15 (93.93%)		1,390.00 (82.74%) 84,099,055.79 (93.97%)		127.09 *<0.0001*	10.55 *0.0002*
missing	7.00 (0.47%) 304,471.69 (0.68%)		134.00 (7.98%) 2,978,853.71 (3.33%)			
Trimester of Pregnancy (tripcorr)						
not pregnant^{R}	1,258.00 (84.26%) 42,581,934.85 (94.61%)		1,523.00 (90.71%) 87,077,909.50 (97.30%)			
1st trimester	76.00 (5.09%) 870,852.45 (1.93%)		73.00 (4.35%) 1,120,713.66 (1.25%)		38.67 *<0.0001*	3.40 *0.026*
2nd trimester	85.00 (5.69%) 825,252.95 (1.83%)		47.00 (2.80%) 698,242.58 (0.78%)			
3rd trimester	74.00 (4.96%) 732,106.32 (1.63%)		36.00 (2.14%) 595,021.13 (0.66%)			
Ever Pregnant (tprg2cat)						
never pregnant^{R}	848.00 (56.80%) 26,236,692.94 (58.29%)		687.00 (40.89%) 33,328,404.04 (37.24%)		80.08 *<0.0001*	17.10 *0.0002*
one or more pregnancies	645.00 (43.20%) 18,773,453.63 (41.71%)		993.00 (59.11%) 56,163,482.82 (62.76%)			
Live Births (live)						
no live births^{R}	1,002.00 (67.11%) 29,467,862.71 (65.47%)		818.00 (48.69%) 37,952,375.50 (42.41%)		109.69 *<0.0001*	19.71 *0.0001*
one or more live births	491.00 (32.89%) 15,542,283.85 (34.53%)		862.00 (51.31%) 51,539,511.36 (57.59%)			
Ever Breastfed (brstfda)						
never breastfed^{R}	1,145.00 (76.69%) 32,986,168.84 (73.29%)		1,178.00 (70.12%) 58,968,153.46 (65.89%)		17.41 *<0.0001*	2.31 *0.136*
breastfed more than one month or currently	348.00 (23.31%) 12,023,977.72 (26.71%)		502.00 (29.88%) 30,523,733.41 (34.11%)			

Table 63
Bivariate Analyses of Independent Variables on Sum of PCBs Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30	Below Geometric Mean		At or Above Geometric Mean		χ^2 *p value unweighted*	χ^2 *p value weighted*
Exposure-Related Attributes						
Acculturation						
Birthplace (born2cat)						
U.S.[R]	1,224.00 (81.98%) 38,922,414.29 (86.47%)		1,449.00 (86.25%) 81,381,282.52 (90.94%)		10.84 *0.001*	3.24 *0.079*
outside U.S.	269.00 (18.02%) 6,087,732.28 (13.53%)		231.00 (13.75%) 8,110,604.35 (9.06%)			
Years in U.S. (yrus5)						
born in U.S.[R]	1,224.00 (82.15%) 38,922,414.29 (86.63%)		1,449.00 (86.40%) 81,381,282.52 (91.03%)		13.75 *0.001*	3.15 *0.053*
five or more years	181.00 (12.15%) 4,358,326.80 (9.70%)		171.00 (10.20%) 6,715,192.41 (7.51%)			
les than five years	85.00 (5.70%) 1,649,779.74 (3.67%)		57.00 (3.40%) 1,306,187.17 (1.46%)			
Language Spoken at Home (lang2cat)						
English[R]	1,306.00 (87.47%) 40,964,135.78 (91.01%)		1,538.00 (91.66%) 85,797,058.59 (95.12%)		14.94 *0.0001*	11.75 *0.0013*
Other	187.00 (12.53%) 4,046,010.79 (8.99%)		140.00 (8.34%) 3,467,760.92 (3.88%)			
U.S. Citizenship (usczn2cat)						
U.S. citizen[R]	1,282.00 (85.87%) 41,083,030.09 (91.28%)		1,532.00 (91.24%) 85,742,241.82 (95.83%)		22.82 *<0.0001*	10.29 *0.0025*
non-U.S. citizen	211.00 (14.13%) 3,927,116.48 (8.72%)		147.00 (8.76%) 3,727,776.36 (4.17%)			
Diet						
Seafood Eaten in Past 30 Days (smpw2cat)						
none[R]	402.00 (26.93%) 11,642,905.87 (25.87%)		284.00 (16.90%) 11,227,934.91 (12.55%)		46.84 *<0.0001*	12.59 *0.0009*
any	1,091.00 (73.07%) 33,367,240.70 (74.13%)		1,396.00 (83.10%) 78,263,951.95 (87.45%)			
Fish Eaten in Past 30 Days (fish2cat)						
none[R]	574.00 (38.45%) 16,052,553.48 (35.66%)		466.00 (27.74%) 20,757,186.19 (23.19%)		41.14 *<0.0001*	6.04 *0.018*
any	919.00 (61.55%) 28,957,593.09 (64.34%)		1,214.00 (72.26%) 68,734,700.67 (76.81%)			

Table 63
Bivariate Analyses of Independent Variables on Sum of PCBs Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30	Below Geometric Mean		At or Above Geometric Mean		χ^2 *p value unweighted*	χ^2 *p value weighted*
Shellfish Eaten in Past 30 Days (shell2cat)						
none[R]	801.00 (53.65%) 23,922,841.35 (53.15%)		756.00 (45.00%) 39,095,797.83 (43.69%)		23.67 *<0.0001*	3.59 *0.064*
any	692.00 (46.35%) 21,087,305.22 (46.85%)		924.00 (55.00%) 50,396,089.03 (56.31%)			
Tap Water Consumed Prior 24h (tap2kct)						
none[R]	568.00 (38.04%) 15,461,106.70 (34.35%)		561.00 (33.39%) 25,043,721.55 (27.98%)			
< 2,000 ml	735.00 (49.23%) 23,331,575.10 (51.84%)		803.00 (47.80%) 47,713,910.32 (53.32%)		38.19 *<0.0001*	3.03 *0.039*
2,000+ ml	132.00 (8.84%) 4,889,498.34 (10.86%)		163.00 (9.70%) 10,640,054.15 (11.89%)			
missing	58.00 (3.88%) 1,327,966.43 (2.95%)		153.00 (9.11%) 6,094,200.84 (6.81%)			
Alcohol Consumption						
Alcohol Consumption (retohuse)						
never, seldom drinker[R] *including 16-19 y/o*	1,009.00 (67.58%) 22,739,993.00 (50.52%)		734.00 (43.69%) 29,480,522.36 (32.94%)			
drinker	245.00 (16.41%) 9,429,807.16 (20.95%)		485.00 (28.87%) 31,240,272.41 (34.91%)		186.93 *<0.0001*	6.47 *0.001*
heavy drinker	201.00 (13.46%) 11,780,202.42 (26.17%)		354.00 (21.07%) 23,985,177.01 (26.80%)			
missing	38.00 (2.55%) 1,060,143.98 (2.36%)		107.00 (6.37%) 4,785,915.08 (5.25%)			
Tobacco Use						
Serum Cotinine (cot3cat)						
< 1.0 ng/ml[R]	1,123.00 (75.37%) 31,818,893.33 (70.72%)		1,245.00 (74.64%) 67,052,580.24 (75.44%)			
1.0 - 10.0 ng/ml	93.00 (6.24%) 2,059,773.91 (4.58%)		97.00 (5.82%) 3,190,527.70 (3.59%)		0.85 *0.655*	0.93 *0.404*
> 10.0 ng/ml	274.00 (18.39%) 11,112,927.46 (24.70%)		326.00 (19.54%) 18,637,413.77 (20.97%)			

Table 63
Bivariate Analyses of Independent Variables on Sum of PCBs Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30	Below Geometric Mean		At or Above Geometric Mean		χ^2 *p value unweighted*	χ^2 *p value weighted*
ETS (ETS)						
no ETS^{R}	1,140.00 (76.36%) 32,938,432.57 (73.18%)		1,277.00 (76.01%) 68,858,939.01 (76.94%)			
ETS at home or work	296.00 (19.83%) 9,460,507.36 (21.02%)		354.00 (21.07%) 17,246,362.24 (19.27%)		2.53 *0.282*	0.47 *0.629*
ETS at home and work	57.00 (3.82%) 2,611,206.64 (5.80%)		49.00 (2.92%) 3,386,585.61 (3.78%)			
Residence						
Tap Water Source (h2os2cat)						
public^{R}	1,341.00 (89.82%) 39,539,285.54 (87.85%)		1,485.00 (88.39%) 77,196,622.63 (86.26%)			
private	109.00 (7.30%) 4,324,982.72 (9.61%)		157.00 (9.35%) 10,166,552.30 (11.36%)		5.30 *0.070*	0.12 *0.886*
missing	43.00 (2.88%) 1,145,878.31 (2.55%)		38.00 (2.26%) 2,128,711.94 (2.38%)			
Residential Tap Water Treatment (h2ox2cat)						
yes	394.00 (26.39%) 14,750,534.69 (32.77%)		469.00 (27.92%) 30,557,699.97 (34.15%)			
no^{R}	1,063.00 (71.20%) 29,276,436.74 (65.04%)		1,176.00 (70.00%) 57,268,900.62 (63.99%)		1.22 *0.544*	0.59 *0.942*
missing	36.00 (2.41%) 983,175.13 (2.18%)		35.00 (2.08%) 1,665,286.27 (1.86%)			
Type of Residence (res3cat)						
attached or detached house^{R}	892.00 (59.75%) 24,693,788.16 (54.86%)		1,180.00 (70.24%) 64,612,182.40 (72.20%)			
mobile home or trailer	101.00 (6.76%) 3,878,638.25 (8.62%)		101.00 (6.01%) 4,523,139.55 (5.05%)		40.49 *<0.0001*	9.78 *0.0003*
all other types *including missing/unknown*	500.00 (33.49%) 16,437,720.16 (36.52%)		399.00 (23.75%) 20,356,564.91 (22.75%)			
Age of Residence (resb60cat)						
1960 or newer^{R}	677.00 (45.34%) 23,503,687.57 (52.22%)		918.00 (54.64%) 54,540,836.45 (60.95%)			
older than 1960	347.00 (23.24%) 9,775,894.29 (21.72%)		419.00 (24.94%) 22,316,305.81 (24.94%)		51.89 *<0.0001*	4.08 *0.024*
missing/unknown	469.00 (31.41%) 11,730,564.71 (26.06%)		343.00 (20.42%) 12,634,744.60 (14.12%)			

Table 63
Bivariate Analyses of Independent Variables on Sum of PCBs Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30	Below Geometric Mean		At or Above Geometric Mean		χ^2 *p value unweighted*	χ^2 *p value weighted*
Age of Residence (resb78cat)						
1978 or newer[R]	447.00 (29.94%) 16,360,463.61 (36.35%)		640.00 (38.10%) 39,027,585.24 (43.61%)			
older than 1978	577.00 (38.65%) 16,919,118.24 (37.59%)		697.00 (41.49%) 37,829,557.02 (42.27%)		54.29 *<0.0001*	4.09 *0.023*
missing/unknown	469.00 (31.41%) 11,730,564.71 (26.06%)		343.00 (20.42%) 12,634,744.60 (14.12%)			
Resident Status (resd3cat)						
own[R]	734.00 (49.16%) 23,922,800.23 (53.15%)		993.00 (59.11%) 53,327,506.90 (59.59%)			
rent	651.00 (43.60%) 18,051,507.75 (40.11%)		627.00 (37.32%) 33,112,387.82 (37.00%)		42.13 *<0.0001*	1.21 *0.308*
other *including missing*	108.00 (7.23%) 3,035,838.58 (6.74%)		60.00 (3.57%) 3,051,992.15 (3.41%)			
Years at Current Residence (re5yrcat)						
more than five years[R]	487.00 (32.62%) 13,709,926.15 (30.46%)		626.00 (37.26%) 32,184,392.54 (35.96%)			
five years or less	981.00 (65.71%) 30,721,629.00 (68.25%)		1,026.00 (61.07%) 55,733,983.94 (62.28%)		7.54 *0.023*	0.84 *0.440*
missing	25.00 (1.67%) 578,591.42 (1.29%)		28.00 (1.67%) 1,573,510.39 (1.76%)			
Household Size (hsize)						
four persons or less[R]	927.00 (62.09%) 32,334,461.84 (71.84%)		1,255.00 (74.70%) 74,119,566.54 (82.82%)			
more than four persons	566.00 (37.91%) 12,675,684.72 (28.16%)		425.00 (25.30%) 15,372,320.32 (17.18%)		58.55 *<0.0001*	7.53 *0.0088*
Rooms in Residence (rm3cat)						
7+ rooms[R]	493.00 (33.02%) 15,009,690.26 (33.35%)		655.00 (38.99%) 37,606,822.55 (42.02%)			
4-6 rooms	819.00 (54.86%) 25,056,886.05 (55.67%)		872.00 (51.90%) 44,043,246.54 (49.21%)		15.92 *0.001*	1.28 *0.293*
1-3 rooms	142.00 (9.51%) 3,897,280.19 (8.66%)		121.00 (7.20%) 6,277,892.92 (7.02%)			
missing	39.00 (2.61%) 1,046,290.06 (2.32%)		32.00 (1.90%) 1,563,924.86 (1.75%)			

Table 63
Bivariate Analyses of Independent Variables on Sum of PCBs Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30	Below Geometric Mean		At or Above Geometric Mean		χ^2 *p value unweighted*	χ^2 *p value weighted*
Occupation						
Current Occupation (cocc2cat)						
not working^{R}	735.00 (49.23%) 17,142,933.21 (38.09%)		589.00 (35.06%) 25,030,024.37 (27.97%)			
management, professional & sales	469.00 (31.41%) 17,786,522.86 (39.52%)		774.00 (46.07%) 49,972,368.87 (55.84%)		81.49 *<0.0001*	6.85 *0.0026*
services & goods	289.00 (19.36%) 10,080,690.49 (22.40%)		317.00 (18.87%) 14,489,493.62 (16.19%)			
Time in Current Employment (cjt)						
not working^{R}	735.00 (49.23%) 17,142,933.21 (38.09%)		589.00 (35.06%) 25,030,024.37 (27.97%)			
less than five years	670.00 (44.88%) 24,183,444.33 (53.73%)		764.00 (45.48%) 43,058,195.40 (48.11%)		149.40 *<0.0001*	11.38 *0.0001*
five or more years	88.00 (5.89%) 3,683,769.03 (8.18%)		327.00 (19.46%) 21,403,667.10 (23.92%)			
Total Hours Worked Prior Week (hrwk)						
not employed^{R}	755.00 (50.57%) 17,890,767.05 (39.75%)		626.00 (37.31%) 27,917,270.98 (31.25%)			
less than 35 hours	353.00 (23.64%) 10,611,357.52 (23.58%)		383.00 (22.82%) 22,756,076.28 (25.47%)		79.27 *<0.0001*	1.51 *0.232*
35+ hours	385.00 (25.79%) 16,508,021.99 (36.68%)		669.00 (39.87%) 38,673,499.01 (43.28%)			
Longest Held Occupation (locc2cat)						
not applicable^{R}	701.00 (46.95%) 20,098,821.46 (44.65%)		861.00 (51.25%) 44,018,535.05 (49.19%)			
management, professional & sales	396.00 (26.52%) 12,437,060.19 (27.63%)		507.00 (30.18%) 28,978,125.96 (32.38%)		29.08 *<0.0001*	2.35 *0.107*
services & goods	396.00 (26.52%) 12,474,264.91 (27.71%)		312.00 (18.57%) 16,495,225.86 (18.43%)			
Time in Longest Employment (ljt)						
not applicable^{R}	701.00 (42.87%) 20,098,821.46 (44.65%)		861.00 (51.25%) 44,018,535.05 (49.19%)			
less than five years	640.00 (43.13%) 19,590,358.38 (43.52%)		357.00 (22.20%) 14,951,910.47 (16.71%)		243.06 *<0.0001*	18.58 *0.0000*
five or more years	152.00 (10.18%) 5,320,966.73 (11.82%)		462.00 (27.50%) 30,521,441.35 (34.11%)			

Table 63
Bivariate Analyses of Independent Variables on Sum of PCBs Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables Sample Frequency Col. Pct. unweighted Population Frequency Col. Pct. weighted R = Reference Group * = cell size less than 30	Below Geometric Mean		At or Above Geometric Mean		χ^2 *p value unweighted*	χ^2 *p value weighted*
Work History (wkcp)						
never employed[R]	241.00 (16.14%) 3,806,816.77 (8.46%)		167.00 (9.94%) 4,431,994.03 (4.95%)			
currently employed	460.00 (30.81%) 16,292,004.69 (36.20%)		694.00 (41.31%) 39,586,541.02 (44.23%)		69.85 *<0.0001*	3.58 *0.021*
employed in the past but not currently	494.00 (33.09%) 13,336,116.44 (29.63%)		422.00 (24.12%) 20,598,030.34 (23.02%)			
employed now and in the past	298.00 (19.96%) 11,575,208.66 (25.72%)		397.00 (23.63%) 24,875,321.48 (27.80%)			
Socioeconomic Factors						
Education						
Highest Education (educ2)						
high school diploma, GED or higher[R]	812.00 (54.42%) 32,046,553.86 (71.30%)		1,225.00 (72.92%) 74,860,607.48 (83.65%)		117.61 *<0.0001*	11.33 *0.002*
less than high school diploma	680.00 (45.58%) 12,896,453.92 (28.70%)		455.00 (27.08%) 14,631,279.38 (16.35%)			
Employment						
Employment Status (emp3cat)						
employed	758.00 (50.84%) 27,867,213.36 (61.93%)		1,095.00 (65.18%) 64,601,586.61 (72.19%)		66.88 *<0.0001*	5.27 *0.026*
not employed[R]	733.00 (49.16%) 17,131,713.69 (38.07%)		585.00 (34.82%) 24,890,300.26 (27.81%)			
Reason for Unemployment (unem2cat)						
working[R]	758.00 (50.77%) 27,867,213.36 (61.91%)		1,095.00 (65.18%) 64,601,586.61 (72.19%)			
voluntary unemployment	524.00 (35.10%) 11,505,486.37 (25.56%)		400.00 (23.81%) 16,759,535.30 (18.73%)		102.69 *<0.0001*	2.29 *0.091*
involuntary unemployment	132.00 (8.84%) 3,668,819.47 (8.15%)		163.00 (9.70%) 6,594,294.54 (7.37%)			
missing	79.00 (5.29%) 1,968,627.37 (4.37%)		22.00 (1.31%) 1,536,470.42 (1.72%)			
Income						
U.S. Poverty Threshold (pov2cat)						
more than 1.00[R]	990.00 (66.31%) 32,255,606.34 (71.66%)		1,237.00 (73.63%) 71,698,016.85 (80.12%)		30.72 *<0.0001*	2.03 *0.144*
1.00 or less	409.00 (27.39%) 8,921,998.51 (19.82%)		321.00 (19.11%) 13,665,198.96 (15.27%)			
missing	94.00 (6.30%) 3,832,541.72 (8.51%)		122.00 (7.26%) 4,128,671.05 (4.61%)			

Table 63
Bivariate Analyses of Independent Variables on Sum of PCBs Among Childbearing-Aged Females
(unweighted and weighted data 1999-2004)

Independent Variables Sample Frequency Col. Pct. $_{unweighted}$ Population Frequency Col. Pct. $_{weighted}$ ^{R} = Reference Group * = cell size less than 30	Below Geometric Mean		At or Above Geometric Mean		χ^2 *p value unweighted*	χ^2 *p value weighted*
Marital Status						
Marital Status (marr3cat)						
	454.00		744.00			
	(30.41%)		(44.29%)			
	15,664,599.68		46,136,048.57			
married or living with partner	(34.80%)		(51.55%)			
	61.00		200.00			
	(4.09%)		(11.90%)			
	2,370,074.71		11,983,877.67		213.66	13.55
widowed, divorced or separated	(5.27%)		(13.39%)		*<0.0001*	*0.0000*
	963.00		674.00			
	(64.50%)		(40.12%)			
	26,043,831.55		27,449,119.94			
never married^{R}	(57.86%)		(30.67%)			
	15.00		62.00			
	(1.00%)		(3.69%)			
	931,640.64		3,922,840.67			
missing	(2.07%)		(4.28%)			
Race-Ethnicity						
Race-Ethnicity (race5cat)						
	625.00		868.00			
Non-Hispanic White^{R}	(41.86%)		(51.67%)			
$n_{unweighted}$ = 1493	30,387,650.82		67,499,893.35			
$n_{weighted}$ = 97,887,544.16	(67.51%)		(75.43%)			
	276.00		347.00			
Non-Hispanic Black	(18.49%)		(20.65%)			
$n_{unweighted}$ = 623	3,895,016.67		8,852,161.70			
$n_{weighted}$ = 12,747,178.37	(8.65%)		(9.89%)			
	430.00		315.00		69.45	3.55
Mexican American	(28.80%)		(18.75%)		*<0.0001*	*0.014*
$n_{unweighted}$ = 745	5,073,367.51		3,597,208.29			
$n_{weighted}$ = 8,670,575.80	(11.27%)		(4.02%)			
	109.00		69.00			
Other Hispanic	(7.30%)		(4.11%)			
$n_{unweighted}$ = 178	3,277,436.73		4,248,555.49			
$n_{weighted}$ = 7,525,992.22	(7.28%)		(4.75%)			
	53.00		81.00			
Asian, Native American, Pacific Islander	(3.55%)		(4.82%)			
& Multi-Racial	2,376,674.85		5,294,068.03			
$n_{unweighted}$ = 134	(5.28%)		(5.92%)			
$n_{weighted}$ = 7,670,742.88						
Race-Ethnicity/Hispanic Grouping (race4cat)						
	625.00		868.00			
Non-Hispanic White^{R}	(41.86%)		(51.67%)			
$n_{unweighted}$ = 1493	30,387,650.82		67,499,893.35			
$n_{weighted}$ = 97,887,544.16	(67.51%)		(75.43%)			
	276.00		347.00			
Non-Hispanic Black	(18.49%)		(20.65%)			
$n_{unweighted}$ = 623	3,895,016.67		8,852,161.70		68.74	3.58
$n_{weighted}$ = 12,747,178.37	(8.65%)		(9.89%)		*<0.0001*	*0.021*
	539.00		384.00			
Hispanic	(36.10%)		(22.86%)			
$n_{unweighted}$ = 923	8,350,804.24		7,845,763.79			
$n_{weighted}$ = 16,196,568.02	(18.55%)		(8.77%)			
	53.00		81.00			
Asian, Native American, Pacific Islander	(3.55%)		(4.82%)			
& Multi-Racial	2,376,674.85		5,294,068.03			
$n_{unweighted}$ = 134	(5.28%)		(5.92%)			
$n_{weighted}$ = 7,670,742.88						

Table 64
Summary of Chi-Square and *p* Values of Weighted Independent Variables on Sum of PCBs (1999-2004)

Variables *p < 0.20*	*p* value	χ^2 *weighted*	Variables *p > 0.20*	*p* value	χ^2 *weighted*
Age (age4cat)	*0.0000*	26.44	Regular Source of Healthcare (hp2cat)	*0.226*	1.51
Time in Longest Employment (ljt)	*0.0000*	18.58	Iron Deficiency and Treatment (FeDTx)	*0.228*	1.49
Marital Status (marr3cat)	*0.0000*	13.55	Total Hours Worked Prior Week (hrwk)	*0.232*	1.51
Live Births (live)	*0.0001*	19.71	Rooms in Residence (rm3cat)	*0.293*	1.28
Time in Current Employment (cjt)	*0.0001*	11.38	Resident Status (resd3cat)	*0.308*	1.21
Ever Pregnant (tprg2cat)	*0.0002*	17.10	Serum Cotinine (cot3cat)	*0.404*	0.93
Current Pregnancy (pregnant)	*0.0002*	10.55	Years at Current Residence (re5yrcat)	*0.440*	0.84
Type of Residence (res3cat)	*0.0003*	9.78	Perceived Health Status (huq2cat)	*0.443*	0.59
Seafood in Past 30 Days [1] (smpw2cat)	*0.0009*	12.59	Calcium Intake/RDA (calc2cat)	*0.454*	0.57
Alcohol Consumption (retohuse)	*0.0010*	6.47	Protein Intake/AMDR (prot3cat)	*0.502*	0.46
Language Spoken at Home (lang2cat)	*0.0013*	11.75	Iron Deficiency (FeD2cat)	*0.516*	0.43
Highest Education (educ2)	*0.0016*	11.33	Charleson Co-Morbidity Scale (CCMS3cat)	*0.626*	0.47
U.S. Citizenship (usczn2cat)	*0.0025*	10.29	Environmental Tobacco Smoke (ETS)	*0.629*	0.47
Current Occupation[1] (cocc2cat)	*0.0026*	6.85	Fat Intake/AMDR (fat3cat)	*0.852*	0.03
Household Size (hsize)	*0.0088*	7.53	Tap Water Source (h2os2cat)	*0.886*	0.12
Race-Ethnicity[1] (race5cat)	*0.014*	3.55	Residential Tap Water Treatment (h2ox2cat)	*0.942*	0.59
Iron Intake/RDA[1] (iron2cat)	*0.017*	6.19			
Fish in Past 30 Days (fish2cat)	*0.018*	6.04			
Work History[1] (wkcp)	*0.021*	3.58			
Race-Ethnicity/Hispanic Grouping (race4cat)	*0.021*	3.58			
Age of Residence (resb78cat)	*0.023*	4.09			
Age of Residence (resb60cat)	*0.024*	4.08			
Health Insurance (hi2cat)	*0.025*	3.44			
Employment Status (emp3cat)	*0.026*	5.27			
Trimester of Pregnancy[1] (tripcorr)	*0.026*	3.40			
Tap Water Consumed 24h (tap2kct)	*0.039*	3.03			
Food Security (food2cat)	*0.043*	3.39			
Years in U.S. (yrus5)	*0.053*	3.15			
Shellfish in Past 30 Days (shell2cat)	*0.064*	3.59			

[1]variable dropped due to low cell size or too similar to other variables

Table 64
Summary of Chi-Square and *p* Values of Weighted Independent Variables on Sum of PCBs (1999-2004)

Variables *p < 0.20*	*p* value	χ^2 *weighted*	Variables *p > 0.20*	*p* value	χ^2 *weighted*
Birthplace[1] (born2cat)	*0.079*	3.24			
Reason for Unemployment[1] (unem2cat)	*0.091*	2.29			
Body Mass Index (bmi30cat)	*0.094*	2.49			
Treatment for Iron Deficiency[1] (FeTx2cat)	*0.096*	2.89			
Selenium Intake/RDA (sele2cat)	*0.102*	2.79			
Longest Held Occupation[1] (locc2cat)	*0.107*	2.35			
Source of Healthcare[1] (hcsre)	*0.114*	2.09			
Ever Breastfed (brstfda)	*0.136*	2.31			
U.S. Poverty Threshold[1] (pov2cat)	*0.144*	2.03			

[1]variable dropped due to low cell size or too similar to other variables

Table 65
Stepwise Logistic Regression Analyses of Sum of PCBs (1999-2004)

Variable Name *p < 0.20*	df	-2LL Wald F	Difference	df	*p* value
Initial Regression	41	1,366.79			
1 Time in Current Employment (cjt)	39	1,366.64	0.15	2	*>0.20* *drop*
2 Health Insurance (hi2cat)	36	1,365.14	1.50	3	*>0.20* *drop*
3 Alcohol Consumption (retohuse)	33	1,360.90	4.24	3	*>0.20* *drop*
4 Marital Status (marr3cat)	30	1,352.84	8.06	3	*<0.05* *keep*
5 Highest Education (educ2)	32	1,363.12	2.22	1	*>0.10* *drop*
6 Race-Ethnicity/Hispanic Grouping (race4cat)	29	1,354.53	8.59	3	*<0.05* *keep*
7 Selenium Intake/RDA (sele2cat)	31	1,360.94	2.18	1	*>0.10* *drop*
8 Language Spoken at Home (lang2cat)	30	1,356.49	4.45	1	*<0.05* *keep*
9 Years in U.S. (yrus5)	29	1,350.69	10.25	2	*<0.01* *keep*
10 U.S. Citizenship (usczn2cat)	30	1,359.07	1.87	1	*>0.01* *drop*
11 Time in Longest Employment (ljt)	28	1,263.41	95.66	2	*<0.001* *keep*
12 Household Size (hsize)	29	1,335.03	6.04	1	*<0.02* *keep*
13 Age of Residence 1978 (resb78cat)	28	1,315.03	44.04	2	*<0.001* *keep*
14 Type of Residence (res3cat)	28	1,328.02	31.05	2	*<0.001* *keep*
15 Tap Water Consumed 24h (tap2kct)	29	1,342.54	16.53	1	*<0.001* *keep*
16 Shellfish in Past 30 Days (shell2cat)	31	1,362.33	3.26	1	*>0.05* *drop*
17 Fish in Past 30 Days (fish2cat)	30	1,333.34	28.99	1	*<0.001* *keep*
18 Age (age4cat)	28	974.94	387.39	3	*<0.001* *keep*

Table 66
Best-Fit Logistic Regression PCBs Model *with no interactions* (1999-2004)

Variable Name	df	-2LL Wald F	R^2		*p* value
Best Fit Regression	31	1,362.33	*0.3498*		
Age (age4cat)	3	29.97			*0.0000*
Body Mass Index (bmi30cat)	2	11.50			*0.0001*
Current Pregnancy (pregnant)	2	10.37			*0.0002*
Time in Longest Employment (ljt)	2	10.32			*0.0002*
Ever Breastfed (brstfda)	1	13.61			*0.0006*
Type of Residence (res3cat)	2	6.07			*0.0047*
Household Size (hsize)	1	6.70			*0.0130*
Live Births (live)	1	6.36			*0.0154*
Tap Water Consumed 24h (tap2kct)	3	3.78			*0.0170*
Fish in Past 30 Days (fish2cat)	1	4.52			*0.0392*
Age of Residence 1978 (resb78cat)	2	2.77			*0.0739*
Food Security (food2cat)	2	2.76			*0.0744*
Years in U.S. (yrus5)	2	2.45			*0.0981*
Language Spoken at Home (lang2cat)	1	1.64			*0.2074*
Race-Ethnicity/Hispanic Grouping (race4cat)	3	0.75			*0.5304*
Marital Status (marr3cat)	3	0.72			*0.5435*

[1]in ascending order by p value

Table 65
Stepwise Logistic Regression Analyses of Sum of PCBs (1999-2004)

Variable Name *p < 0.20*	df	-2LL Wald F	Difference	df	*p* value
19 Food Security (food2cat)	29	1,343.08	19.25	2	*<0.001* *keep*
20 Body Mass Index (bmi30cat)	29	1,245.42	116.91	2	*<0.001* *keep*
21 Current Pregnancy (pregnant)	29	1,277.07	85.26	2	*<0.001* *keep*
22 Live Births (live)	30	1,345.88	16.45	1	*<0.001* *keep*
23 Ever Breastfed (brstfda)	30	1,309.47	52.86	1	*<0.001* *keep*

Table 66
Best-Fit Logistic Regression PCBs Model *with no interactions* (1999-2004)

Variable Name	df	-2LL Wald F	R^2		*p* value

Table 67
Variance Inflation Factor Test for Collinearity Among Independent Variables using the Best-Fit Logistic Regression PCBs Model (1999-2004)

	df	sum of squares	mean square	F value	pr>F	
Best Fit PCBs Model *with no interactions*	16	118.561	7.410	55.32	*<0.0001*	

Variable Name	df	parameter estimate	std error	t value	pr>t	VIF
Intercept	1	0.053	0.033	1.61	*0.1085*	0.0000
Age (age4cat)	1	0.044	0.003	14.99	*<0.0001*	1.9151
Food Security (food2cat)	1	-0.019	0.005	-4.13	*<0.0001*	1.0533
Body Mass Index (bmi30cat)	1	-0.038	0.005	-7.09	*<0.0001*	1.0488
Current Pregnancy (pregnant)	1	0.050	0.005	9.98	*<0.0001*	1.6317
Live Births (live)	1	0.032	0.010	3.19	*0.0014*	2.8670
Ever Breastfed (brstfda)	1	-0.043	0.011	-3.87	*0.0001*	2.3533
Years in U.S. (yrus5)	1	-0.008	0.006	-1.31	*0.1917*	2.1005
Language Spoken at Home (lang2cat)	1	-0.032	0.011	-2.95	*0.0032*	2.0401
Fish in Past 30 Days (fish2cat)	1	0.059	0.006	10.68	*<0.0001*	1.2683
Tap Water Consumed 24h (tap2kct)	1	-0.002	0.003	-0.58	*0.5599*	1.0307
Type of Residence (res3cat)	1	0.002	0.003	0.71	*0.4789*	1.1747
Age of Residence (resb78cat)	1	0.008	0.003	2.50	*0.0125*	1.1597
Household Size (hsize)	1	-0.008	0.005	-1.49	*0.1360*	1.1343
Time in Longest Employment (ljt)	1	0.003	0.003	0.88	*0.3814*	1.0483
Marital Status (marr3cat)	1	-0.010	0.003	-3.06	*0.0022*	1.8336
Race-Ethnicity/Hispanic Grouping (race4cat)	1	-0.001	0.003	-0.50	*0.6193*	1.2783

Table 68
Statistical Significance of Interactions Between Independent Variables using the Best-Fit Logistic Regression PCBs Model (1999-2004)

Independent Variables	Age (age4cat)	Food Security (food2cat)	Body Mass Index (bmi30cat)	Current Pregnancy (pregnant)	Live Births (live)	Ever Breastfed (brstfda)	Years in U.S. (yrus5)	Language Spoken at Home (lang2cat)	Fish in Past 30 Days (fish2cat)	Tap Water Consumed 24h (tap2kct)	Type of Residence (res3cat)	Age of Residence 1978 (resb78cat)	Household Size (hsize)	Time in Longest Employment (ljt)	Marital Status (marr3cat)	Race-Ethnicity Hispanic Grouping (race4cat)
Age (age4cat)																
Food Security (food2cat)	*<0.001*															
Body Mass Index (bmi30cat)	*op*	*op*														
Current Pregnancy (pregnant)	*op*	*op*	*op*													
Live Births (live)	*ns*	*ns*	*op*	*ns*												
Ever Breastfed (brstfda)	*ns*	*<0.01*	*op*	*ns*	*op*											
Years in U.S. (yrus5)	*ns*	*<0.01*	*op*	*op*	*ns*	*ns*										
Language Spoken at Home (lang2cat)	*<0.01*	*ns*	*ns*	*<0.05*	*ns*	*ns*	*<0.05*									
Fish in Past 30 Days (fish2cat)	*ns*	*<0.01*	*<0.001*	*ns*	*<0.05*	*ns*	*<0.02*	*<0.02*								
Tap Water Consumed 24h (tap2ket)	*<0.001*	*<0.001*	*<0.001*	*op*	*<0.001*	*<0.001*	*op*	*<0.01*	*<0.001*							
Type of Residence (res3cat)	*<0.001*	*op*	*op*	*op*	*ns*	*ns*	*<0.02*	*ns*	*<0.02*	*<0.01*						
Age of Residence 1978 (resb78cat)	*<0.001*	*<0.01*	*ns*	*ns*	*<0.01*	*<0.02*	*<0.05*	*ns*	*<0.001*	*ns*	*<0.01*					
Household Size (hsize)	*<0.02*	*ns*	*ns*	*ns*	*ns*	*<0.05*	*ns*	*<0.02*	*ns*	*<0.01*	*<0.001*	*<0.001*				
Time in Longest Employment (ljt)	*<0.001*	*<0.001*	*<0.01*	*op*	*<0.01*	*<0.01*	*<0.001*	*<0.001*	*<0.05*	*<0.001*	*<0.001*	*<0.001*	*<0.02*			
Marital Status (marr3cat)	*op*	*op*	*op*	*op*	*<0.001*	*<0.001*	*ns*	*ns*	*<0.001*	*op*	*<0.001*	*<0.001*	*<0.001*	*<0.001*		
Race-Ethnicity/Hispanic Grouping (race4cat)	*op*	*<0.001*	*op*	*op*	*ns*	*<0.001*	*op*	*ns*	*<0.001*	*<0.001*	*<0.001*	*ns*	*ns*	*<0.001*	*op*	

op = over parameterized unable to calculate
ns = not statistically significant p>0.05

Table 69
Odds Ratios and Confidence Intervals for Best-Fit Logistic Regression PCBs Model *with no interactions*
(1999-2004)

Variable Names	df	-2LL Wald F	*p* value	Odds Ratios	Confidence Intervals
Susceptibility-Related Attributes					
Age (age4cat)	3	29.97	*0.0000*		
16-19[R]				1.00	*ns*
20-29				2.75	1.42 - 5.31 %
30-39				7.31	3.55 - 15.06 %
40-49				95 27	35.91 - 252.78 %
Nutritional Status					
Food Security (food2cat)	2	2.76	*0.0744*		
food secure[R]				1.00	*ns*
food insecure				0.56	0.30 - 1.06 %
missing				1.64	0.46 - 5.82 %
Body Mass Index (bmi30cat)	2	11.50	*0.0001*		
<30.0[R] underweight, normal, overweight				1.00	*ns*
30.0+ obese				0.26	0.14 - 0.48 %
missing				5.13	0.08 - 328.61 %
Reproductive Status					
Current Pregnancy (pregnant)	2	10.37	*0.0002*		
pregnant				0.61	0.28 - 1.30 %
not pregnant[R]				1.00	*ns*
missing				25.23	5.62 - 113.27 %
Live Births (live)	1	6.36	*0.0154*		
no live births[R]				1.00	*ns*
one or more live births				2.16	1.17 - 3.99 %
Ever Breastfed (brstfda)	1	13.61	*0.0006*		
never breastfed[R]				1.00	*ns*
breastfed more than one month or currently				0.27	0.13 - 0.55 %
Exposure-Related Attributes					
Acculturation					
Years in U.S. (yrus5)	2	2.45	*0.0981*		
born in U.S.[R]				1.00	*ns*
five or more years				0.46	0.20 - 1.04 %
less than five years				1.02	0.24 - 4.31 %
Language Spoken at Home (lang2cat)	1	1.64	*0.2074*		
English[R]				1.00	*ns*
Other				0.53	0.20 - 1.44 %
Diet					
Fish in Past 30 Days (fish2cat)	1	4.52	*0.0392*		
none[R]				1.00	*ns*
any				1.83	1.03 - 3.24 %

[R] = referent group
ns = not significant

Table 69
Odds Ratios and Confidence Intervals for Best-Fit Logistic Regression PCBs Model *with no interactions*
(1999-2004)

Variable Names	df	-2LL Wald F	*p* value	Odds Ratios	Confidence Intervals
Tap Water Consumed 24h (tap2kct)	3	3.78	*0.0170*		
none[R]				1.00	*ns*
< 2,000 ml				0.80	0.44 - 1.45 %
2,000+ ml				0.88	0.37 - 2.14 %
missing				3.02	1.06 - 8.60 %
Residence					
Type of Residence (res3cat)	2	6.07	*0.0047*		
attached or detached house[R]				1.00	*ns*
mobile home or trailer				0.27	0.13 - 0.58 %
all other types *including missing/unknown*				0.89	0.52 - 1.52 %
Age of Residence 1978 (resb78cat)	2	2.77	*0.0739*		
1978 or newer[R]				1.00	*ns*
older than 1978				0.81	0.56 - 1.16 %
missing/unknown				0.38	0.15 - 0.92 %
Household Size (hsize)	1	6.70	*0.0130*		
four persons or less[R]				1.00	*ns*
more than four persons				0.53	0.32 - 0.87 %
Occupation					
Time in Longest Employment (ljt)	2	10.32	*0.0002*		
not applicable[R]				1.00	*ns*
less than five years				0.48	0.31 - 0.75 %
five or more years				1.92	0.78 - 4.71 %
Socioeconomic Factors					
Marital Status					
Marital Status (marr3cat)	3	0.72	*0.5435*		
married or living with partner				1.17	0.65 - 2.12 %
widowed, divorced or separated				1.11	0.52 - 2.36 %
never married[R]				1.00	*ns*
missing				2.66	0.66 - 10.75 %
Race-Ethnicity					
Race-Ethnicity/Hispanic Grouping (race4cat)	3	0.75	*0.5304*		
Non-Hispanic White[R]				1.00	*ns*
Non-Hispanic Black				1.63	0.77 - 3.49 %
Hispanic				0.96	0.41 - 2.29 %
Asian, Native American, Pacific Islander & Multi-Racial				1.41	0.55 - 3.62 %

[R] = referent group
ns = not significant

Table 70
Comparisons Among Logistic Regression Models for Exposure as Outcome with Two Categories, Lead, Methylmercury and PCBs *with no interactions* (1999-2004)

Category	Exposure: Best Fit Model *with no interactions*				Lead: Best Fit Model *with no interactions*				Methylmercury Best Fit Model *with no interactions*				PCBs: Best Fit Model *with no interactions*			
	Variable Name	df	-2LL Wald F	*p* value	Variable Name	df	-2LL Wald F	*p* value	Variable Name	df	-2LL Wald F	*p* value	Variable Name	df	-2LL Wald F	*p* value
	Best Fit Regression	24	1,002.16	$r^2 = 0.2719$	Best Fit Regression	31	965.55	$r^2 = 0.2636$	Best Fit Regression	25	845.61	$r^2 = 0.2340$	Best Fit Regression	31	1,362.33	$r^2 = 0.3498$
Age																
Age	Age (age4cat)	3	11.92	*0.0000*	Age (age4cat)	3	6.12	*0.0014*	Age (age4cat)	3	3.02	*0.0395*	Age (age4cat)	3	29.97	*0.0000*
Health Status																
Perceived Health Status									Perceived Health Status (huq2cat)	1	5.62	*0.0222*				
Charleson Co-Morbidity					Charleson Co-Morbidity Scale (CCMS3cat)	2	0.44	*0.6481*								
Health Insurance					Health Insurance (hi2cat)	3	4.18	*0.0109*								
Nutritional Status																
Food Security	Food Security (food2cat)	2	5.94	*0.0052*									Food Security (food2cat)	2	2.76	*0.0744*
Body Mass Index									Body Mass Index (bmi30cat)	2	1.22	*0.3056*	Body Mass Index (bmi30cat)	2	11.50	*0.0001*
Protein Intake/AMDR					Protein Intake/AMDR (prot3cat)	1	1.92	*0.1727*								
Selenium Intake/AMDR	Selenium Intake/RDA (sele2cat)	1	2.44	*0.1255*					Selenium Intake/RDA (sele2cat)	1	2.56	*0.1165*				
Reproductive Status																
Current Pregnancy					Current Pregnancy (pregnant)	2	4.96	*0.0114*	Current Pregnancy (pregnant)	2	2.49	*0.0949*	Current Pregnancy (pregnant)	2	10.37	*0.0002*
Live Births													Live Births (live)	1	6.36	*0.0154*
Ever Breastfed more than one month	Ever Breastfed (brstfda)	1	5.32	*0.0258*									Ever Breastfed (brstfda)	1	13.61	*0.0006*
Acculturation																
Years in U.S.													Years in U.S. (yrus5)	2	2.45	*0.0981*
Language Spoken at Home													Language Spoken at Home (lang2cat)	1	1.64	*0.2074*
U.S. Citizenship					U.S. Citizenship (usczn2cat)	1	29.11	*0.0000*								
Diet																
Fish Past 30 Days	Fish Eaten in Past 30 Days (fish2cat)	1	26.26	*0.0000*					Fish in Past 30 Days (fish2cat)	1	49.55	*0.0000*	Fish in Past 30 Days (fish2cat)	1	4.52	*0.0392*
Shellfish Past 30 Days	Shellfish Eaten in Past 30 Days (shell2cat)	1	3.73	*0.0598*					Shellfish in Past 30 Days (shell2cat)	1	7.84	*0.0076*				
Tap Water Consumed 24h									Tap Water Consumed 24h (tap2kct)	3	1.66	*0.1898*	Tap Water Consumed 24h (tap2kct)	3	3.78	*0.0170*
Alcohol Consumption																
Alcohol Consumption	Alcohol Consumption (retohuse)	3	1.60	*0.2020*	Alcohol Consumption (retohuse)	3	3.59	*0.0209*	Alcohol Consumption (retohuse)	3	1.24	*0.3081*				
Tobacco Use																
Serum Cotinine	Serum Cotinine (cot3cat)	2	1.37	*0.2641*	Serum Cotinine (cot3cat)	2	5.16	*0.0097*								

Table 70
Comparisons Among Logistic Regression Models for Exposure as Outcome with Two Categories, Lead, Methylmercury and PCBs*with no interactions* (1999-2004)

Category	Exposure: Best Fit Model *with no interactions*				Lead: Best Fit Model *with no interactions*				Methylmercury Best Fit Model *with no interactions*				PCBs: Best Fit Model *with no interactions*			
	Variable Name	df	-2LL Wald F	*p* value	Variable Name	df	-2LL Wald F	*p* value	Variable Name	df	-2LL Wald F	*p* value		df	-2LL Wald F	*p* value
Residence																
Type of Residence					Type of Residence (res3cat)	2	1.62	*0.2089*	Type of Residence (res3cat)	2	3.47	*0.0397*	Type of Residence (res3cat)	2	6.07	*0.0047*
Age of Residence 1960					Age of Residence 1960 (resb60cat)	2	6.26	*0.0041*								
Age of Residence 1978													Age of Residence 1978 (resb78cat)	2	2.77	*0.0739*
Household Size	Household Size (hsize)	1	1.55	*0.2193*					Household Size (hsize)	1	1.65	*0.2057*	Household Size (hsize)	1	6.70	*0.0130*
Occupation																
Time Current Employment					Time in Current Employment (cjt)	2	0.97	*0.3877*								
Total Hours Worked					Total Hours Worked Prior Week (hrwk)	2	0.20	*0.8160*								
Time Longest Employment	Time in Longest Employment (ljt)	2	1.68	*0.1976*					Time in Longest Employment (ljt)	2	2.42	*0.1011*	Time in Longest Employment (ljt)	2	10.32	*0.0002*
Education																
Highest Education	Highest Education (educ2)	1	3.81	*0.0572*												
Marital Status																
Marital Status	Marital Status (marr3cat)	3	2.13	*0.1106*	Marital Status (marr3cat)	3	2.29	*0.0917*					Marital Status (marr3cat)	3	0.72	*0.5435*
Race-Ethnicity																
Race-Ethnicity/Hispanic	Race-Ethnicity/Hispanic Grouping (race4cat)	3	0.96	*0.4210*	Race-Ethnicity/Hispanic Grouping (race4cat)	3	3.36	*0.0269*	Race-Ethnicity/Hispanic Grouping (race4cat)	3	0.53	*0.6645*	Race-Ethnicity/Hispanic Grouping (race4cat)	3	0.75	*0.5304*

Table 71
Odds Ratios and Confidence Intervals for Current Pregnancy in Four Best-Fit Logistic Regression Models *with no interactions* (1999-2004)

Variable	Exposure Model		Lead Model		Methylmercury Model		PCBs Model	
	Odds Ratios (95% CI)	Confidence Intervals	Odds Ratios	Confidence Intervals	Odds Ratios	Confidence Intervals	Odds Ratios	Confidence Intervals
Current Pregnancy (pregnant)								
pregnant	NA	NA	0.31	0.14 - 0.65 %	0.65	0.37 - 1.11 %	0.61	0.28 - 1.30 %
not pregnant[R]	NA	NA	1.00	*ns*	1.00	*ns*	1.00	*ns*
missing	NA	NA	1.23	0.45 - 3.36 %	0.24	0.03 - 2.06 %	25.23	5.62 - 113.27 %

Table 72
Odds Ratios and Confidence Intervals for Age in Four Best-Fit Logistic Regression Models *with no interactions* (1999-2004)

Variable	Exposure Model		Lead Model		Methylmercury Model		PCBs Model	
	Odds Ratios	Confidence Intervals	Odds Ratios	Confidence Intervals	Odds Ratios	Confidence Intervals	Odds Ratios	Confidence Intervals
Age (age4cat)								
16-19[R]	1.00	*ns*	1.00	*ns*	1.00	*ns*	1.00	*ns*
20-29	3.50	1.56 - 7.85	0.87	0.47 - 1.63 %	2.32	1.30 - 4.14 %	2.75	1.42 - 5.31 %
30-39	8.48	3.16 - 22.74	1.60	0.70 - 3.68 %	2.14	1.17 - 3.93 %	7.31	3.55 - 15.06 %
40-49	30.20	8.36 - 109.15	4.31	1.93 - 9.62 %	2.11	1.06 - 4.22 %	95 27	35.91 - 252.78 %

Table 73
Birth Cohorts by Age and Survey Years (1999 - 2004)

Age	1999	2001	2003	1999 - 2004
16 - 19	1980 - 1989	1982 - 1991	1984 - 1993	1980 - 1993
20 - 29	1970 - 1979	1972 - 1981	1974 - 1983	1970 - 1983
30 - 39	1960 - 1969	1962 - 1971	1964 - 1973	1960 - 1973
40 - 49	1950 - 1959	1952 - 1961	1954 - 1963	1950 - 1963

FIGURE 3

HISTOGRAM OF LOG DETECTABLE LEAD (1999-2000)

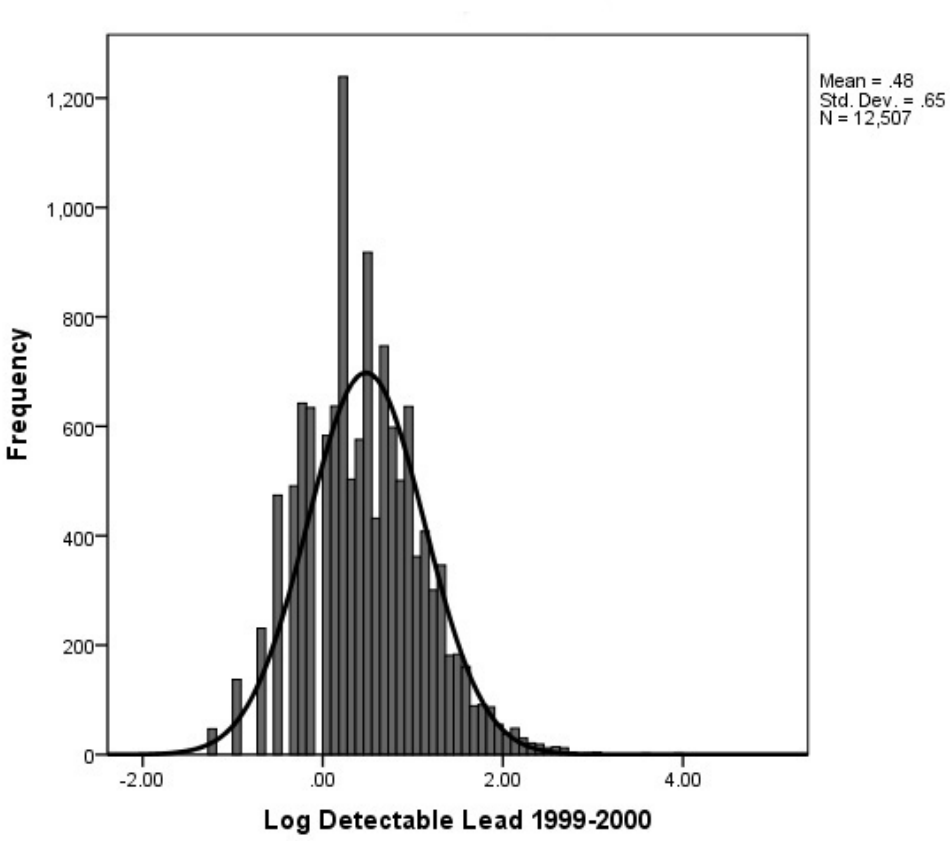


FIGURE 4

HISTOGRAM OF LOG DETECTABLE LEAD (2001-2002)

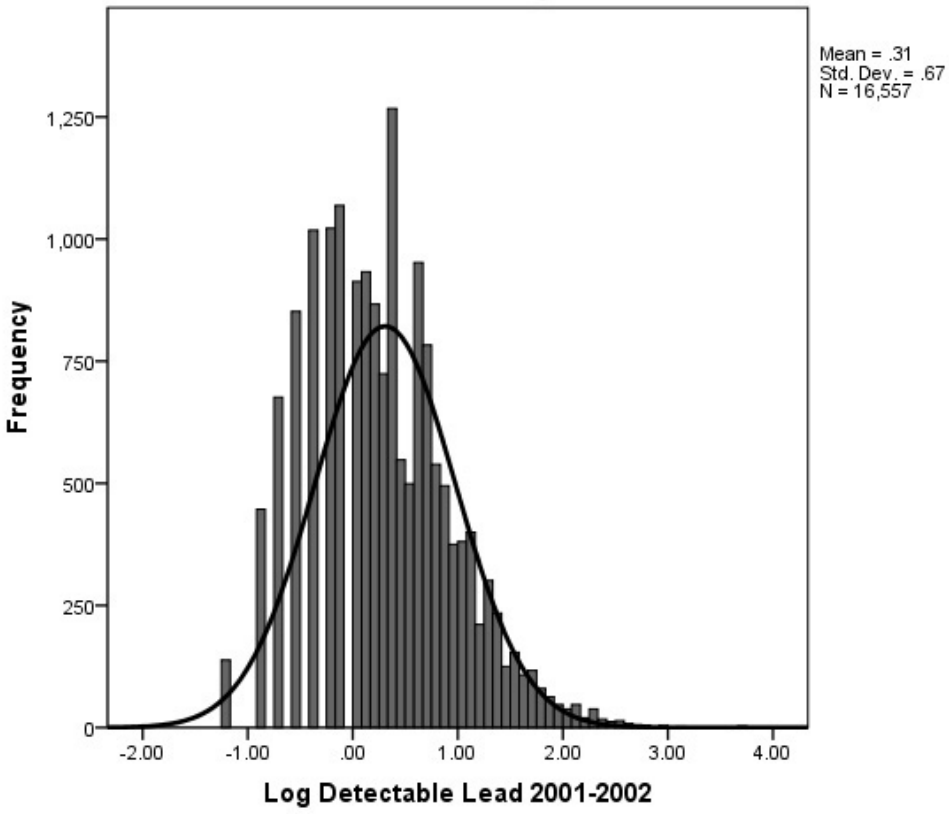


FIGURE 5

HISTOGRAM OF LOG DETECTABLE LEAD (2003-2004)

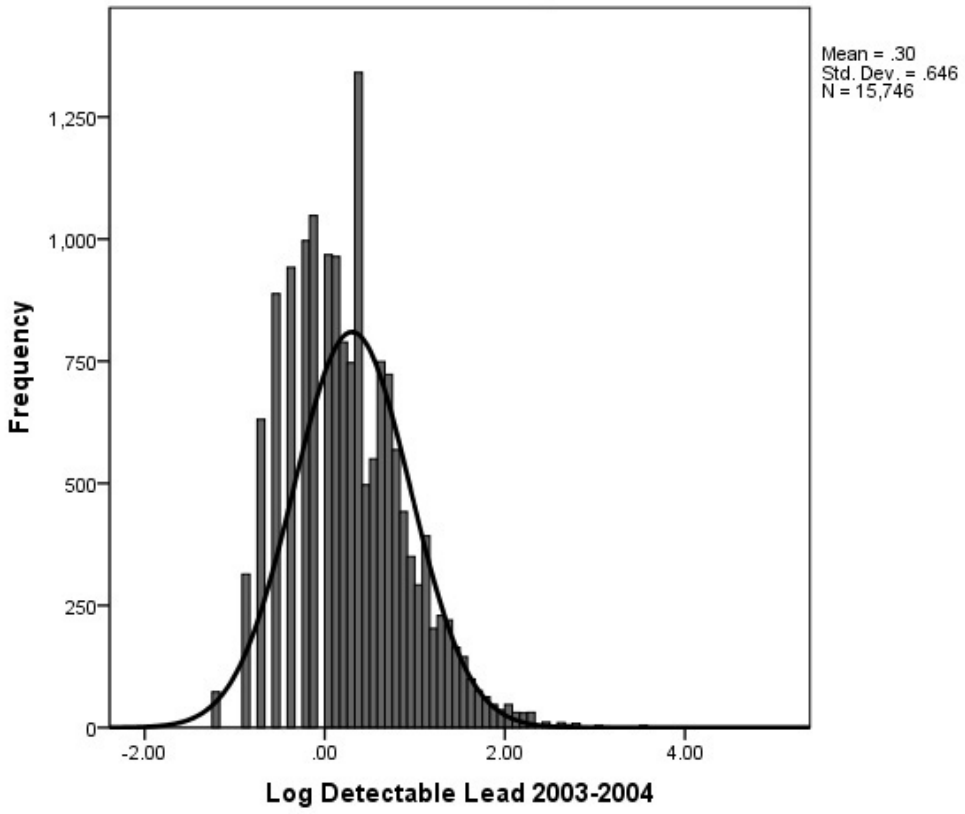


FIGURE 6

HISTOGRAM OF LOG DETECTABLE TOTAL MERCURY (1999-2000)

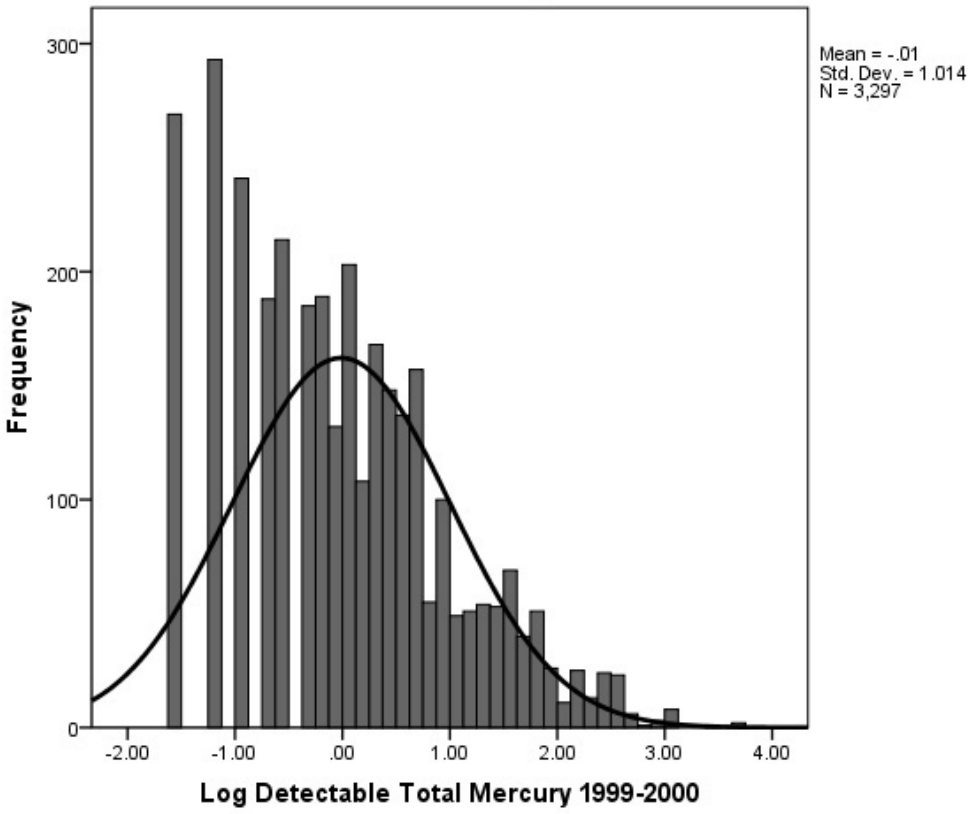


FIGURE 7

HISTOGRAM OF LOG DETECTABLE TOTAL MERCURY (2001-2002)

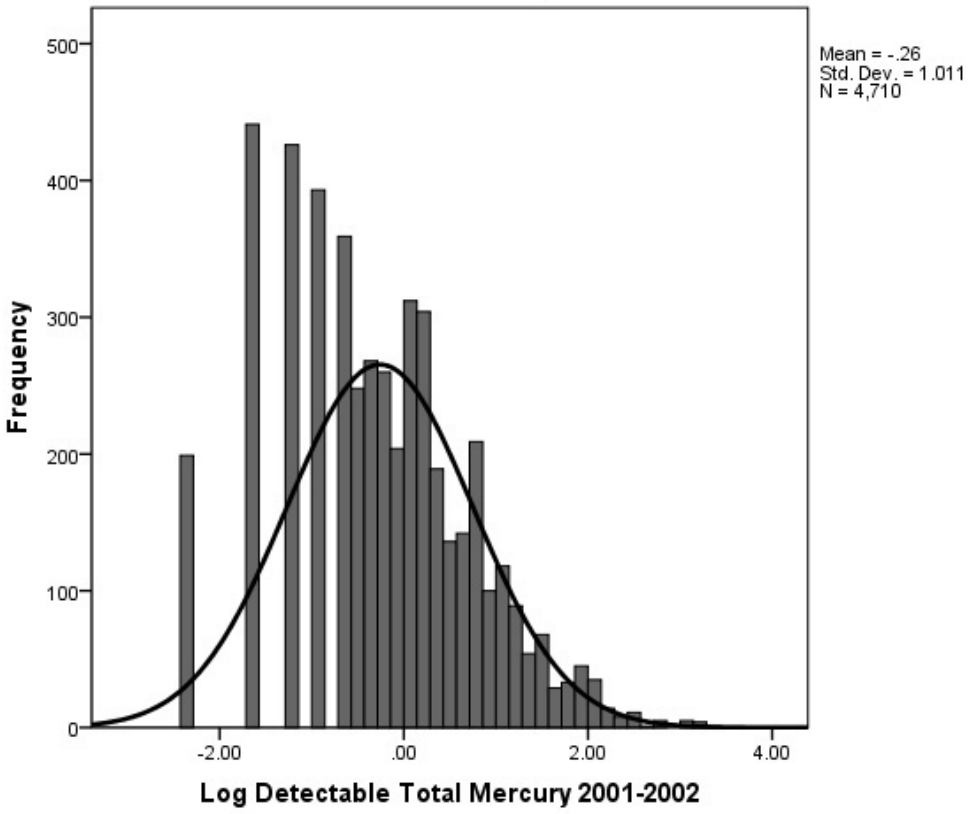


FIGURE 8

HISTOGRAM OF LOG DETECTABLE TOTAL MERCURY (2003-2004)

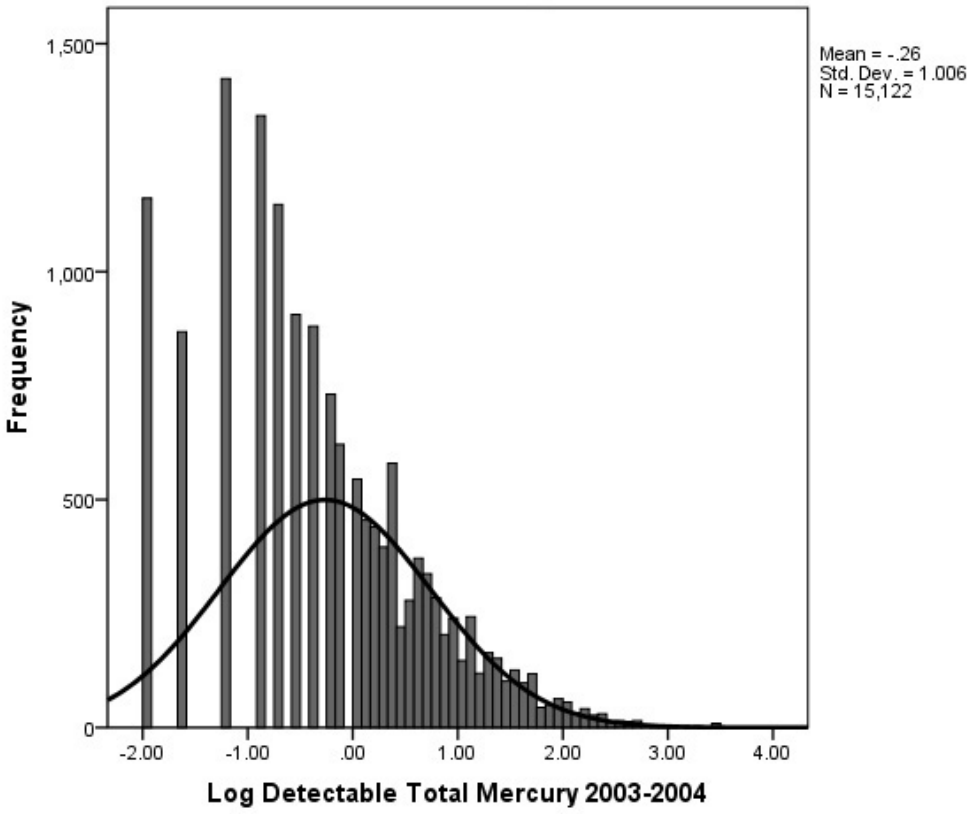


FIGURE 9

HISTOGRAM OF LOG DETECTABLE INORGANIC MERCURY (1999-2000)

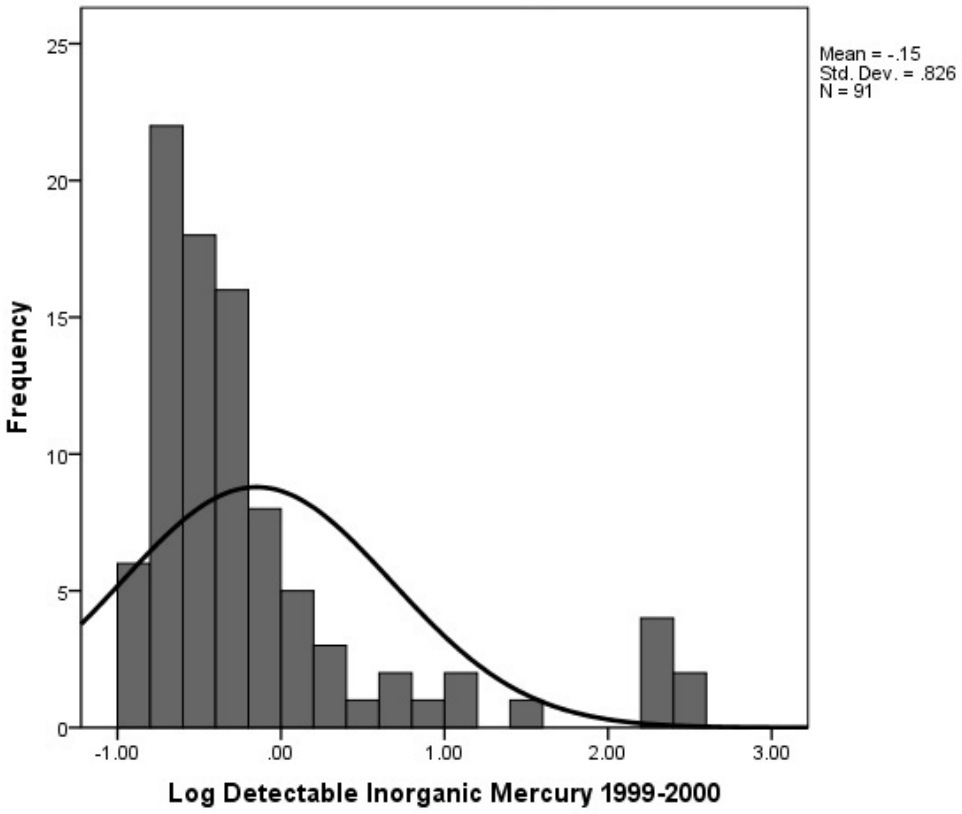


FIGURE 10

HISTOGRAM OF LOG DETECTABLE INORGANIC MERCURY (2001-2002)

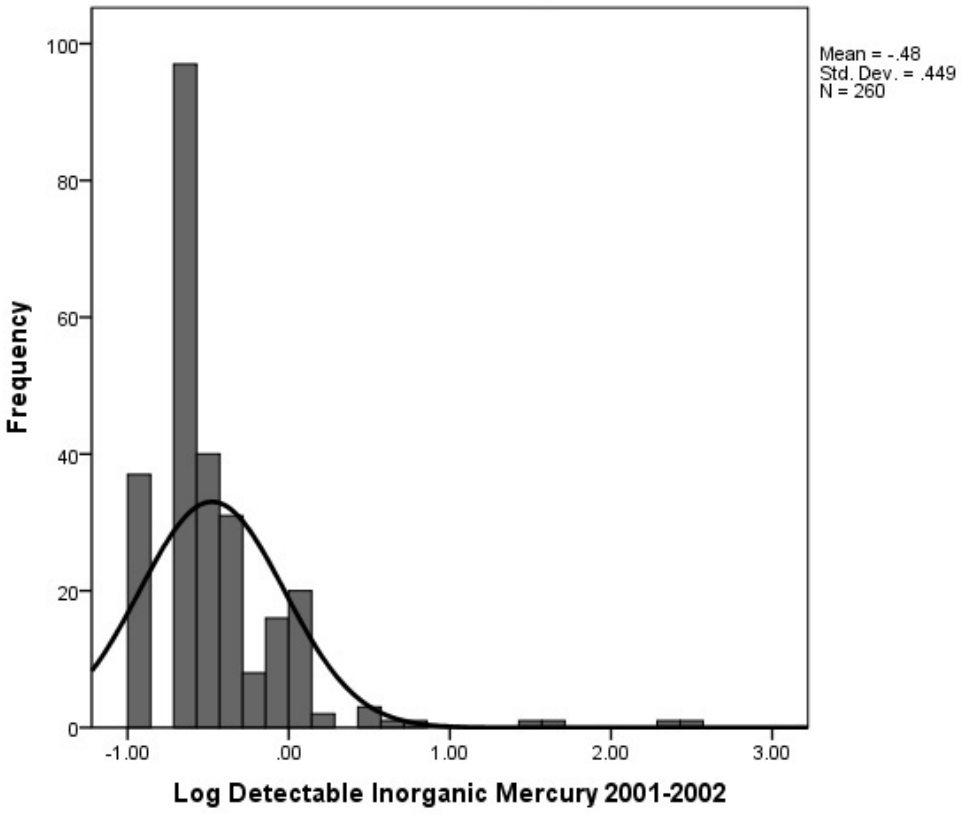


FIGURE 11

HISTOGRAM OF LOG DETECTABLE INORGANIC MERCURY (2003-2004)

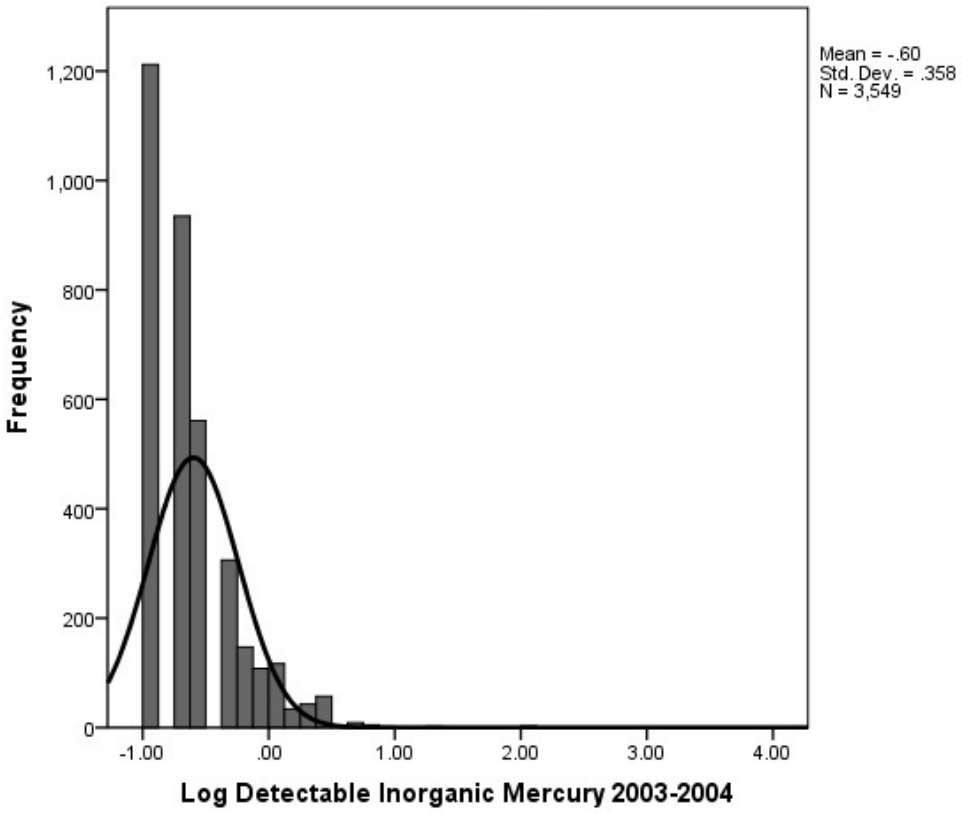


FIGURE 12

HISTOGRAM OF LOG DETECTABLE LIPID-ADJUSTED PCB 118

(1999-2000)

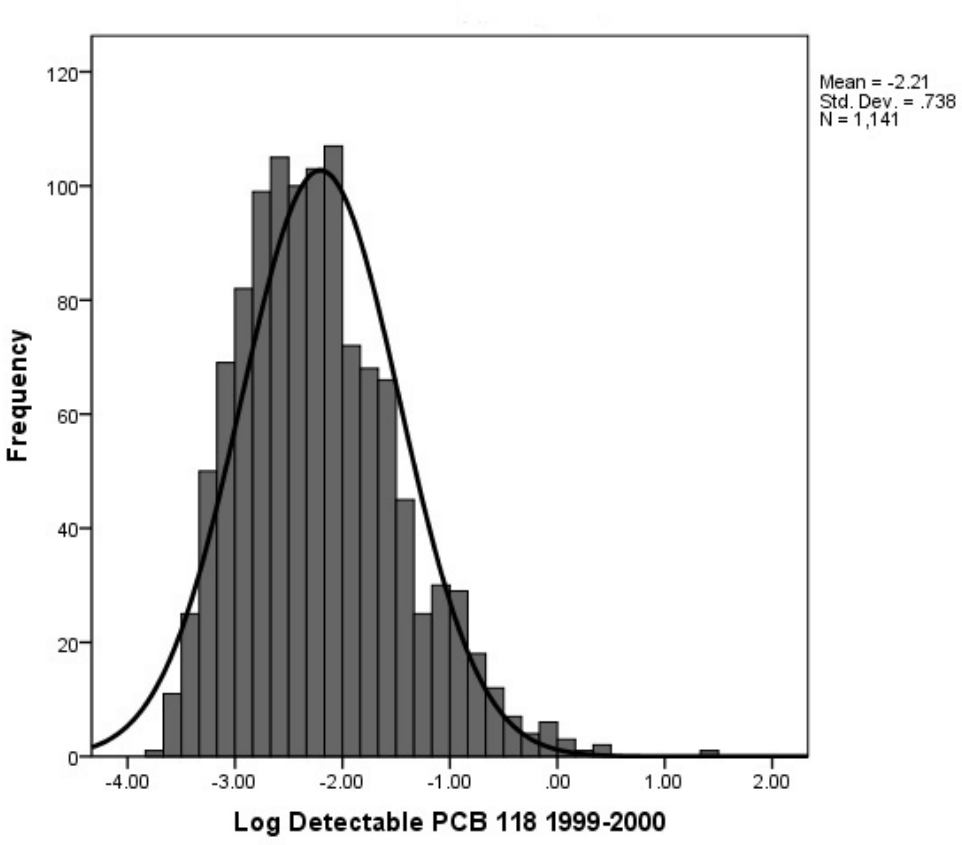


FIGURE 13

HISTOGRAM OF LOG DETECTABLE LIPID-ADJUSTED PCB 118

(2001-2002)

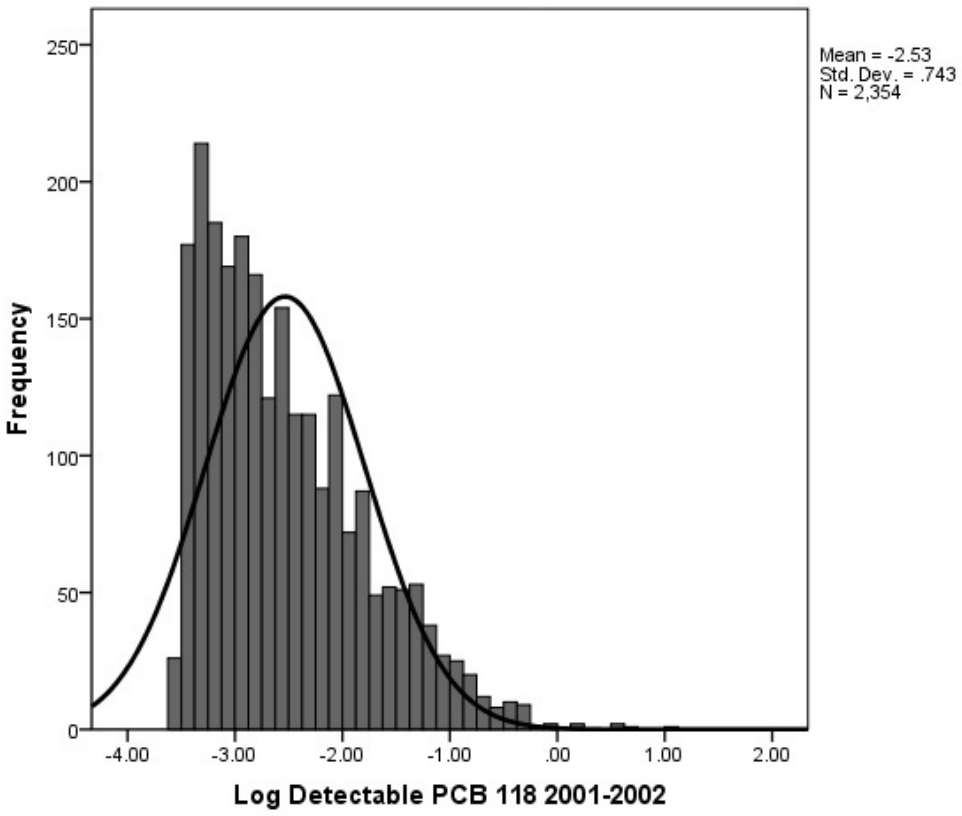


FIGURE 14

HISTOGRAM OF LOG DETECTABLE LIPID-ADJUSTED PCB 118

(2003-2004)

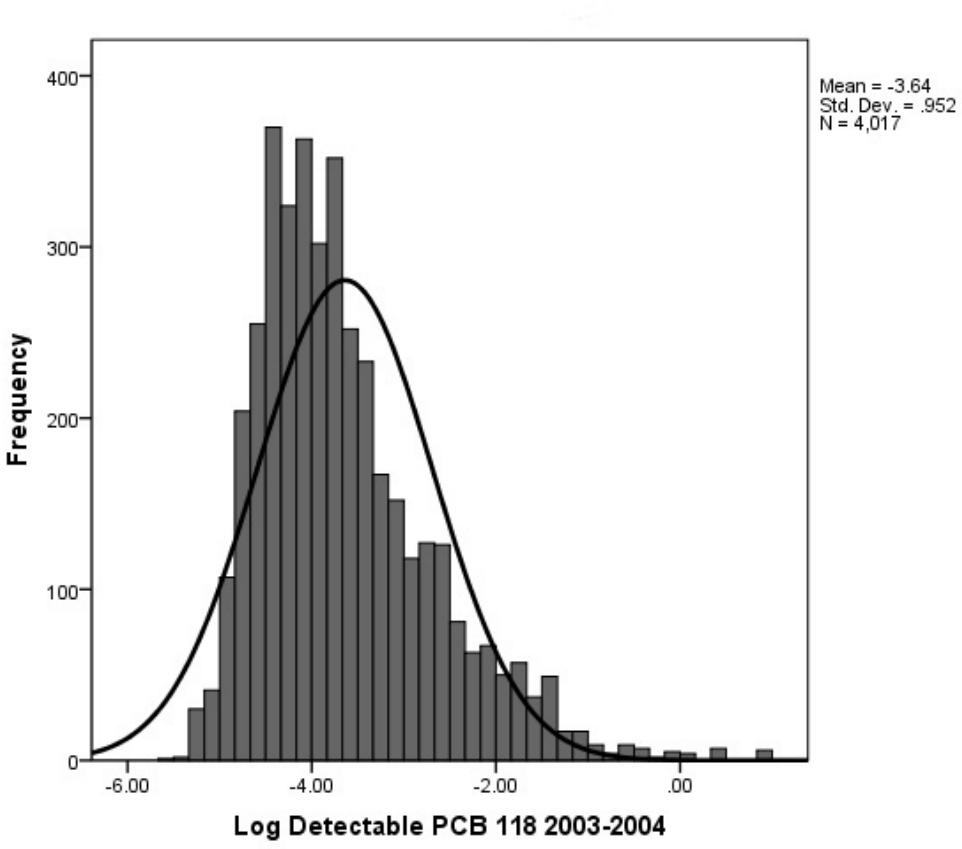


FIGURE 15

HISTOGRAM OF LOG DETECTABLE LIPID-ADJUSTED PCB 138

(1999-2000)

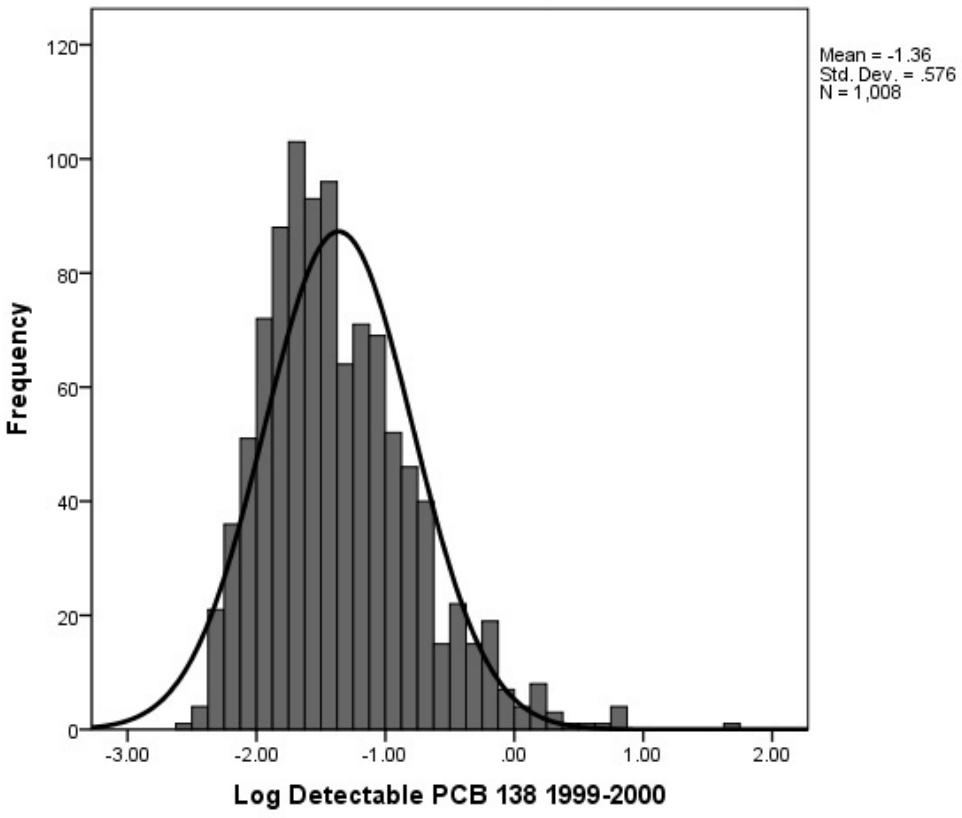


FIGURE 16

HISTOGRAM OF LOG DETECTABLE LIPID-ADJUSTED PCB 138

(2001-2002)

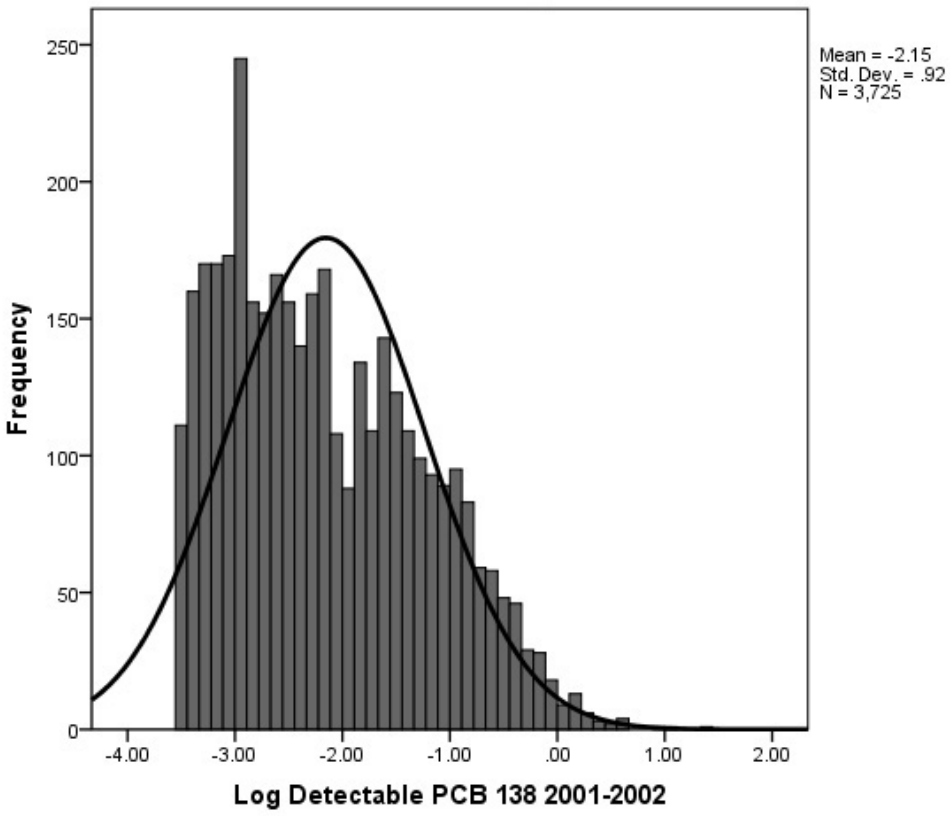


FIGURE 17

HISTOGRAM OF LOG DETECTABLE LIPID-ADJUSTED PCB 138

(2003-2004)

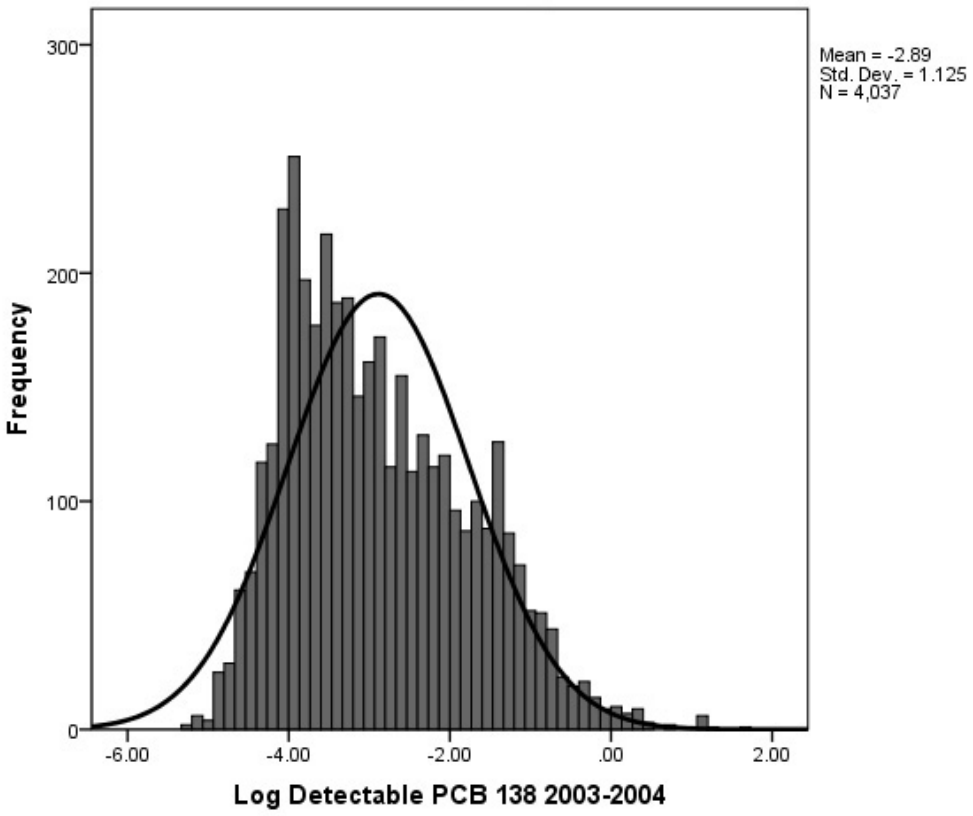


FIGURE 18

HISTOGRAM OF LOG DETECTABLE LIPID-ADJUSTED PCB 153

(1999-2000)

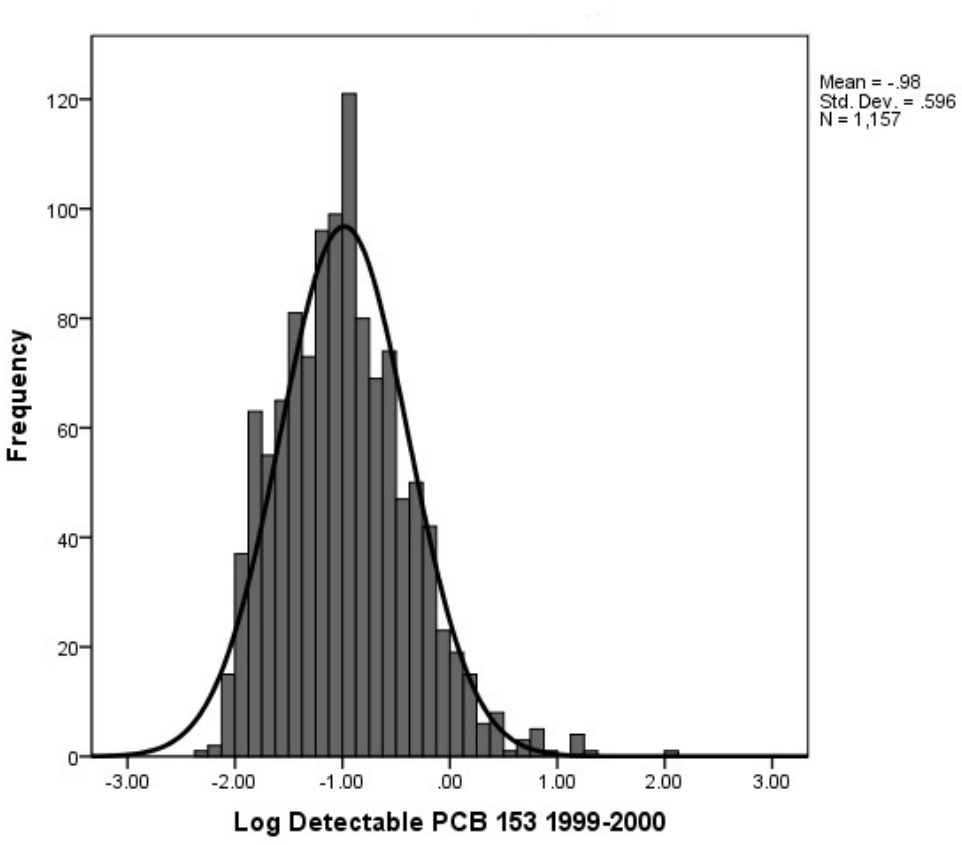


FIGURE 19

HISTOGRAM OF LOG DETECTABLE LIPID-ADJUSTED PCB 153

(2001-2002)

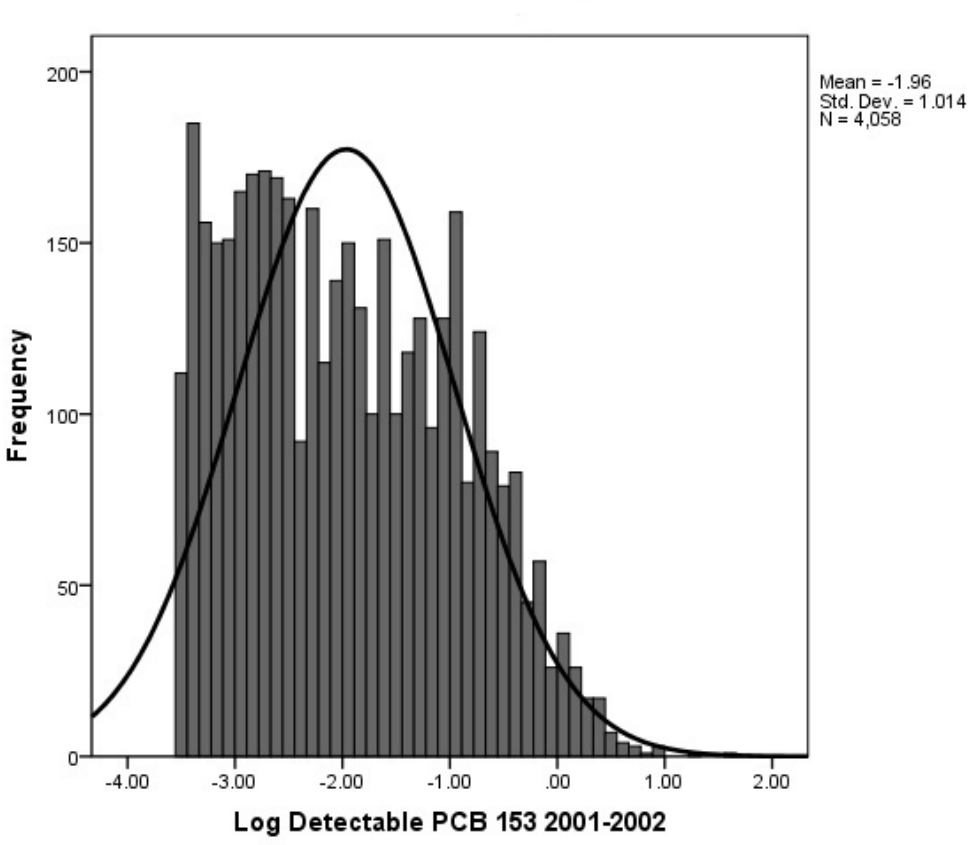


FIGURE 20

HISTOGRAM OF LOG DETECTABLE LIPID-ADJUSTED PCB 153

(2003-2004)

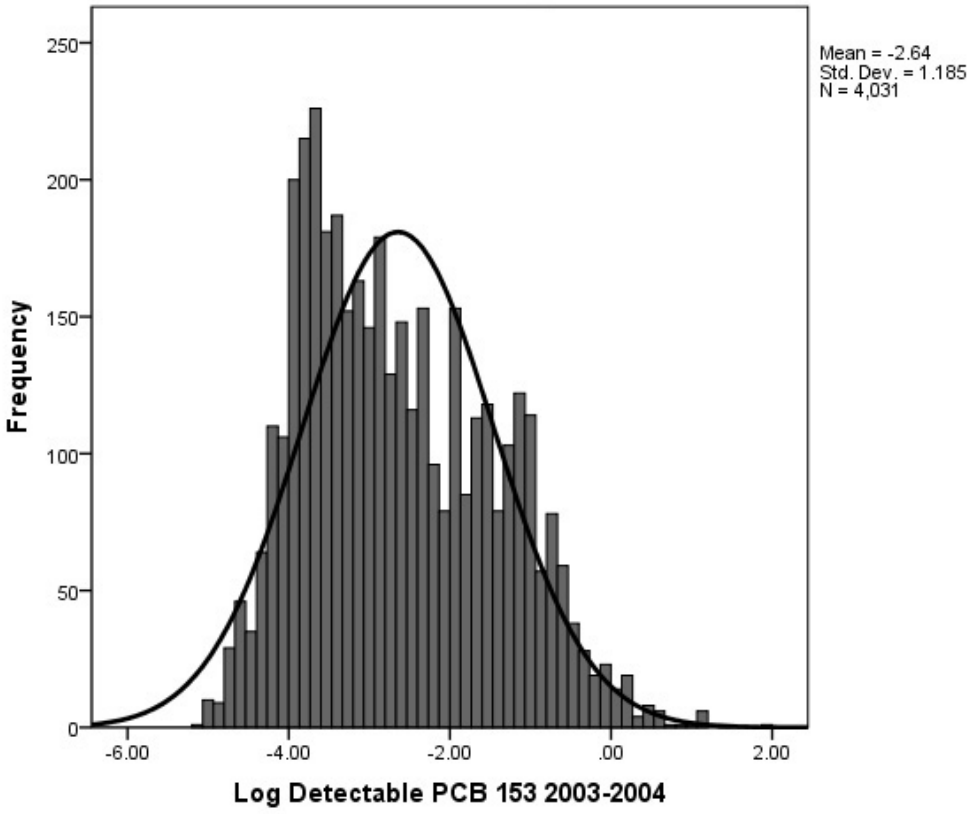


FIGURE 21

HISTOGRAM OF LOG DETECTABLE LIPID-ADJUSTED PCB 180

(1999-2000)

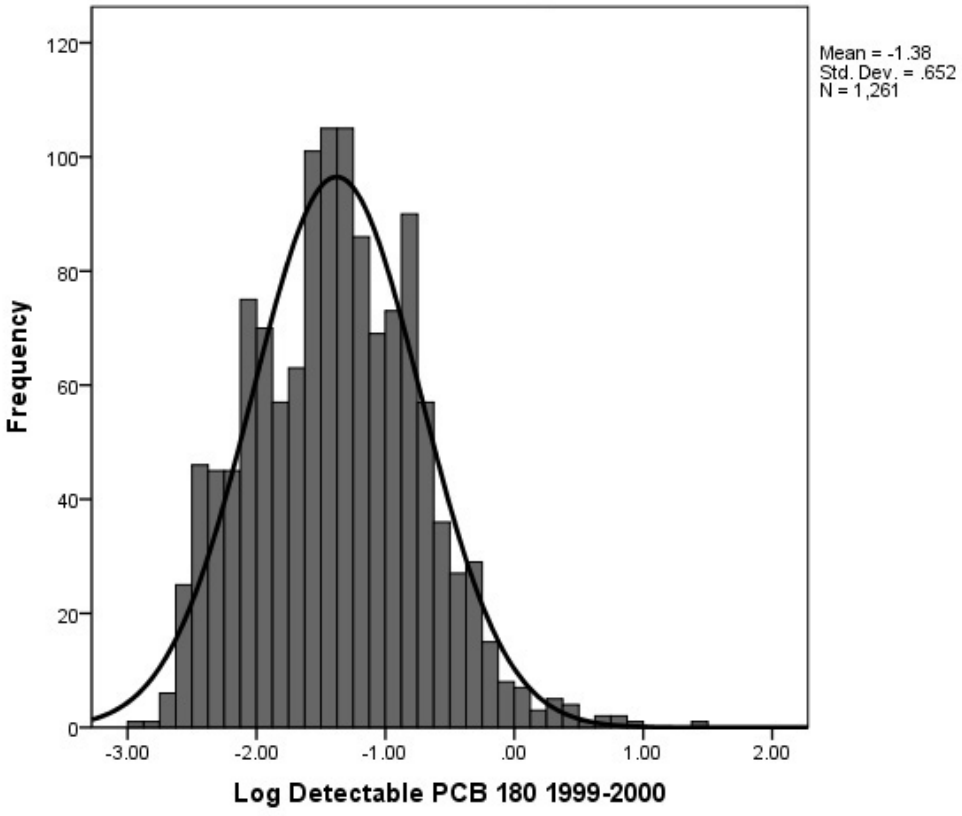


FIGURE 22

HISTOGRAM OF LOG DETECTABLE LIPID-ADJUSTED PCB 180

(2001-2002)

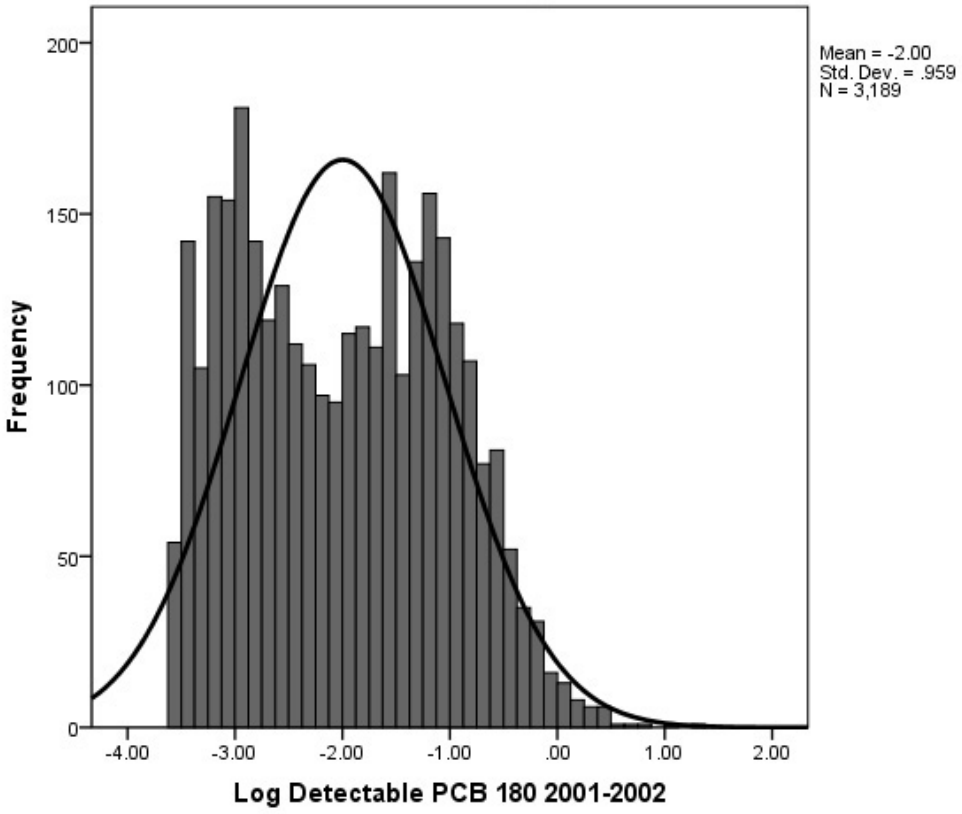


FIGURE 23

HISTOGRAM OF LOG DETECTABLE LIPID-ADJUSTED PCB 180

(2003-2004)

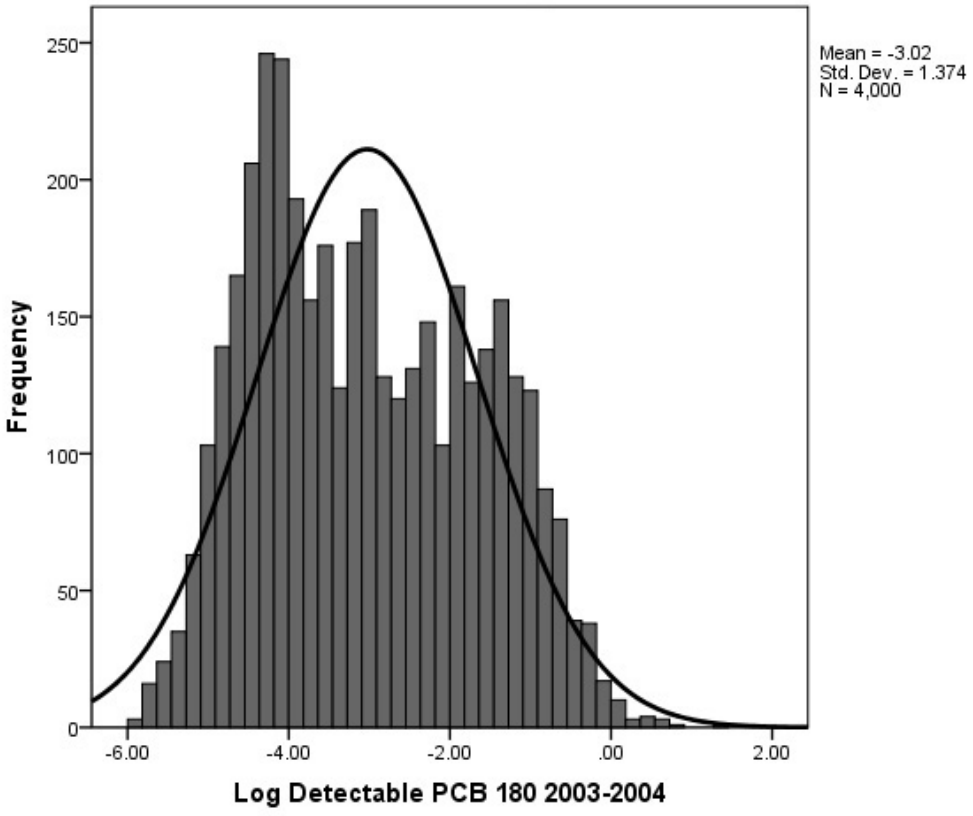


FIGURE 24

FREQUENCY DISTRIBUTION OF LEAD

PRIOR TO LOGARITHMIC TRANSFORMATION (1999-2004)

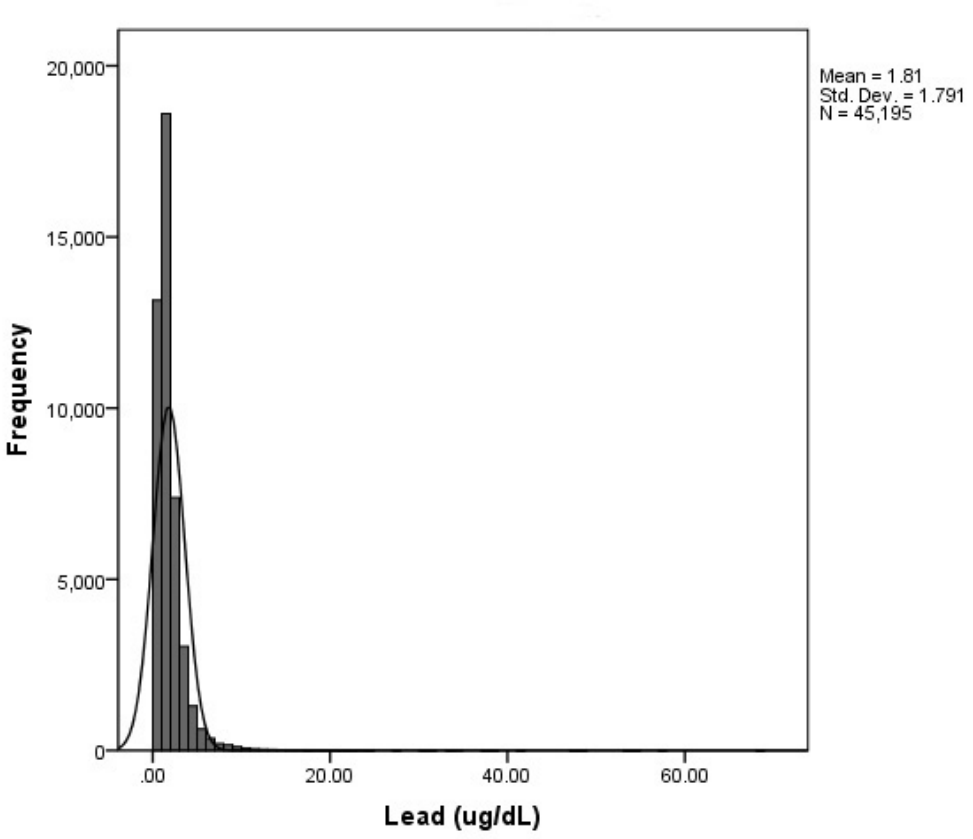


FIGURE 25

FREQUENCY DISTRIBUTION OF METHYLMERCURY

PRIOR TO LOGARITHMIC TRANSFORMATION (1999-2004)

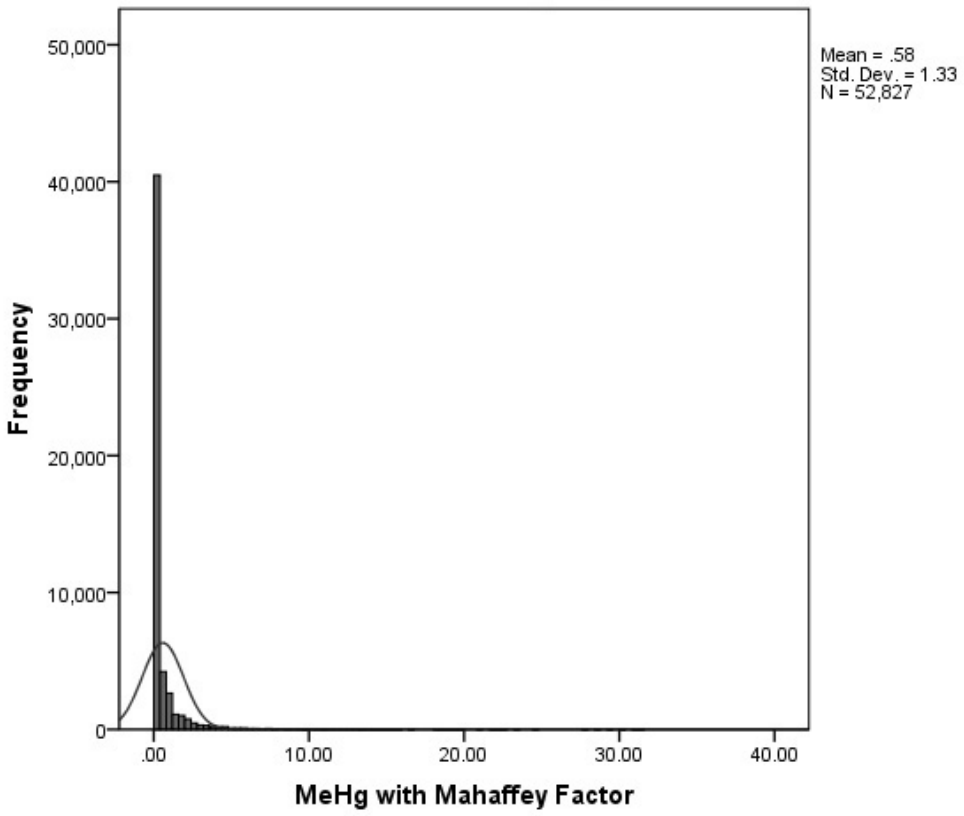


FIGURE 26

FREQUENCY DISTRIBUTION OF LIPID-ADJUSTED SUM OF PCBS

PRIOR TO LOGARITHMIC TRANSFORMATION (1999-2004)

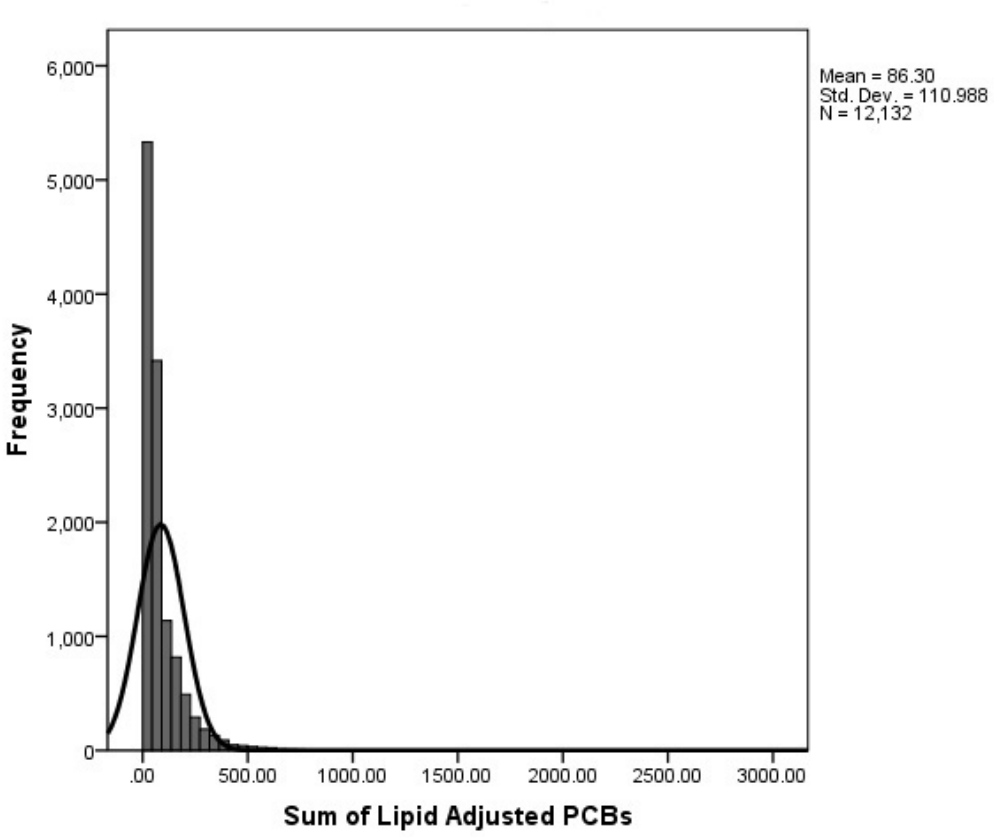


FIGURE 27

FREQUENCY DISTRIBUTION OF LEAD

POST LOGARITHMIC TRANSFORMATION (1999-2004)

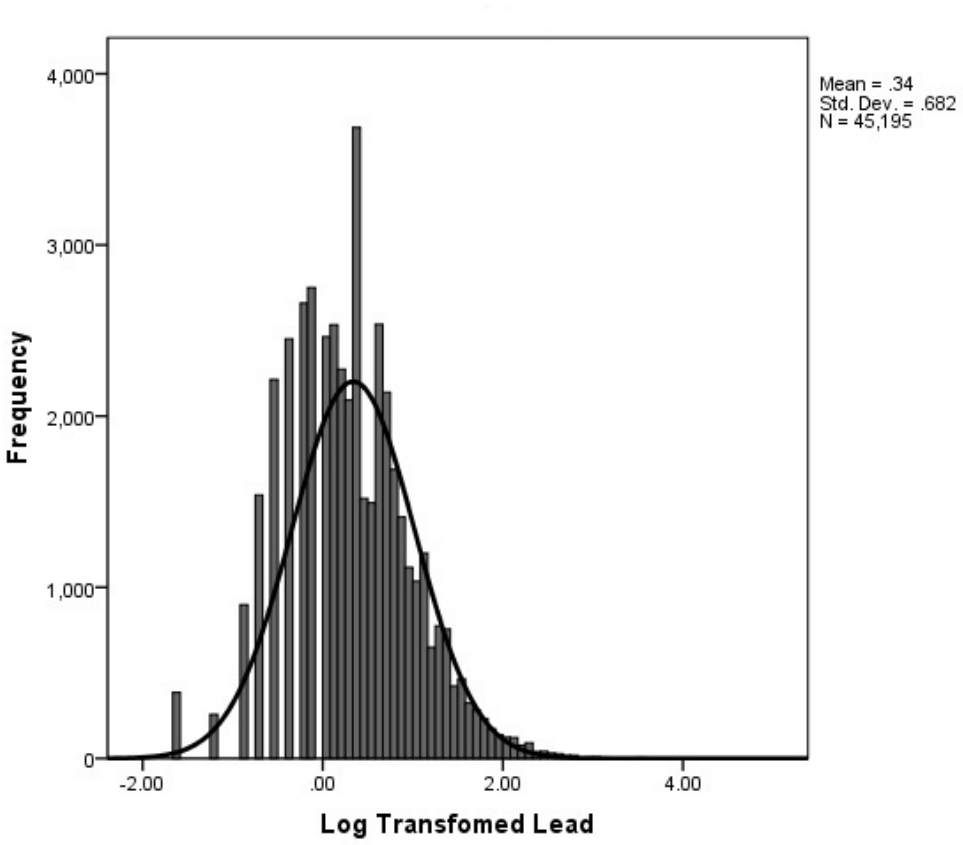


FIGURE 28

FREQUENCY DISTRIBUTION OF METHYLMERCURY

POST LOGARITHMIC TRANSFORMATION (1999-2004)

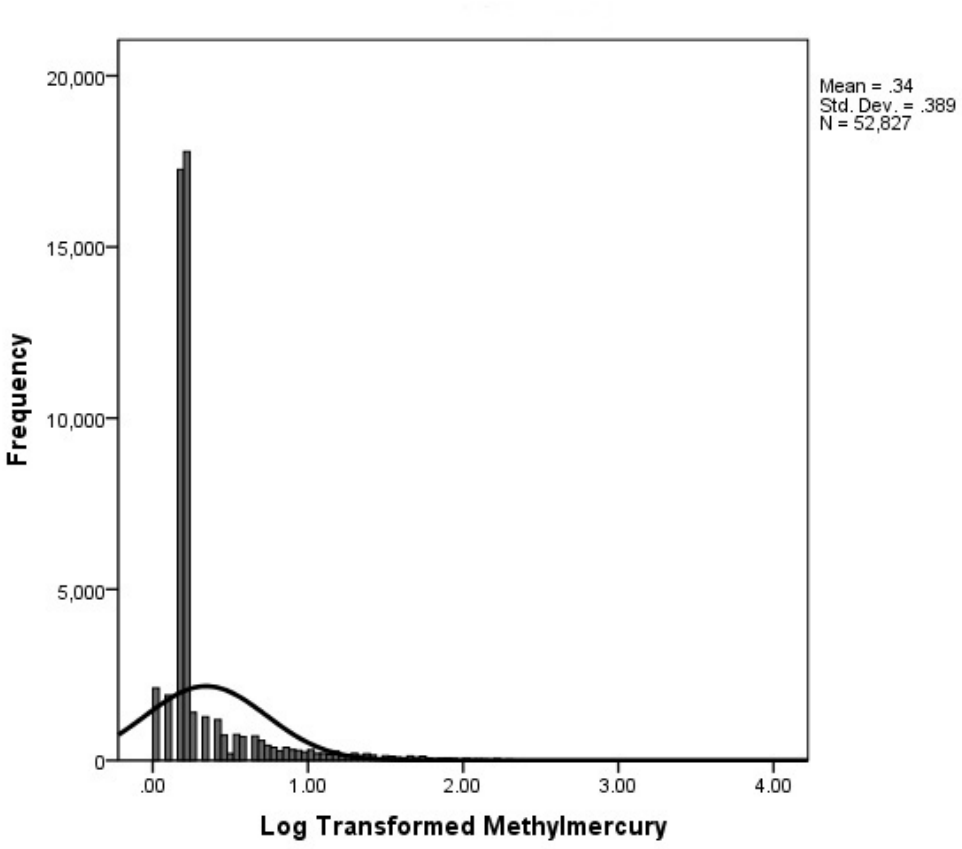


FIGURE 29

FREQUENCY DISTRIBUTION OF LIPID-ADJUSTED PCBS

POST LOGARITHMIC TRANSFORMATION (1999-2004)

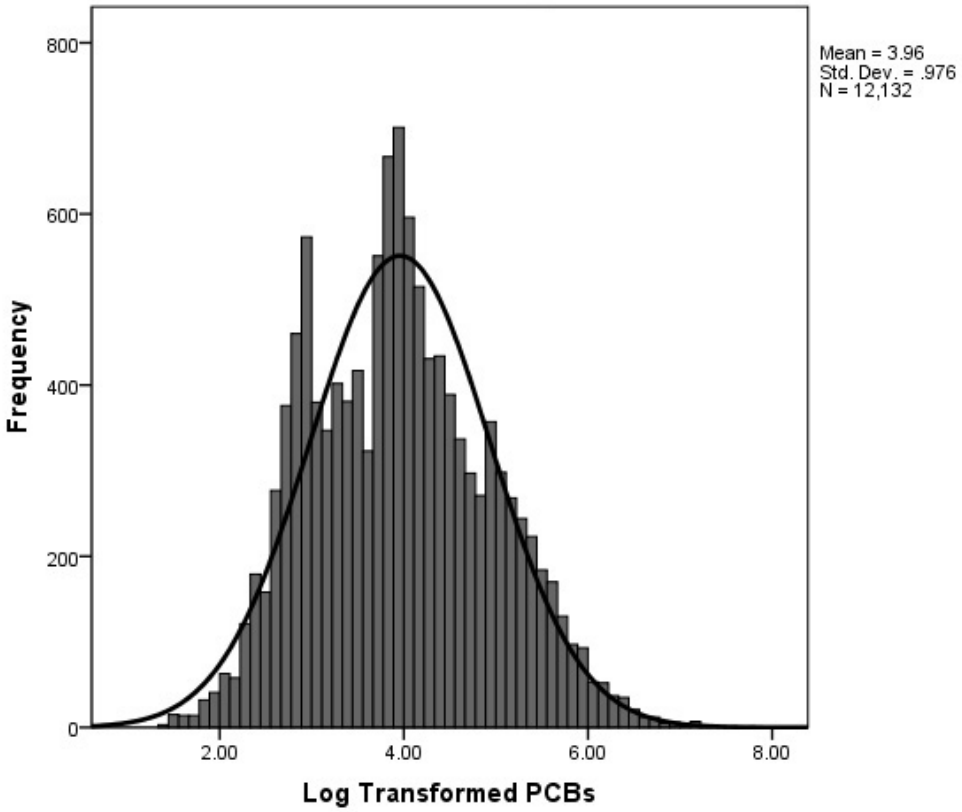


FIGURE 30

HISTOGRAM OF LOG DETECTABLE COTININE (1999-2000)

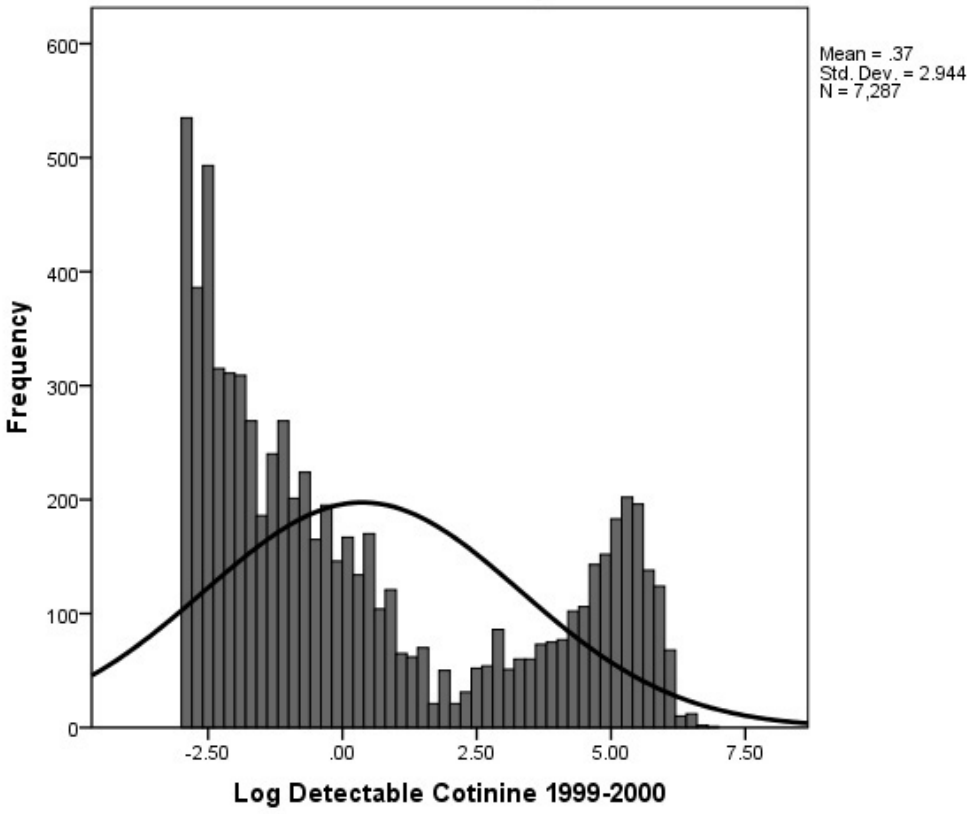


FIGURE 31

HISTOGRAM OF LOG DETECTABLE COTININE (2001-2002)

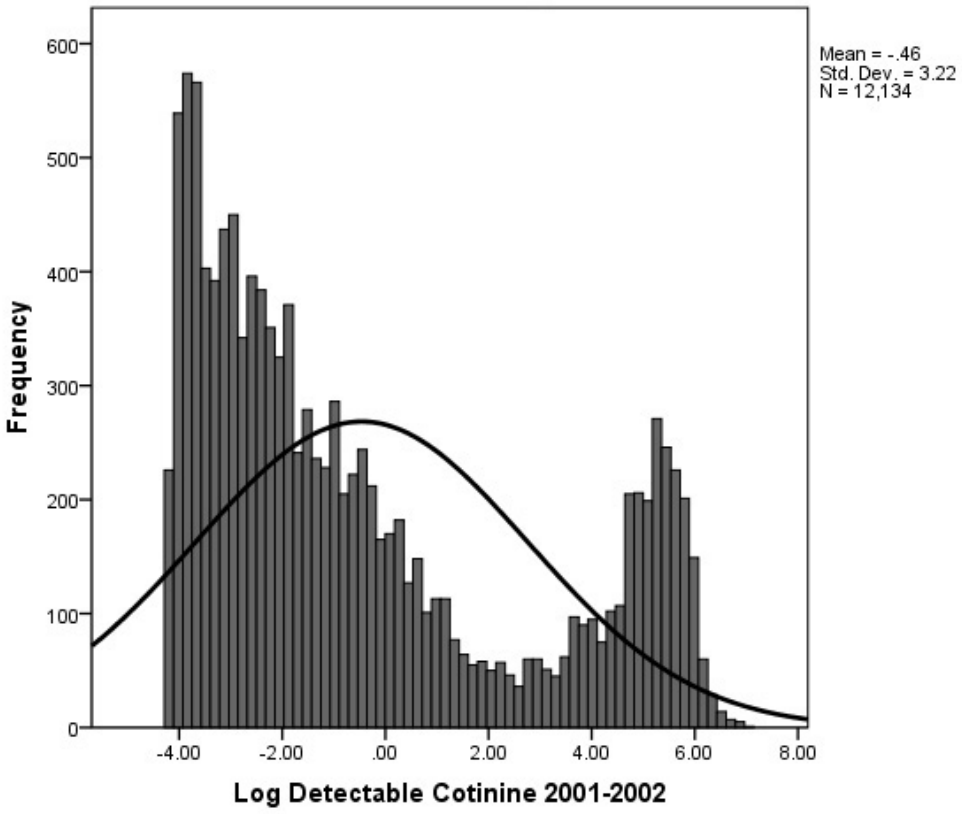


FIGURE 32

HISTOGRAM OF LOG DETECTABLE COTININE (2003-2004)

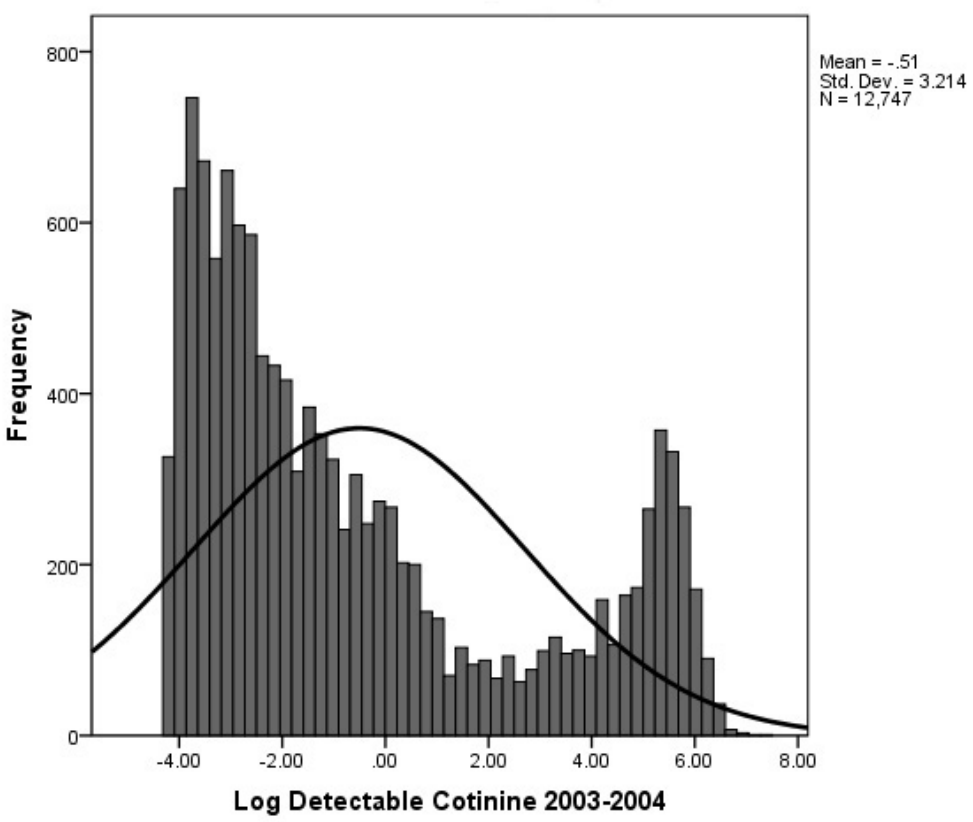


FIGURE 33

FREQUENCY DISTRIBUTION OF COTININE

PRIOR TO LOGARITHMIC TRANSFORMATION (1999-2004)

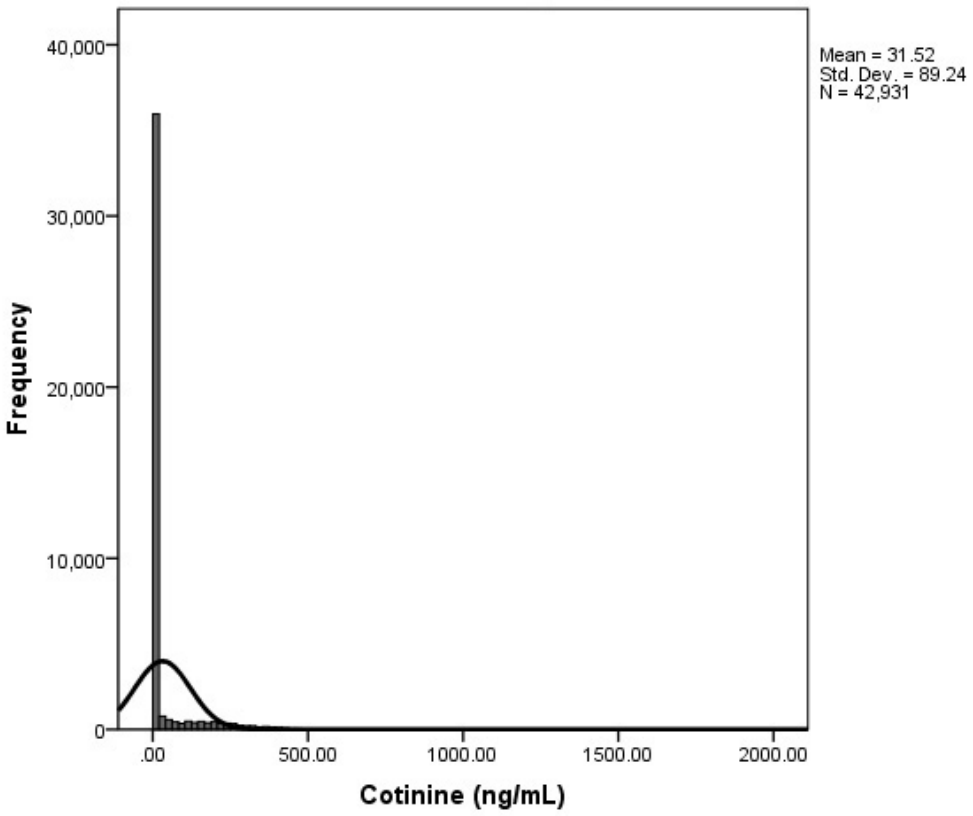


FIGURE 34

FREQUENCY DISTRIBUTION OF COTININE

POST LOGARITHMIC TRANSFORMATION (1999-2004)

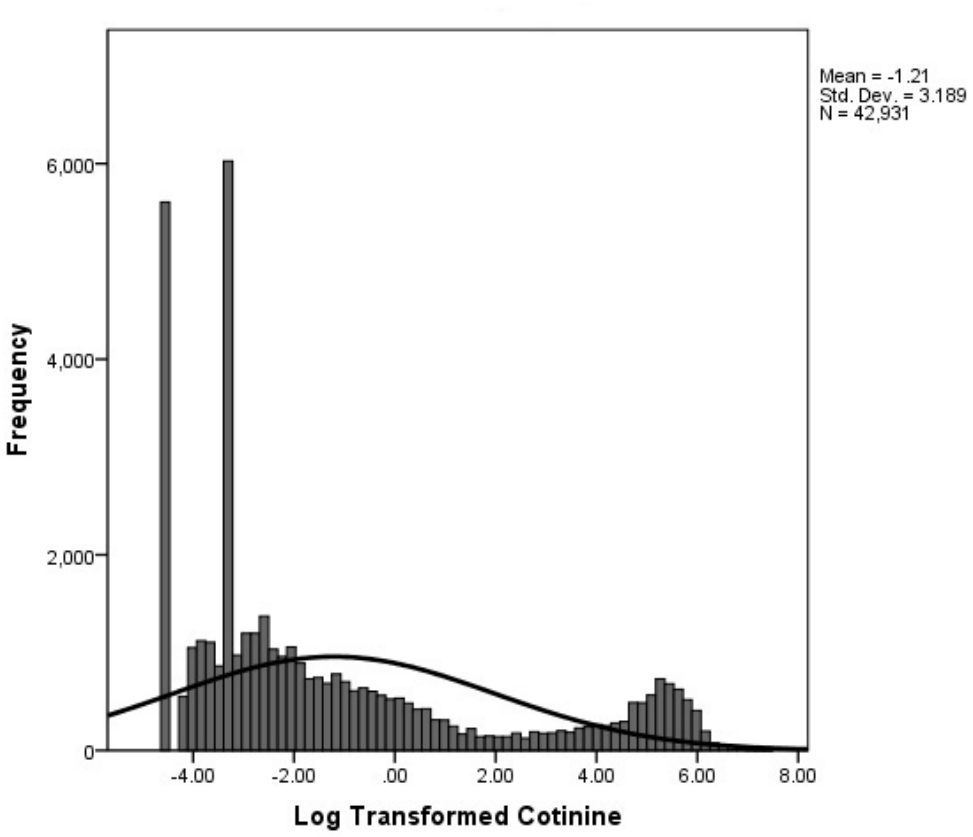


Figure 37. Exposure as Outcome with Four Categories: Percentage of Childbearing-Aged Females in U.S. with Xenobiotic Blood Levels At or Above the Geometric Mean (weighted data 1999-2004)

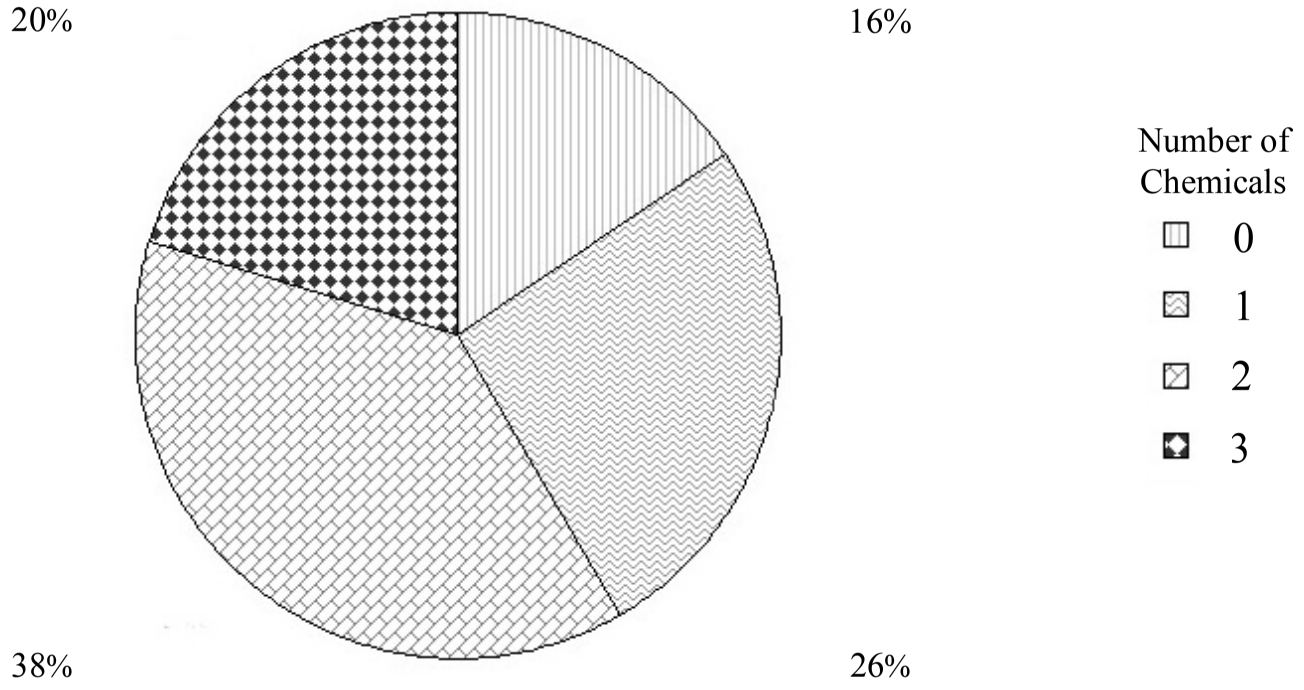


Figure 38. Exposure as Outcome with Four Categories: Percentage of Pregnant Childbearing-Aged Females in U.S. with Xenobiotic Blood Levels At or Above the Geometric Mean (weighted data 1999-2004)

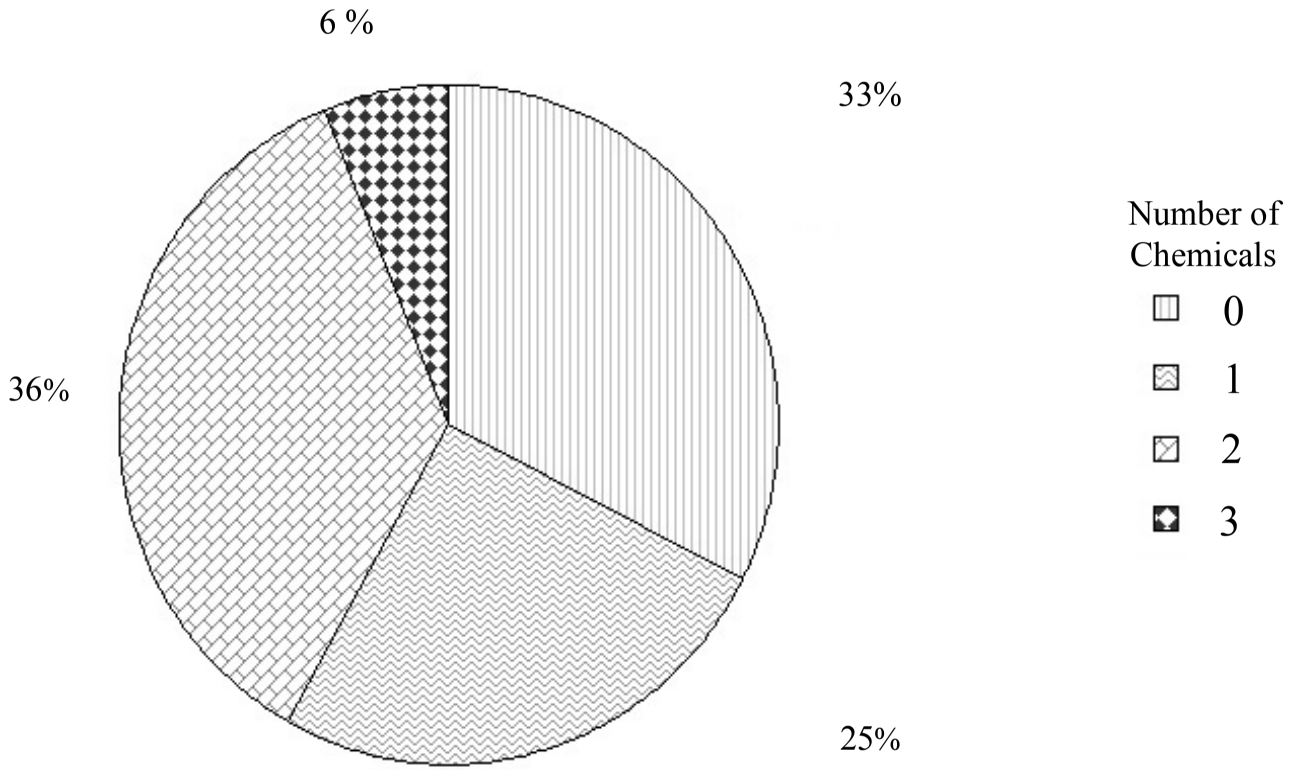


Figure 39. Exposure as Outcome with Two Categories: Percentage of Childbearing-Aged Females in U.S. with Xenobiotic Blood Levels At or Above the Geometric Mean (weighted data 1999-2004)

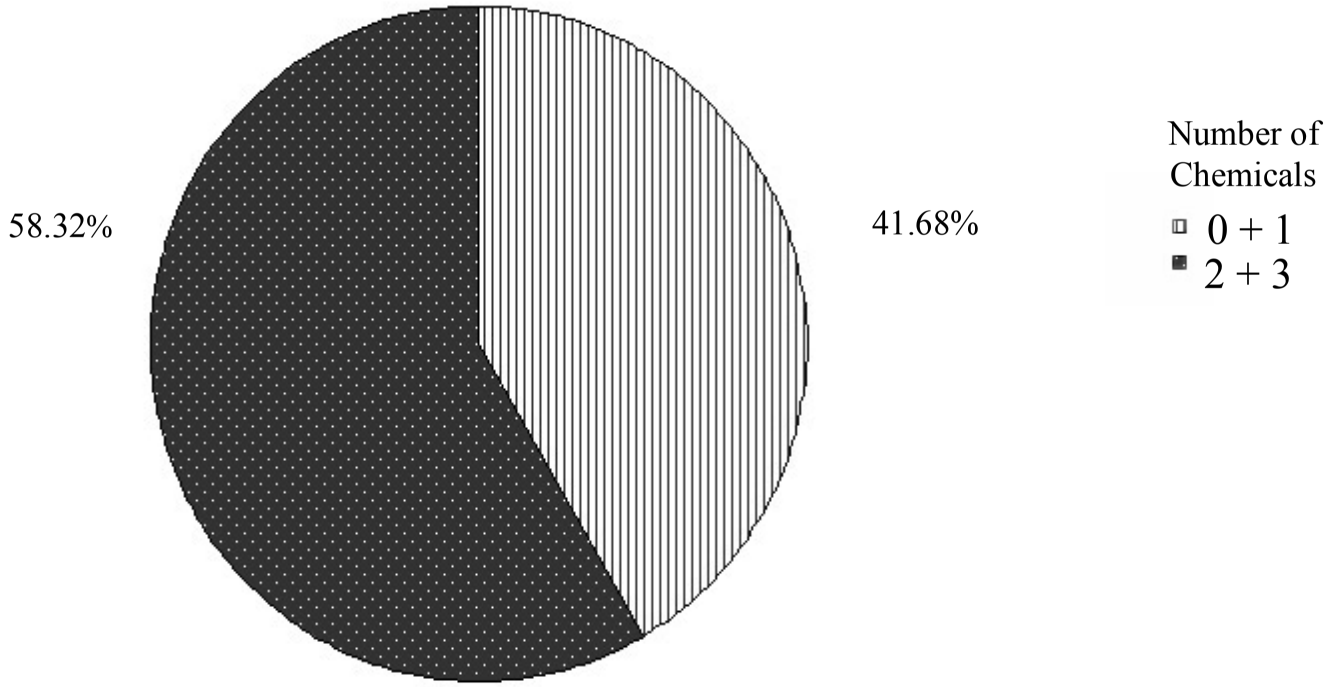


Figure 40. Exposure as Outcome with Two Categories: Percentage of Pregnant Childbearing-Aged Females in U.S. with Xenobiotic Blood Levels At or Above the Geometric Mean (weighted data 1999-2004)

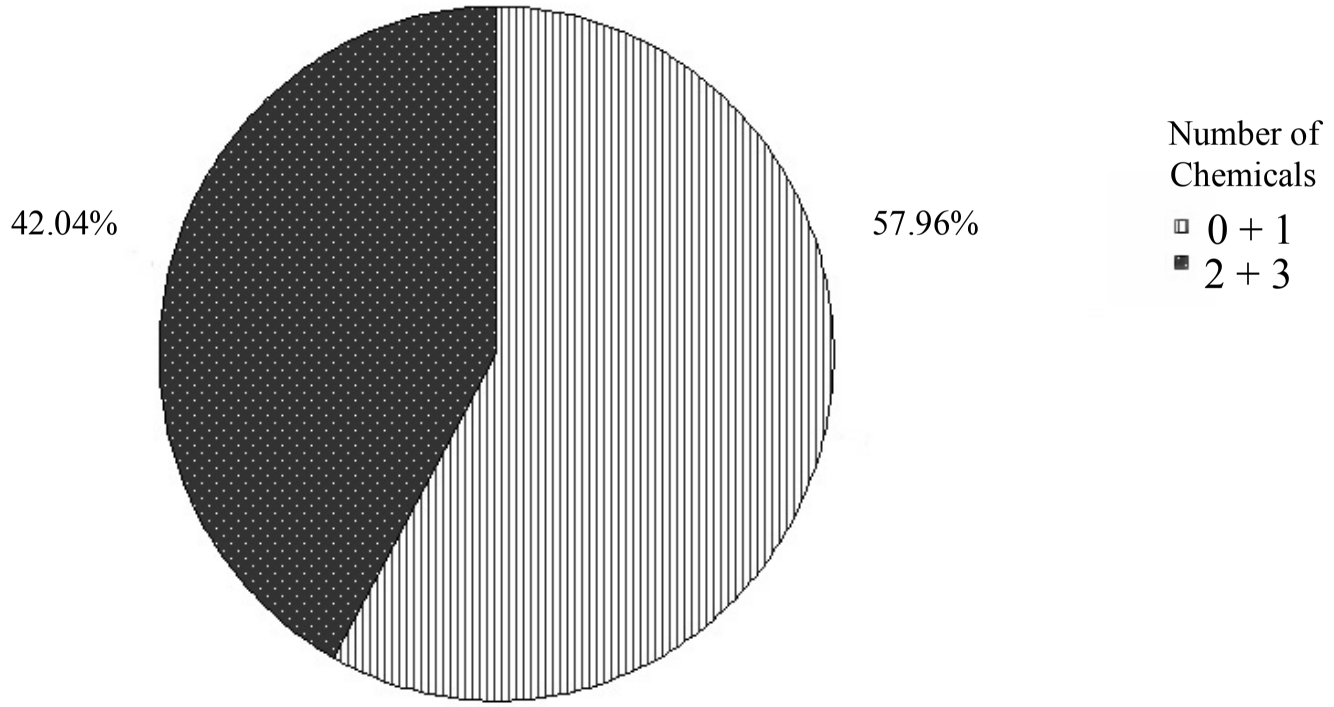


Figure 41. Odds of Childbearing-Aged Females in U.S. Having Two or More Xenobiotic Blood Levels At or Above the Geometric Mean Based on Fish Meals Eaten Past 30 Days (1999-2004)

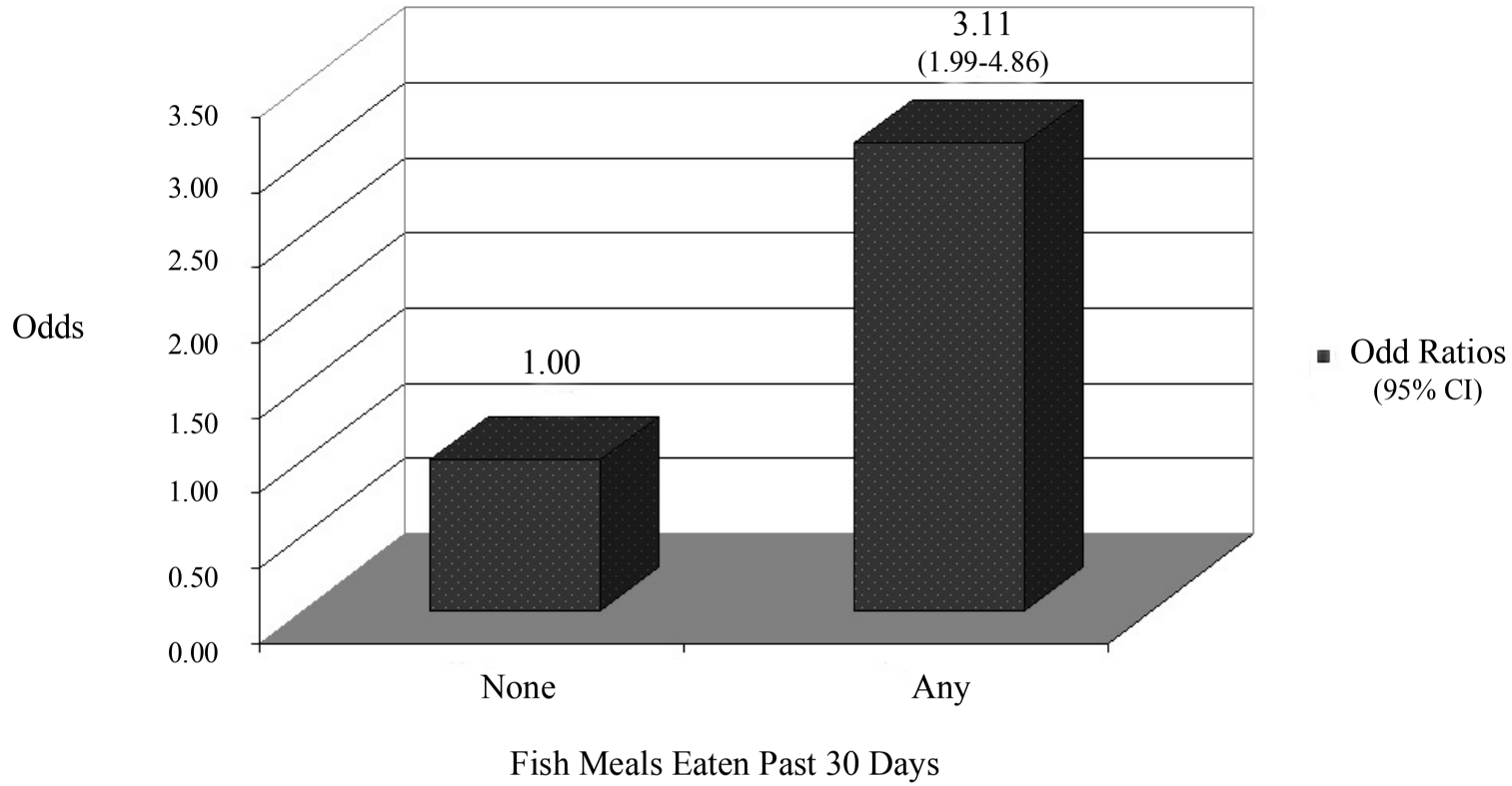


Figure 42. Odds of Childbearing-Aged Females in U.S. Having Two or More Xenobiotic Blood Levels At or Above the Geometric Mean Based on Age (1999-2004)

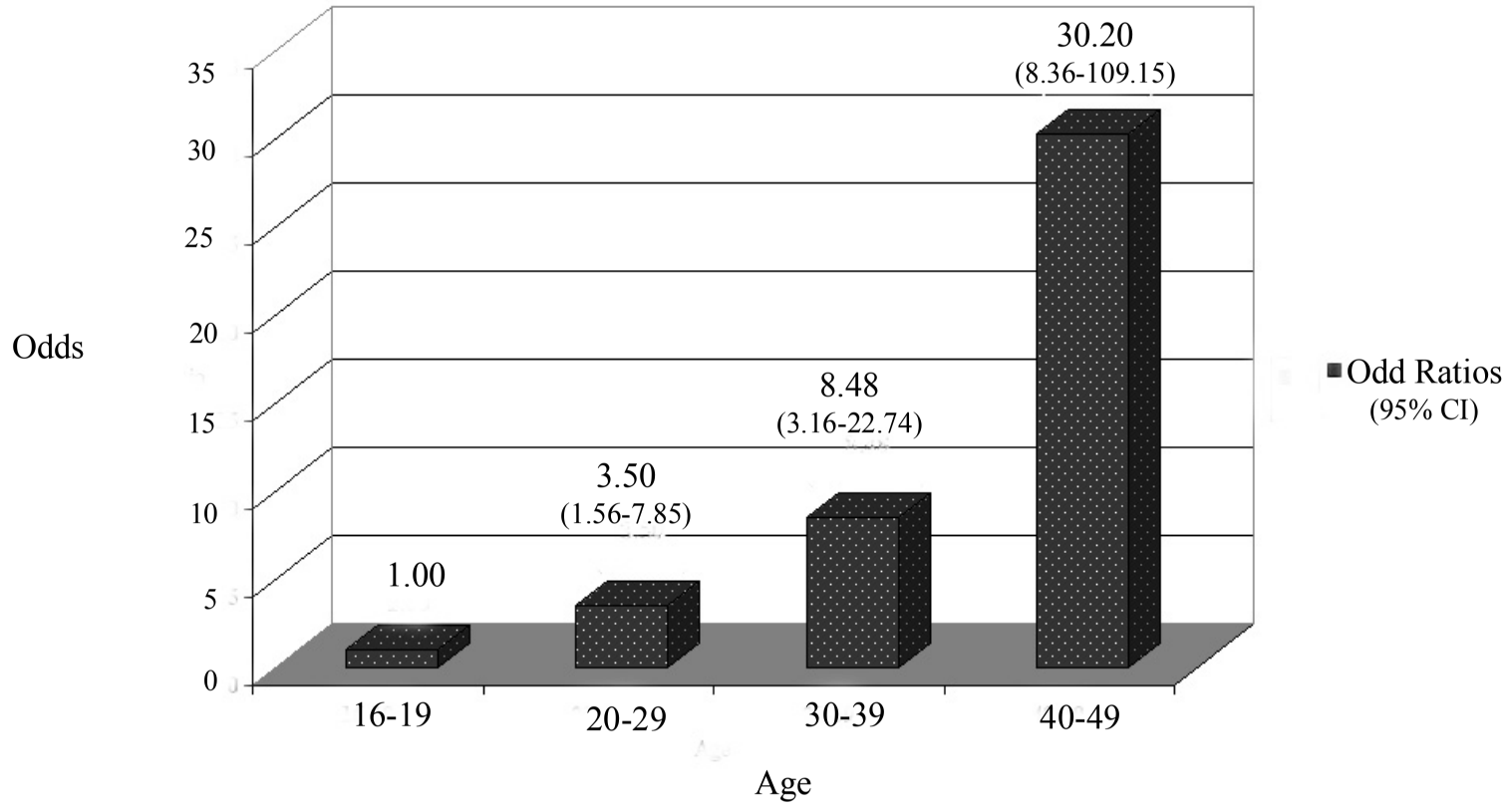


Figure 43. Odds of Childbearing-Aged Females in U.S. Having Two or More Xenobiotic Blood Levels At or Above the Geometric Mean Based on Breastfeeding (1999-2004)

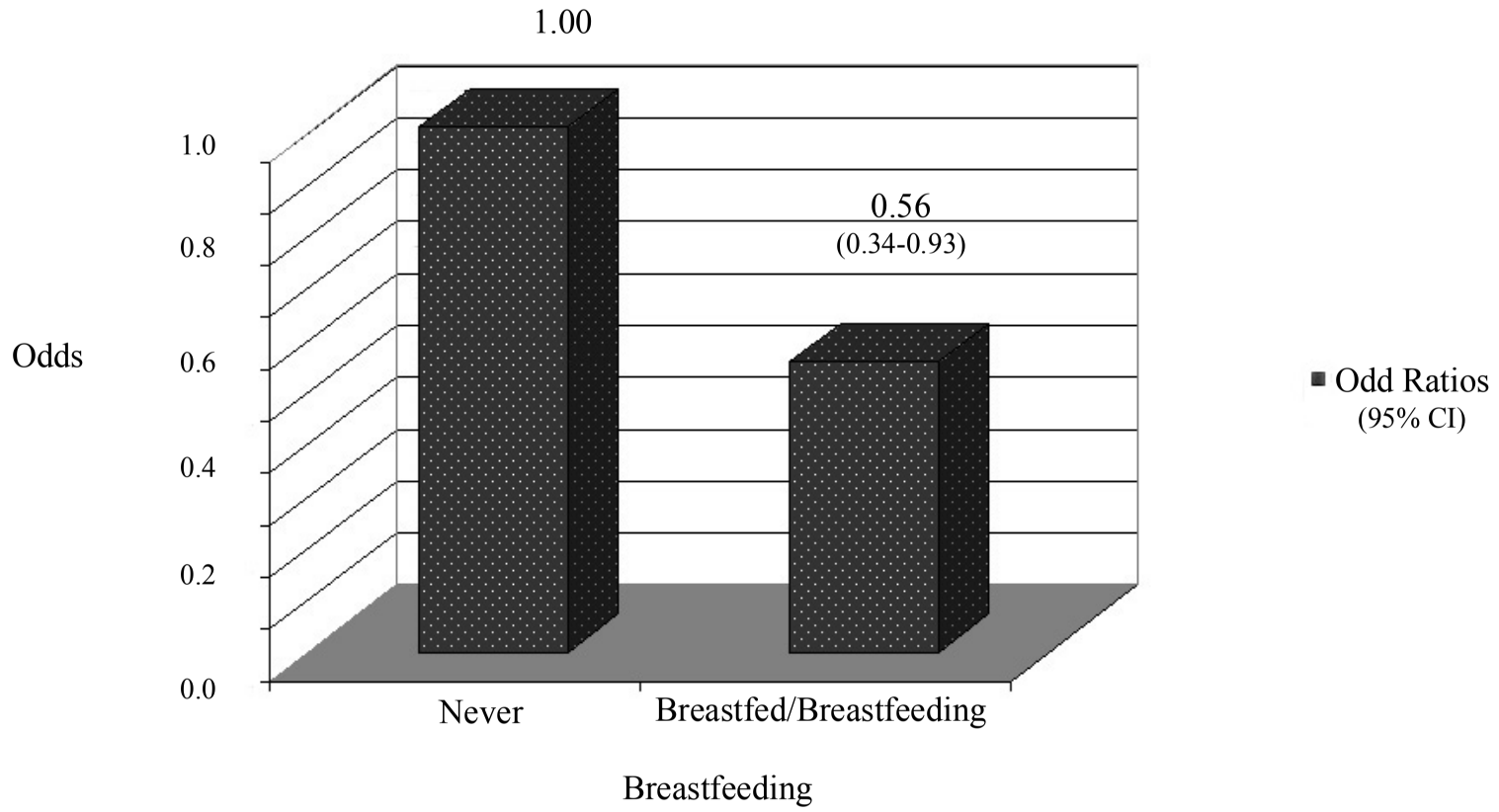
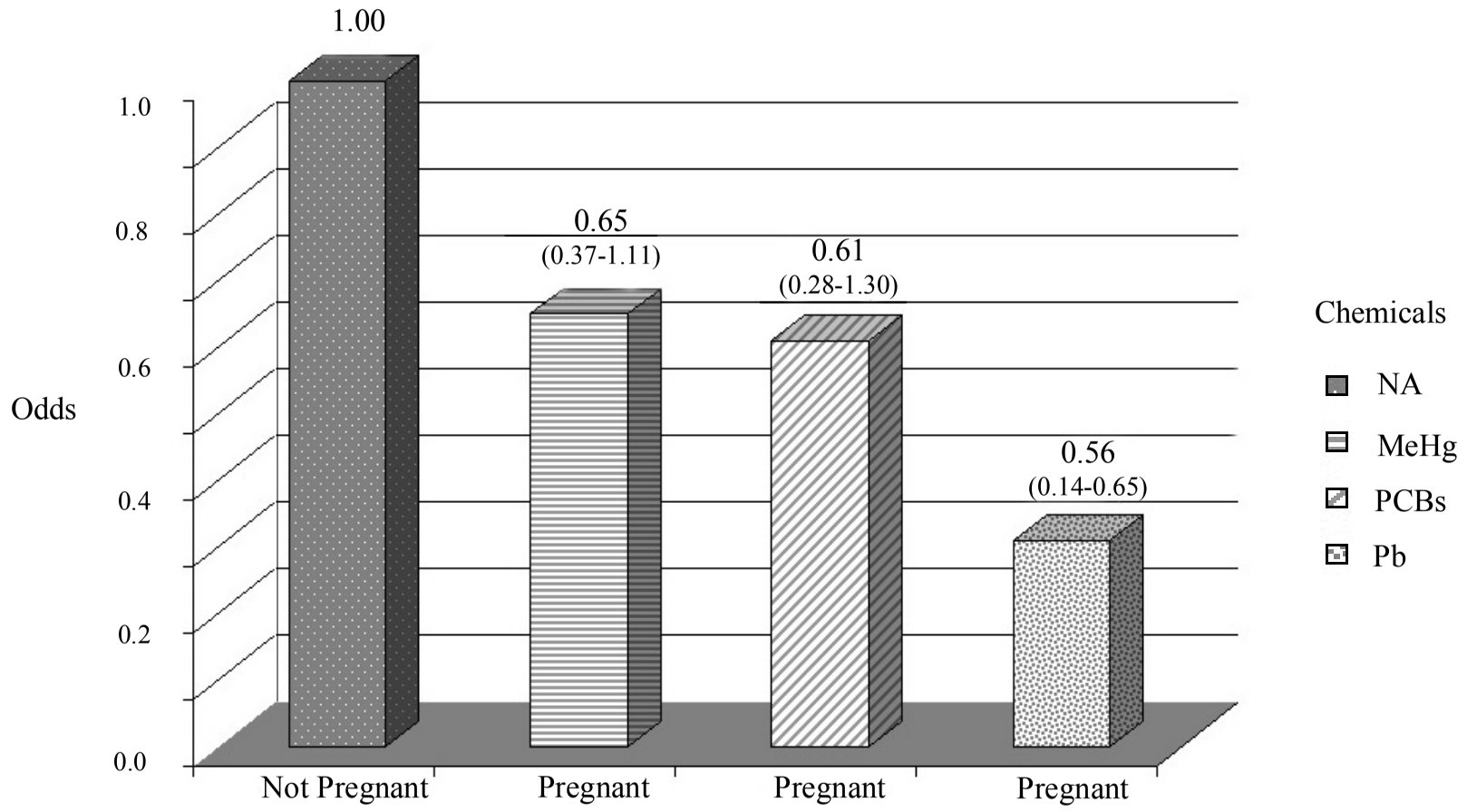


Figure 44. Odds of Childbearing-Aged Females in U.S. Having Two or More Xenobiotic Blood Levels At or Above the Geometric Mean by Pregnancy Status (1999-2004)



BIBLIOGRAPHY

Abdullah, M., & Al-Salamah, S. M. (2009). Impact of co-morbidity on outcome among acute non-traumatic surgical patients: evaluation of Charlson Co-Morbidity Index. *Saudi Medical Journal, 30*(2), 228-233. PMID 19198711

Abe-Kim, J., Okzaki, S., & Goto, S. G. (2001). Unidimensional Versus Multidimensional Approaches to the Assessment of Acculturation for Asian American Populations. *Cultural Diversity and Ethnic Minority Psychology, 7*(3), 232-246. doi:10.1037//1099-9809.7.3.232

Aburto, N. J., Ramirez-Zea, M., Neufeld, L. M., & Flores-Ayala, R. (2009, September). Some Indicators of Nutritional Status Are Associated with Activity and Exploration in Infants at Risk for Vitamin and Mineral Deficiencies. *The Journal of Nutrition*, *139*(9), 1751-1757. PMID 19640971. doi:10.3945/jn.108.100487

Acevedo-Garcia, D., & Osypuk, T. L. (2008). Residential Segregation and Health – The Complexity of Modeling Separate Social Contexts (Peer Commentary on the article "Effect of racial residential segregation on black infant mortality" by Hearst, Oakes & Johnson, 2008, December 1). *American Journal of Epidemiology, 168*(11), 1255-1258. PMID 18974060. doi:10.1093/aje/kwn290

Acevedo-Garcia, D., Soobader, M.-J., & Berkman, L. F. (2005, January). The Differential Effect of Foreign-Born Status on Low Birth Weight by Race/Ethnicity and Education. *Pediatrics, 115*(1), 20-30. PMID 15629963. doi:10.1542/peds.2004-1306

Aday, L. A. (2001). *At Risk in America: The Health and Health Care Needs of Vulnerable Populations in the United States*. San Francisco, CA: Jossey-Bass Publishers

Addison, G., Beamish, M., Hales, C., Hodgkins, M., Jacobs, A., & Llewellin, P. (1972, April). An immunoradiometric assay for ferritin in the serum of normal patients and patients with iron deficiency and iron overload. *Journal of Clinical Pathology, 25*(4), 326-329. PMID 5063755

Adgate, J. L., Barteková, A., Raynor, P. C., Griggs, J. G., Ryan, A. D., Acharya, B. R., … Bonds, M. D. (2009, January). Detection of organophosphate pesticides using a prototype liquid crystal monitor. *Journal of Environmental Monitoring, 11*(1), 49-55. PMID 19137139. doi:10.1039/b806954a

Adgate, J. L., Clayton, C. A., Quackenboss, J. J., Thomas, K. W., Whitmore, R. W., Pellizzari, E. D., … Sexton, K. (2000). Measurement of multi-pollutant and multi-pathway exposures in a probability-based sample of children: practical strategies for effective field studies. *Journal of Exposure Analysis and Environmental Epidemiology, 10,* 650-661. PMID 11138657

Adgate, J. L., & Sexton, K. (2001). Emerging Issues: Children's Exposure to Pesticides in Residential Settings. In R. I. Kreiger (Ed.), *Handbook of Pesticide Toxicology* (2nd ed., Vol. I, pp. 887-904). San Diego, CA: Academic Press

Adibi, J. J., Hauser, R., Williams, P. L., Whyatt, R. M., Thaker, H. M., Nelson, H., … Bhat, H. K. (2009, April 23). Placental biomarkers of phthalate effects on mRNA transcription: application in epidemiologic research. *Environmental Health, 8*, 1-20. PMID 19389254. doi:10.1186/1476-069X-8-20

Adinolfi, M. (1985, August). The development of the human blood-CSF-brain barrier. *Developmental Medicine and Child Neurology, 27*(4), 532-537. PMID 4029526

Adler, N. E., & Newman, K. (2002, March-April). Socioeconomic disparities in health: pathways and policies. *Health Affairs, 21*(2), 60-76. PMID 11900187

Aelion, C., Davis, H., McDermott, S., & Lawson, A. (2009, March 15). Soil metal concentrations and toxicity: associations with distances to industrial facilities and implications for human health. *Science and the Total Environment, 407*(7), 2216-2223. PMID 19155049. doi:10.1016/j.scitotenv.2008.11.033

Agency for Toxic Substances and Disease Registry. (1997, September). *Toxicological Profile for Chlorpyrifos*. Atlanta, GA: Author

Agency for Toxic Substances and Disease Registry. (1999, March). *Toxicological Profile for Mercury.* Atlanta, GA: Author

Agency for Toxic Substances and Disease Registry. (2000, November). *Toxicological Profile for Polychlorinated Biphenyls (PCBs)*. Atlanta, GA: Author

Agency for Toxic Substances and Disease Registry. (2001, February). *Guidance Manual for the Assessment of Joint Toxic Action of Chemical Mixtures*. Atlanta, GA: Author

Agency for Toxic Substances and Disease Registry. (2004, May). *Interaction Profile for Persistent Chemicals Found in Breast Milk (Chlorinated Dibenzo-p-Dioxins, Hexachlorobenzene, p,p'-DDE, Methylmercury, and Polychlorinated Biphenyls)*. Atlanta, GA: Author

Agency for Toxic Substances and Disease Registry. (2006, August). *Interaction Profile for: Chlorpyrifos, Lead, Mercury and Methylmercury*. Atlanta, GA: Author

Agency for Toxic Substances and Disease Registry. (2007, August). *Toxicological Profile for Lead*. Atlanta, GA: Author

Agnew, J. (2001). Scientific Foundations of Occupational and Environmental Health Nursing Practice. In M. K. Salazar (Ed.), *Core Curriculum for Occupational & Environmental Health Nursing* (2nd ed., pp. 111-145). Philadelphia, PA: W. B. Saunders Company

Agocs, M., & Clarkson, T. W. (1995). ATSDR Case Studies in Environmental Medicine: Mercury Toxicity. In A. M. Pope & D. P. Rall (Eds.), *Environmental Medicine: Integrating a Missing Element into Medical Education* (pp. 450-472). Washington, D.C.: National Academy Press

Agocs, M., Clarkson, T. W., Ambre, J., Becker, C., Borak, J., Cannella, J., … Wummer, B. A. (1992, December). Mercury Toxicity. Agency for Toxic Substance and Disease Registry. *American Family Physician, 46*(6), 1731-1741. PMID 1456196

Agocs, M., Etzel, R. A., Parrish, R. G., Paschal, D. C., Campagna, P. R., Cohen, D. S., … Hesse, J. L. (1990, October 18). Mercury exposure from interior latex paint. *The New England Journal of Medicine, 323*(16), 1096-1101. PMID 2215577

Ahamed, M., & Siddiqui, M. K. (2007, August). Environmental lead toxicity and nutritional factors. *Clinical Nutrition, 26*(4), 400-408. PMID 17499891. doi:10.1016/j.clnu.2007.03.010

Ahern, N. (2007). *Resiliency in adolescent college students* (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 3256904)

Ainsworth, B. E., Haskell, W. L., Whitt, M. C., Irwin, M. L., Swartz, A. M., Strath, S. J., … Leon, A. S. (2000, September). Compendium of physical activities: an update of activity codes and MET intensities. *Medicine & Science in Sports & Exercise, 32*(9 Supplement), S498-S504. PMID 10993420

Aitio, A. (1988). Biological Monitoring. In T. W. Clarkson, L. Friberg, G. F. Nordberg, & P. R. Sager (Eds.), *Biological Monitoring of Toxic Metals* (pp. 75-81). New York, NY: Plenum Press

Albers, J. W., Garabrant, D. H., Mattsson, J. L., Burns, C. J., Cohen, S. S., Sima, C., … Berent, S. (2007, May). Dose-Effect Analyses of Occupational Chlorpyrifos Exposure and Peripheral Nerve Electrophysiology. *Toxicological Sciences, 97*(1), 196-204. PMID 17324952. doi:10.1 093/toxscilkfm028

Albers, J. W., Garabrant, D. H., Schweitzer, S. J., Garrison, R. P., Richardson, R. J., & Berent, S. (2004). The effects of occupational exposure to chlorpyrifos on the peripheral nervous system: a prospective cohort study. *Occupational and Environmental Medicine, 61,* 201-211. PMID 14985514. doi:10.1136/oem.2003.008847

Albert, M., & Blacker, D. (2006, April). Mild Cognitive Impairment and Dementia. *Annual Review of Clinical Psychology, 2*, 379-388. doi:10.1146/annurev.clinpsy.1.102803.144039

Albertini, R., Bird, M., Doerrer, N., Needham, L., Robison, S., Sheldon, L., & Zenick, H. (2006, November). The Use of Biomonitoring Data in Exposure and Human Health Risk Assessments. *Environmental Health Perspectives, 114*(11), 1755-1762. PMID 17107864. doi:10.1289/ehp.9056

Alegria, M. (2009, October). The challenge of acculturation measures: what are we missing? (Peer commentary on the article "Defining and measuring acculturation: a systematic review of public health studies with Hispanic populations in the United States" by Thomson & Hoffman-Goetz). *Social Science & Medicine, 69*(7), 996-998. PMID 19664868. doi:10.1016/j.socscimed.2009.07.006

Alessio, L., Apostoli, P., & Crippa, M. (1995, May). Influence of individual factors and personal habits on the levels of biological indicators of exposure. *Toxicology Letters, 77*(1-3), 93-103. PMID 7618174. doi:10.1016/0378-4274(95)03277-0

Alfthan, G. V. (1997). Toenail mercury concentration as a biomarker of methylmercury exposure. *Biomarkers, 2,* 233-238

Ali, R., Olden, K., & Xu, S. (2008, October). Community-based participatory research: a vehicle to promote public engagement for environmental health in China. *Environmental Health Perspectives, 116*(10), 1281-1284. PMID 18941566. doi:10.1289/ehp.11399

Allen, B. C., Hack, C. E., & Clewell, H. J. (2007, August). Use of Markov Chain Monte Carlo Analysis with a Physiologically-Based Pharmacokinetic Model of Methylmercury to Estimate Exposures in U.S. Women of Childbearing Age. *Risk Analysis, 27*(4), 947-959. PMID 17958503. doi:10.1111/j.1539-6924.2007.00934.x

Allen, R. H., Mage, D. T., Gondy, G., Kodali, A., Christensen, C., Coble, J., & Stewart, P. (2006, March-April). Investigation of Job-Related Pesticide Exposure in the Third National Health and Nutrition Examination Survey. *Archives of Environmental & Occupational Health, 61*(2), 75-86. PMID 17649959. doi:10.01111/j.1539-6924.2007.000934.x

Alliance of Nurses for Healthy Environments. (2009). *Wingspread Statement*. Retrieved April 1, 2011 from http://e-commons.org/anhe/2009/07/01/1909/

Altenburger, R. (2008, October 13). *Toxicological Interaction by Mixture Exposure.* Retrieved April 1, 2011 from http://chm.pops.int/Portals/0/vg/presentations/Monday_02_Altenburger.pdf

Altshul, L., Covaci, A., & Hauser, R. (2004). The Relationship between Levels of PCBs and Pesticides in Human Hair and Blood: Preliminary Results. *Environmental Health Perspectives, 112,* 1193-1199. PMID 15289166. doi:10.1289/ehp.69l6

Altshuller, L. F., Halak, D. B., Landing, B. H., & Kehoe, R. A. (1962, February). Deciduous teeth as an index of body burden of lead. *The Journal of Pediatrics, 60*(2), 224-229. PMID 13860784

American Cancer Society Subcommittee on Cancer in the Economically Disadvantaged. (1986). *Special Report on Cancer in the Economically Disadvantaged*. New York, NY: Author

American Industrial Hygiene Association. (2007, August 16). *What Is Industrial Hygiene?* Retrieved April 1, 2011 from http://www.aiha.org/aboutaiha/Pages/WhatIsanIH.aspx

American Nurses Association. (2003, October). *American Nurses Association Adopts Precautionary Approach.* Retrieved April 1, 2011 from http://nursingworld.org/MainMenuCategories/OccupationalandEnvironmental/PrecautionaryApproach.aspx

American Nurses Association. (2004). *American Nurses Association House of Delegates Resolution: Environmental Health Principles in Nursing Practice.* Retrieved April 1, 2011 from http://www.ana.org/2004hod/resenviron.pdf

American Public Health Association. (2005, December 14). *Environmental Health Principles for Public Health Nursing.* Retrieved April 1, 2011 from http://www.apha.org/membergroups/newsletters/sectionnewsletters/public_nur/winter06/2550.htm

Amin-Zaki, L., Elhassani, S., Majeed, M. A., Clarkson, T. W., Doherty, R. A., & Greenwood, M. (1974, November). Intra-uterine Methylmercury Poisoning in Iraq. *Pediatrics, 54*(5), 587-595. PMID 4480317

Amin-Zaki, L., Elhassani, S., Majeed, M. A., Clarkson, T. W., Doherty, R. A., Greenwood, M. R., & Giovanoli-Jakubczak, T. (1976, October). Perinatal

Methylmercury Poisoning in Iraq. *American Journal of Diseases of Children, 130,* 1070-1076. PMID 973609

Amin-Zaki, L., Majeed, M. A., Clarkson, T. W., & Greenwood, M. R. (1978, March 11). Methylmercury poisoning in Iraqi children: clinical observations over two years. *British Medical Journal, 1,* 613-616. PMID 630256

Amin-Zaki, L., Majeed, M. A., Elhassani, S. B., Clarkson, T. W., Greenwood, M. R., & Doherty, R. A. (1979, February). Prenatal Methylmercury Poisoning. *American Journal of Diseases of Children, 133,* 172-177. PMID 84530

Amin-Zaki, L., Majeed, M. A., Greenwood, M. R., Elhassani, S. B., Clarkson, T. W., & Doherty, R. A. (1981, August). Methylmercury Poisoning in the Iraqi Suckling Infant: A Longitudinal Study over Five Years. *Journal of Applied Toxicology, 1*(4), 210-214. PMID 6892222

Amler, R. W., Barone, S., Jr., Belger, A., Berlin, C. M., Jr., Cox, C., & Frank, H. (2006). Optimizing the design and interpretation of epidemiologic studies for assessing neurodevelopmental effects from *in utero* chemical exposure. *NeuroToxicology, 27,* 861-874. PMID 16889835. doi:10.1016/j.neuro.2006.07.008

Anderson, J., Moeschberger, M., Chen, M. S. Jr., Kunn, P., Wewers, M. E., & Guthrie, R. (1993, May). An acculturation scale for Southeast Asians. *Social Psychiatry and Psychiatric Epidemiology, 28*(3), 134-141. PMID 8378809

Anderson, S. (1988, October). Guidelines for use of dietary intake data. *Journal of American Dietetic Assocation, 88*(10), 1258-1260. PMID 3171018

Anderson, Y. B., Coulberson, S. L., & Phelps, J. (1993). Overview of the EPA/NIEHS/ATSDR Workshop – 'Equity in Environmental Health: Research Issues and Needs'. *Toxicology and Industrial Health, 9*(5), 679-683. PMID 8184440

Angerer, J., Ewers, U., & Wilhelm, M. (2007). Human biomonitoring: State of the art. *International Journal of Hygiene and Environmental Health, 210,* 201-228. PMID 17376741. doi:10.1016/j.ijheh.2007.01.024

Anttila, P., Heikkila, P., Makela, M., Schlunssen, V., & Priha, E. (2009, March). Retrospective Exposure Assessment for Carcinogenic Agents in Bitumen Waterproofing Industry in Finland and Denmark. *Annals of Occupational Hygiene*, 53(2), 139-151. PMID 19190074. doi:10.1093/annhyg/men082

Aprea, C., Strambi, M., Novelli, M., Lunghini, L., & Bozzi, N. (2000). Biologic Monitoring of Exposure to Organophosphorus Pesticides in 195 Italian Children. *Environmental Health Perspectives, 108,* 521-525. PMID 10856025

Araki, S., & Murata, K. (1993). Determination of Evoked Potentials in Occupational and Environmental Medicine: A Review. *Environmental Research, 63,* 133-147. PMID 8404768. doi:10.1006/enrs.1993.1135

Arble, J. (2004, June). Toxicology Primer. *AAOHN Journal, 52*(6), 254-263. PMID 15219112

Archer, T., Kostrzewa, R., Beninger, R., & Palomo, T. (2008, October). Cognitive Symptoms Facilitatory for Diagnoses in Neuropsychiatric Disorders: Executive Functions and Locus of Control. *Neurotoxicity Research, 14*(2-3), 205-225. PMID19073427

Aremu, D., Madejczyk, M., & Ballatori, N. (2008, January). N-Acetylcysteine as a Potential Antidote and Biomonitoring Agent of Methylmercury Exposure. *Environmental Health Perspectives, 116*(1), 26-31. PMID 18197295. doi:10.1289/ehp.10383

Arnold, S. F., & Price, P. S. (2007, September). Modeling mixtures resulting from concurrent exposures to multiple sources. *Toxicology and Applied Pharmacology, 223*, 121-124. PMID 17258780. doi:10.1016/j.taap.2006.11.032

Arquette, M., Cole, M., Cook, K., LaFrance, B., Peters, M., Ransom, J., … Stairs A. (2002, April). Holistic risk-based environmental decision making: a native perspective. *Environmental Health Perspectives, 110*(Supplement 2), 259-264. PMID 11929736

Ascherio, A., Chen, H., Weisskopf, M. G., O'Reilly, E., McCullough, M. L., Calle, E., … Thun, M. J. (2006, August). Pesticide Exposure and Risk for Parkinson's

Disease. *Annals of Neurology, 60*(2), 197-203. PMID 16802290. doi:10.1002/ana.20904

Aschner, M., & Walker, S. J. (2002). The neuropathogenesis of mercury toxicity (correspondence). *Molecular Psychiatry, 7*, S40-S41. PMID 12142946. doi:10.1038/sj.mp.4001176

Ashford, N. A. (1999). A Conceptual Framework for the Use of the Precautionary Principle in Law. In C. Raffensperger & J. Tickner (Eds.), *Protecting Public Health & the Environment: Implementing the Precautionary Principle* (pp. 198-206). Washington, DC: Island Press

Ashley, K., Applegate, G. T., Marcy, A. D., Drake, P. L., Pierce, P. A., Carabin, N., & Demange, M. (2009, February). Evaluation of sequential extraction procedures for soluble and insoluble hexavalent chromium compounds in workplace air samples. *Journal of Environmental Monitoring, 11*(2), 318-315. PMID 19212588. doi:10.1039/b812236a

Ashton, K., Hooper, L., Harvey, L., Hurst, R., Casgrain, A., & Fairweather-Tait, S. (2009). Methods of assessment of selenium status in humans: a systematic review. *American Journal of Clinical Nutrition, 89*(Supplement), 2025S-2039S. PMID 19420095. doi:10.3945/ajcn.2009.27230F

Askin, D. P., & Volkmann, M. (1997, October). Effect of personal hygiene on blood lead levels of workers at a lead processing facility. *American Industrial Hygiene Association Journal, 58*(10), 752-753. PMID 9342837

Assistant Secretary for Planning and Evaluation, U.S. Department of Health and Human Services. (2010, January 25). *Further Resources on Poverty Measurement, Poverty Lines and Their History*. Retrieved April 1, 2011 from http://aspe.hhs.gov/poverty

Association of American Occupational Health Nurses. (2007, November). Competencies in Occupational and Environmental Health Nursing. *AAOHN Journal, 55*(11), 442-447. PMID 18019767

Association of American Occupational Health Nurses. (2008). *The Occupational and Environmental Health Nursing Profession.* Retrieved April 1, 2011 from

https://www.aaohn.org/fact-sheets/the-occupational-and-environmental-health-nursing-profession.htm

Association of American Occupational Health Nurses. (2009, January). *Key Public Policy Issues*. Retrieved April 1, 2011 from https://www.aaohn.org/public-policy-items

Astegiano, M., Sapone, N., Demarchi, B., Rossetti, S., Bonardi, R., & Rizzetto, M. (2004). Laboratory evaluation of the patient with liver disease. *European Review for Medical and Pharmacological Sciences, 8*, 3-9. PMID 15209149

Atkinson, A. B. (1987, July). On the Measurement of Poverty. *Econometrica, 55*(4), 749-764

Audesirk, G. (1990). Effects of Heavy Metals on Neuronal Calcium Channels. In E. C. Foulkes, (Ed.), *Biological Effects of Heavy Metals, Volume 1* (pp. 1-18). Boca Raton, FL: CRC Press

Aune, H. V. (1962, March). APHA Conference Report on Population Studies: Race-Color Item Used and Abused. *Public Health Reports, 77*(3), 219-256

Automobile Workers v. Johnson Controls, Inc., 499 U.S. 187 (1991)

Axelrad, D. A., Bellinger, D. C., Ryan, L. M., & Woodruff, T. J. (2007). Dose-Response Relationship of Prenatal Mercury Exposure and IQ: An Integrative Analysis of Epidemiologic Data. *Environmental Health Perspectives, 115*, 609-615. PMID 17450232. doi:10.1289/ehp.9303

Axelrad, D. A., & Cohen, J. (2011, January). Calculating summary statistics for population chemical biomonitoring in women of childbearing age with adjustment for age-specific natality. *Environmental Research*, *111*(1), 149-155. PMID 21035114. doi:10.1016/j.envres.2010.10.002

Axelrad, D. A., Goodman, S., & Woodruff, T. (2009, May). PCB body burden in U.S. women of childbearing age 2001-2002: an evaluation of alternate summary metrics of NHANES data. *Environmental Research, 109*(4), 368-378. PMID 19251256. 10.1016/j.envres.2009.01.003

Axmon, A., Rylander, L., & Rignell-Hydbom, A. (2008, May 28). Reproductive toxicity of seafood contaminants: Prospective comparisons of Swedish east and west coast fishermen's families. *Environmental Health, 7*(20), 1-26. PMID 18507855. doi:10.1186/1476-069X-7-20

Axtell, C. D., Cox, C., Myers, G. J., Davidson, P. W., Choi, A. L., Cernichiari, E., … Clarkson, T. W. (2000). Association between Methylmercury Exposure from Fish Consumption and Child Development at Five-and-a-Half Years of Age in the Seychelles Child Development Study: An Evaluation of Nonlinear Relationships. *Environmental Research, 84,* 71-80. PMID 11068920. doi:10.1006/enrs.2000.4082

Ayotte, P., Dewailly, E., Lambert, G. H., Perkins, S. L., Poon, R., Feeley, M., … Pereq, D. (2005). Biomarker Measurements in a Coastal Fish-Eating Population Environmentally Exposed to Organochlorines. *Environmental Health Perspectives, 113,* 1318-1324. PMID 16203240

Baibergenova, A., Kudyakov, R., Zdeb, M., & Carpenter, D. O. (2003). Low Birth Weight and Residential Proximity to PCB-Contaminated Waste Sites. *Environmental Health Perspectives, 111*, 1352-1357. PMID 12896858

Baker, B. A., Herbrandson, C., Eshenaur, T., & Messing, R. B. (2005, February 18). Measuring Exposure to an Elemental Mercury Spill – Dakota County, MN, 2004. *Morbidity and Mortality Weekly Report, 54*(6), 146-149. PMID 15716806.

Baker, E. & Matte, T. (2005). Occupational Health Surveillance. In L. Rosenstock, M. R. Cullen, C. A. Brodkin & C. A. Redlich (Eds.), *Textbook of Clinical Occupational and Environmental Medicine* (2nd ed., pp. 76-82). Philadelphia, PA: Elsevier Saunders.

Baker, J. L., Olsen, L. W., & Sorensen, T. I. (2008, March). Weight at Birth and All-Cause Mortality in Adulthood. *Epidemiology, 19*(2), 197-203. PMID 18300695. doi:10.1097/EDE.0b013e31816339c6

Bakir, F., Damluji, S. F., Amin-Zaki, L., Murtadha, M., Khalidi, A., Al-Rawi, N. Y., … Doherty, R. A. (1973, July 20). Methylmercury Poisoning in Iraq. *Science, 181*(4096), 230-241. PMID 4719063

Bakir, F., Rustam, H., Tikriti, S., Al-Damluji, S. F., & Shihristani, H. (1980, January). Clinical and epidemiological aspects of methylmercury poisoning. *Postgraduate Medical Journal, 56*(1), 1-10. PMID 7383945

Ballatori, N. (2002, October). Transport of toxic metals by molecular mimicry. *Environmental Health Perspectives, 110*(Supplement 5), 689-694. PMID 12426113

Ball, E. (2008, September). *Olden Leaves NIH to Start New School of Public Health*. Environmental Factor. Retrieved April 1, 2011 from http://www.niehs.nih.gov/news/newsletter/2008/september/oldenleavesnih.cfm

Banned Hazardous Products, 16 Fed. Reg. §1303.4 (1978).

Banned Hazardous Substances, 16 Fed. Reg. §1500.17, 6(i) (1972).

Bandiera, S. (2001). Cytochrome P450 Enzymes as Biomarkers of PCB Exposure and Modulators of Toxicity. In L. W. Robertson & L. G. Hansen (Eds.), *PCBs: Recent Advances in Environmental Toxicology and Health Effects* (pp. 185-192.) Lexington, KY: The University Press of Kentucky.

Baquet, C.R., Horm, J.W., Gibbs, T., & Greenwald, P. (1991, April 17). Socioeconomic factors and cancer incidence among blacks and whites. *Journal of the National Cancer Institute, 83*(8), 551-557. PMID 2005640

Barker, D. (2004). Developmental origins of adult health and disease. *Journal of Epidemiology and Community Health*, *58*, 114-115. PMID 14729887

Barker, D., Eriksson, J., Forsen, T., & Osmond, C. (2002). Fetal origins of adult disease: strength of effects and biological basis. *International Journal of Epidemiology, 31*, 1235-1239. PMID 12540728

Barker, M., Lawrence, W., Crozier, S., Robinson, S., Baird, J., Margetts, B., … Food Choice Group of University of Southampton. (2009, June). Educational attainment, perceived control and the quality of women's diets. *Appetite, 52*(3), 631-636. PMID 19501760

Barnes, C. (2005). The Nature of Social Justice. In M. de Chesnay (Ed.) *Caring for the Vulnerable: Perspectives in Nursing Theory, Practice and Research,* (pp. 13-19). Sudbury, MA: Jones and Bartlett Publishers

Barr, D. B., & Angerer, J. (2006a). Potential Uses of Biomonitoring Data: A Case Study Using the Organophosphorus Pesticides Chlorpyrifos and Malathion. *Environmental Health Perspectives, 114,* 1763-1769. PMID 17107865

Barr, D. B., Bravo, R., Weerasekera, G., Caltabiano, L. M., Whitehead, R. D. Jr., Olsson, A.O., … Needham, L. L. (2004, February). Concentrations of dialkyl phosphate metabolites of organophosphorus pesticides in the U.S. population. *Environmental Health Perspectives, 112*(2), 186-200. PMID 14754573

Barr, D. B., Thomas, K., Curwin, B., Landsittel, D., Raymer, J., Lu, C., … Acquavella, J. (2006b, June). Biomonitoring of Exposure in Farmworker Studies. *Environmental Health Perspectives, 114*(6), 936-942. PMID 16759998

Barr, D. B., Wang, R. Y., & Needham, L. L. (2005a, August). Biologic Monitoring of Exposure to Environmental Chemicals throughout the Life Stages: Requirements and Issues for Consideration for the National Children's Study. *Environmental Health Perspectives, 113*(8), 1083-1091. PMID 16079083

Barr, D. B., Wilder, L. C., Caudill, S. P., Gonzalez, A. J., Needham, L. L., & Pirkle, J. L. (2005b, February). Urinary Creatinine Concentrations in the U.S. Population: Implications for Urinary Biologic Monitoring Measurements. *Environmental Health Perspectives, 113,* 192-200. PMID 15687057

Barr, J. R., Maggio, V. L., Barr, D. B., Turner, W. E., Sjödin, A., Sandau, C. D., … Patterson, D. G. (2003). New high-resolution mass spectrometric approach for the measurement of polychlorinated biphenyls and organochlorine pesticides in human serum. *Journal of Chromatography, B (Analytical Technologies in the Biomedical and Life Sciences), 794*, 137-148. PMID 12888206. doi:10.1016 /S1570-0232(03)00451-3

Barregard, L., Sällsten, G., Schutz, A., Attewell, R., Skerfving, S., & Jarvholm, B. (1992, May-June). Kinetics of Mercury in Blood and Urine after Brief Occupational Exposure. *Archives of Environmental Health, 47*(3), 176-184. PMID 1596100

Barrett, J. R. (2007a, April). Pesticides: toxic legacy. *Environmental Health Perspectives, 115*(4), A190. PMID 17450200

Barrett, J. R. (2007b, May). Toxic Neighbors? Fetal death risk near hazardous waste sites. *Environmental Health Perspectives, 115*(5), A263. PMID 17520049

Bartell, S. M. (2005). Risk Assessment. In H. Frumkin (Ed.), *Environmental Health from global to local* (pp. 940-960). San Francisco, CA: Jossey-Bass

Bartell, S. M., Ponce, R. A., Sanga, R. N., & Faustman, E. M. (2000). Human Variability in Mercury Toxicokinetics and Steady State Biomarker Ratios. *Environmental Research, 84*, 127-132. PMID 11068925. doi:10.1006/enrs.2000.4104

Barton, C. A. (2008). *The measurement, partitioning and near-field modeling of perfluorooctanoate (PFO) in air* (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 3329805)

Basso, Olga. (2008). Birth Weight is Forever. *Epidemiology, 19*, 204-205. PMID 18277158.doi:10.1097/EDE.0b013e31816379d9

Bates, M. N., Buckland, S. J., Garrett, N., Caudill, S. P., & Ellis, H. (2005a). Methodological aspects of a national population-based study of persistent organochlorine compounds in serum. *Chemosphere, 58,* 943-951. PMID 15639266. doi:10.1016/j.chemosphere.2004.08.095

Bates, M. N., Hamilton, J. W., LaKind, J. S., Langenberg, P., O'Malley, M., & Snodgrass, W. (2005b). Workgroup Report: Biomonitoring Study Design, Interpretation, and Communication – Lessons Learned and Path Forward. *Environmental Health Perspectives, 113*, 1615-1621. PMID 16263520. doi:10.1289/ehp.8197

Bates, N. (2003, April). Metallic and Inorganic Mercury Poisoning. *Emergency Nurse, 1*(1), 25-31. PMID 12733281

Bauman, K. J., & Graf, N. L. (2003, August). *Educational Attainment: 2000*. Retrieved April 1, 2011 from http://www.census.gov/prod/2003pubs/c2kbr-24.pdf

Baxter, D. C., Rodushkin, I., Engstrom, E., Klockare, D., & Waara, H. (2007). Methylmercury Measurement in Whole Blood by Isotope-Dilution GC-ICPMS with Two Sample Preparation Methods. *Clinical Chemistry, 53*(1), 111-116. PMID 17095539. doi:10.1373/clinchem.2007.072520

Beattie, A. D., Moore, M. R., Goldberg, A., Finlayson, M. J., Graham, J. F., Mackie, E. M., … Steward, G. T. (1975, March 15). Role of chronic low level lead exposure in the etiology of mental retardation. *Lancet (Br.), 1*(7907), 589-592. PMID 47943

Beauchamp, T. L., & Childress, J. F. (2001). *Principles of Biomedical Ethics* (5th ed.) New York, NY: Oxford University Press

Bellés, M., Albina, M., Sánchez, D., Corbella, J., & Domingo, J. (2002, January). Interactions in developmental toxicology: effects of concurrent exposure to lead, organic mercury, and arsenic in pregnant mice. *Archives of Environmental Contamination and Toxicology, 42*(1), 93-98. PMID 11706373. doi:10.1007/s002440010296

Bellés-Isles, M., Ayotte, P., Dewailly, E., Weber, J.P., & Roy, R. (2002, January 25). Cord blood lymphocyte functions in newborns from a remote maritime population exposed to organochlorines and methylmercury. *Journal of Toxicology and Environmental Health Part A, 65*(2), 165-182. PMID 11820504

Bellinger, D. C. (2007, October). Lead Neurotoxicity in Children: Decomposing the Variability in Dose-Effect Relationships. *American Journal of Industrial Medicine, 50*(10), 720-728. PMID 17290364. doi:10.1002/ajim.20438

Bellinger, D. C. (2008, May). Neurological and Behavioral Consequences of Childhood Lead Exposure. *PLoS Medicine, 5*(5), 691-692. PMID 18507501. doi:10.1371/journal.pmed.0050115

Bellinger, D. C., & Bellinger, A. M. (2006, April). Childhood lead poisoning: the torturous path from science to policy. *Journal of Clinical Investigation, 116*(4), 853-857. PMID 16585952. doi:10.1172/JCI28232

Bellinger, D. C., Leviton, A., Needleman, H. L., Waternaux, C., & Rabinowitz, M. (1986a). Low-Level Lead Exposure and Infant Development in the First Year. *Neurobehavioral Toxicology and Teratology, 8,* 151-161. PMID 2423895.

Bellinger, D. C., Leviton, A., Rabinowitz, M., Allred, E., Needleman, H., & Schoenbaum, S. (1991). Weight Gain and Maturity in Fetuses Exposed to Low Levels of Lead. *Environmental Research, 54,* 151-158. PMID 2029876

Bellinger, D. C., Leviton, A., Rabinowitz, M., Needleman, H., & Waternaux, C. (1986b, June 6). Correlates of Low-Level Lead Exposure in Urban Children at 2 Years of Age. *Pediatrics, 77*(6)*,* 826-833. PMID 3714374

Bellinger, D. C., Leviton, A., & Sloman, J. (1990). Antecedents and Correlates of Improved Cognitive Performance in Children Exposed *in utero* to Low Levels of Lead. *Environmental Health Perspectives, 89,* 5-11. PMID 2088755

Bellinger, D. C., Leviton, A., Waternaux, C., & Allred, E. (1985). Methodological Issues in Modeling the Relationship between Low-Level Lead Exposure and Infant Development: Examples from the Boston Lead Study. *Environmental Research, 38*, 119-129. PMID 3841053

Bellinger, D. C., Leviton, A., Waternaux, C., Needleman, H., & Rabinowitz, M. (1987, April 23). Longitudinal Analyses of Prenatal and Postnatal Lead Exposure and Early Cognitive Development. *The New England Journal of Medicine, 316*(17), 1037-1042. PMID 3561456

Bellinger, D. C., & Needleman, H. L. (1994). The Neurotoxicity of Prenatal Exposure to Lead: Kinetics, Mechanisms, and Expressions. In H. L. Needleman & D. C. Bellinger (Eds.), *Prenatal Exposure to Toxicants* (pp. 89-111). Baltimore, MD: The Johns Hopkins University Press

Bellinger, D. C., Needleman, H. L., Leviton, A., Waternaux, C., Rabinowitz, M. B., & Nichols, M. L. (1984). Early Sensory-Motor Development and Prenatal Exposure to Lead. *Neurobehavioral Toxicology and Teratology, 6,* 387-402. PMID 6514103

Bellinger, D. C., Sloman, J., Leviton, A., Rabinowitz, M., Needleman, H. L., & Waternaux, C. (1991, February). Low-Level Lead Exposure and Children's Cognitive Function in the Preschool Years. *Pediatrics, 87*(2), 219-227. PMID 1987535

Bellinger, D. C., Trachtenberg, F., Barregard, L., Tavares, M., Cernichiari, E., Daniel, D., & McKinlay, S. (2006, April 19). Neuropsychological and Renal Effects of Dental Amalgam in Children: A Randomized Clinical Trial. *Journal of the American Medical Association, 295*(15), 1775-1783. PMID 16622139. doi:10.1001/jama.295.15.1775

Belson, M. G., Schier, J. G., & Patel, M. M. (2005, January 14). Case Definitions for Chemical Poisoning: Case Definitions for Potential Terrorism Agents: Toxins and Toxicants. *Morbidity and Mortality Weekly Report, 54*(RR-1), 1-5, 11-13. PMID 15660014

Bemis, J. C., & Seegal, R. F. (1999, November). Polychlorinated Biphenyls and Methylmercury Act Synergistically to Reduce Rat Brain Dopamine Content in Vitro. *Environmental Health Perspectives, 107*(11), 879-885. PMID 10544155

Bemis, J., & Seegal, R. (2000, December). Polychlorinated biphenyls and methylmercury alter intracellular calcium concentrations in rat cerebellar granule cells. *Neurotoxicology, 21*(6), 1123-1134. PMID 11233759

Bengiamin, M., Capitman, J., & Ruwe, M. (2010, July). Disparities in Initiation and Adherence to Prenatal Care: Impact of Insurance, Race-Ethnicity and Nativity. *Maternal and Child Health Journal, 14*(4), 618-624. PMID 19557508. doi:10.1007/s10995-009-0485-y

Bennett, D. H., McKone, T. E., Evans, J. S., Nazaroff, W. W., Margni, M. D., Jolliet, O., & Smith, K. R. (2002, May 1). Defining Intake Fraction. *Environmental Science & Technology, 36*(9), 206A-211A. PMID 12026996

Benowitz, N. (1999, May). Biomarkers of Environmental Tobacco Smoke Exposure. *Environmental Health Perspectives, 107*(Supplement 2), 349-355. PMID 10350520

Benowitz, N., Bernert, J., Caraballo, R., Holiday, D., & Wang, J. (2009). Optimal Serum Cotinine Levels for Distinguishing Cigarette Smokers and Nonsmokers within Different Racial Ethnic Groups in the United States Between 1999 and 2004. *American Journal of Epidemiology, 169*(2), 236-248. PMID 19019851. doi:10.1093/aje/kwn301

Berglund, M., Lind, B., Bjornberg, K. A., Palm, B., Einarsson, O., & Vahter, M. (2005, October 3). Inter-individual variations of human mercury exposure biomarkers: a cross-sectional assessment. *Environmental Health, 4*(20), 1-11. PMID 16202128. doi:10.1186/1476-069X-4-20

Bergonzi, R., Specchia, C., Dinolfo, M., Tomasi, C., De Palma, G., Frusca, T., & Apostoli, P. (2009, August). Distribution of persistent organochlorine pollutants in maternal and foetal tissues: data from an Italian polluted urban area. *Chemosphere, 76*(6), 747-754. PMID 19539348. doi: 10.1016/j.chemosphere.2009.05.026

Berkman, L., & Epstein, A. M. (2008, June 5). Beyond Health Care – Socioeconomic Status and Health. *The New England Journal of Medicine, 358*(23), 2509-2510. PMID 18525049. doi:10.1056/NEJMe0802773

Berkowitz, G. S., Wetmur, J. G., Birman-Deych, E., Obel, J., Lapinski, R. H., Godbold, J. H., … Wolff, M. S. (2004). *In Utero* Pesticide Exposure, Maternal Paraoxonase Activity, and Head Circumference. *Environmental Health Perspectives, 112,* 388-391. PMID 14998758.

Berman, E. (1966, May). The Biochemistry of Lead: Review of the Body Distribution and Methods of Lead Determination. *Clinical Pediatrics, 5*(5), 287-291

Berman, L., Ostchega, Y., Reed-Gillette, D., & Porter, K. (2003). Public Health Informatics in the National Health and Nutrition Examination Survey. In P. O'Carroll, W. Yasnoff, M. E. Ward, L. Ripp, & E. Martin, (Eds.), *Public*

Health Informatics and Information Systems (pp. 712-742). New York, NY: Springer Publishing Company

Bernert, J. T., Turner, W. E., Patterson, D. G. Jr., & Needham, L. L. (2007, June). Calculation of serum "total lipid" concentrations for the adjustment of persistent organohalogen toxicant measurements in human samples. *Chemosphere, 68*(5), 824-831. PMID 17408721. doi:10.1016/j.chemosphere.2007.02.043

Bernert, J. T., Turner, W. E., Pirkle, J. L., Sosnoff, C., Akins, J., Waldrep, M., … Sampson, E. J. (1997, December). Development and validation of sensitive method for determination of serum cotinine in smokers and nonsmokers by liquid chromatography/atmospheric pressure ionization tandem mass spectrometry. *Clinical Chemistry, 43*(12), 2281-2291. PMID 9439445

Bernstein, J. A., Alexis, N., Bacchus, H., Bernstein, I. L., Fritz, P., Horner, E., ... Tarlo, S. M. (2008, March). The health effects of nonindustrial indoor air pollution. *Journal of Allergy and Clinical Immunology, 121*(3), 585-591. PMID 18155285. doi:10.1016/j.jaci.2007.10.045

Besharov, D. J., & Couch, K. (2009, Fall). European Measures of Income, Poverty and Social Exclusion: Recent Developments and Lessons for U.S. Poverty Measurement. *Journal of Policy Analysis and Management, 28*(4), 713-715

Bickel, G., Nord, M., Price, C., Hamilton, W. & Cook, J. (2000). *Guide to Measuring Household Food Security*. Retrieved April 1, 2011 from http://www.fns.usda.gov/fsec/files/fsguide.pdf

Birkner, R. (1965, July). Plan and Initial Program of the Health Examination Survey. *Vital and Health Statistics, 1*(4), 1-48. PMID 14339866

Björnberg, K. A., Vahter, M., Petersson-Grawé, K., Glynn, A., Cnattingius, S., Darnerud, P. O., … Berglund, M. (2003, April). Methyl mercury and inorganic mercury in Swedish pregnant women and in cord blood: influence of fish consumption. *Environmental Health Perspectives, 111*(4), 637-641. PMID 12676628. doi:10.1289/ehp.5618

Björkman, L., Lundekvam, B. F., Laegreid, T., Bertelsen, B. I., Morild, I., Lilleng, P., … Vahter, M. (2007, October 11). Mercury in human brain, blood, muscle and toenails in relation to exposure: an autopsy study. *Environmental Health, 6*(30), 1-30. PMID 17931423. doi:10.1186/1476-069X-6-30

Björkman, L., Sandborgh-Englund, G., & Ekstrand, J. (1997). Mercury in Saliva and Feces after Removal of Amalgam Fillings. *Toxicology and Applied Pharmacology, 144*, 156-162. PMID 9169079. doi:10.1006/taap.1997.8128

Bjorling-Poulsen, M., Andersen, H. R., & Grandjean, P. (2008, October 22). Potential developmental neurotoxicity of pesticides used in Europe. *Environmental Health, 7,* 1-50. PMID 18945337. doi:10.1186/1476-069X-7-50

Bjornberg, K. A., Vahter, M., Berglund, B., Niklasson, B., Blennow, M., & Sandborgh-Englund, G. (2005, October). Transport of Methylmercury and Inorganic Mercury to the Fetus and Breast-Fed Infant. *Environmental Health Perspectives, 113*(10), 1381-1385. PMID 16203251

Black, H. (2006, November). Setting a Baseline for Biomonitoring. *Environmental Health Perspectives, 114*(11), A652-A654. PMID 17107843

Blackburn, S. T. (2007). *Maternal, Fetal & Neonatal Physiology: A Clinical Perspective*, 3rd edition. St. Louis, MO: Saunders, Inc.

Bleecker, M. L., Ford, D. P., Lidgren, K. N., Scheetz, K., & Tiburzi, M. J. (2003). Association of Chronic and Current Measures of Lead Exposure with Different Components of Brainstem Auditory Evoked Potentials. *NeuroToxicology, 24*, 625-631. PMID 12900075

Bloom, M. S., Vena, J. E., Olson, J. R., & Kostyniak, P. J. (2009, November). Assessment of polychlorinated biphenyl congeners, thyroid stimulating hormone, and free thyroxine among New York State anglers. *International Journal of Hygiene and Environmental Health*, *212*(6), 599-601. PMID 19493696. doi:10.1016/j.ijheh.2009.04.005

Bloom, M. S., Vena, J. E., Swanson, M. K., Moysich, K. B., & Olson, J. R. (2005, February). Profiles of ortho-polychlorinated biphenyl congeners, dichlorodiphenyldichloroethylene, hexachlorobenzene, and Mirex among male

Lake Ontario sportfish consumers: the New York State Angler cohort study. *Environmental Research, 97*(2), 178-194. PMID 15533334. doi:10.1016/j.envres.2004.06.001

Bloom, M. S., Weiner, J. M., Vena, J. E., & Beehler, G. P. (2003, September). Exploring associations between serum levels of select organochlorines and thyroxine in a sample of New York state sportsmen: the New York State Angler Cohort Study. *Environmental Research, 93*(1), 52-66. PMID 12865048

Blumenthal, M. N. (2009, April 23). Review on Biomedical Ambiguity: Race, Asthma and the Contested Meaning of Genetic Research in the Caribbean (Whitmarsh, 2008). *The New England Journal of Medicine, 360*(17), 1796-1797

Blumer, M. (1975, August). Organic Compounds in Nature: Limits of Our Knowledge. *Angewandte Chemie (International Edition in English), 14*(8), 507-514.

Boersma, E. R., & Lanting, C. I. (2000). Environmental exposure to polychlorinated biphenyls (PCBs) and dioxins: consequences for long-term neurological and cognitive development of child lactation. *Advances in experimental medicine and biology, 478,* 271-287. PMID 11065080

Bohle, H.-G. (2001, February). Vulnerability and Criticality. *Newsletter of the International Human Dimensions Programme on Global Environmental Change*. Bonn, Germany: International Human Dimensions Programme on Global Environmental Change

Bohle, H.-G., Etzold, B., & Keck, M. (2009, February). Resilience as Agency. *Newsletter of the International Human Dimensions Programme on Global Environmental Change*. (pp. 8-13). Bonn, Germany: International Human Dimensions Programme on Global Environmental Change

Boischio, A. P., & Henshel, D. (2000). Fish Consumption, Fish Lore, and Mercury Pollution – Risk Communication for the Madeira River People. *Environmental Research, 84*(Section A), 108-126. PMID 11068924. doi:10.1006/enrs.2000.4035

Bolte, G., Fromme, H., & GME Study Group. (2009, January). Socioeconomic determinants of children's environmental tobacco smoke exposure and family's home smoking policy. *European Journal of Public Health, 19*(1), 52-58. PMID 19033356. doi:10.1093/eurpub/ckn114

Bonacci, S., Browne, M. A., Dissanayake, A., Hagger, J. A., Corsi, I., Focardi, S., & Galloway, T. S. (2004, September). Esterase activities in the bivalve mollusc Adamussium colbecki as a biomarker for pollution monitoring in the Antarctic marine environment. *Marine Pollution Bulletin, 49*(5-6), 445-455. PMID 15325212. doi:10.1016/j.marpolbul.2004.02.033

Bondy, S., Victor, J., & Diemert, L. (2009, June 1). Origin and use of the 100 cigarette criterion in tobacco surveys. *Tobacco Control, 18*, 317-323. PMID 19491091. doi:10.1136/tc.2008.027276

Bornschein, R., & Kuang, S.-R. (1990). Behavioral Effects of Heavy Metal Exposure. In E. C. Foulkes, (Ed.), *Biological Effects of Heavy Metals, Volume 1* (pp. 201-220). Boca Raton, FL: CRC Press

Borum, D., Manibusan, M. K., Schoeny, R., & Winchester, E. L. (2001, January 3). *Water Quality Criterion for the Protection of Human Health: Methylmercury* (EPA-823-R-01-001 ed.) Washington, DC: Environmental Protection Agency

Bouchard, M., Carrier, G., Brunet, R., Bonvalot, Y., & Gosselin, N. (2005, March). Determination of Biological Reference Values for Chlorpyrifos Metabolites in Human Urine Using a Toxicokinetic Approach. *Journal of Occupational and Environmental Hygiene, 2*, 155-168. PMID 15764539. doi:10.1080/15459620590922407

Boucher, O., Bastien, C., Saint-Amour, D., Dewailly, E., Ayotte, P., Jacobson, J., …, Muckle, G. (2010, August). Prenatal exposure to methylmercury and PCBs affects distinct stages of information processing: an event-related potential study with Inuit children. *NeuroToxicology, 31*(4), 373-384. PMID 20403381. doi:10.1016/j.neuro.2010.04.005

Boucher, O., Muckle, G., & Bastien, C. (2009, January). Prenatal Exposure to Polychlorinated Biphenyls: A Neuropsychologic Analysis. *Environmental Health Perspectives, 117*(1), 7-16. PMID 19165381

Boutain, D. M. (2005). Social Justice in Nursing: A Review of the Literature. In M. de Chesnay (Ed.) *Caring for the Vulnerable: Perspectives in Nursing Theory, Practice and Research,* (pp. 21-29). Sudbury, MA: Jones and Bartlett Publishers

Bowers, T. S., & Beck, B. D. (2006). What is the meaning of non-linear dose-response relationships between blood lead concentrations and IQ? *NeuroToxicology, 27,* 520-524. PMID 16551479. doi:10.1016/j.neuro.2006.02.001

Boyce, N. (2004, October 10). Is there a tonic in the toxin? *U.S. News and World Report*. Retrieved April 1, 2011 from http://www.usnews.com/usnews/culture/articles/041018/18calabrese.htm

Boyle, C. A., Decoufle, P., & Yeargin-Allsopp, M. (1994, March). Prevalence and Health Impact of Developmental Disabilities in U.S. Children. *Pediatrics, 93*(3), 399-403. PMID 7509480

Bramer, S., & Kallungal, B. (2003, August). Commentary: Clinical considerations in study designs that use cotinine as a biomarker. *Biomarkers, 8*(3-4), 187-203. PMID 12944172. doi:10.1080/13547500310012545

Braun, J. M., Daniels, J. L., Poole, C., Olshan, A., Hornung, R., Bernert, J., … & Lanphear, B. (2010, August 27). A prospective cohort study of biomarkers of prenatal tobacco smoke exposure: the correlation between serum and meconium and their association with infant birth weight. *Environmental Health, 9*, 53. PMID 20799929. doi:10.1186/1476-069X-9-53

Braun, J. M., Kahn, R. S., Froelich, T., Auinger, P., & Lanphear, B. P. (2006). Exposures to Environmental Toxicants and Attention Deficit Hyperactivity Disorder in U.S. Children. *Environmental Health Perspectives, 114*, 1904-1909. PMID 17185283

Braun, J. M., & Lanphear, B. (2007, March). Comments (Peer Commentary on the article "Lead neurotoxicity in children: is prenatal exposure more important than postnatal exposure?" by Ronchetti, Van Den Hazel, Schoeters, Hanke, Rennezova, Barreto & Villa, 2006, October). *Acta Paediatrica (Oslo), 96*(3), 474-475. PMID 17407486. doi:10.1111/j.1651-2227.2007.00131.x

Braveman, P. (2005, December). The question is not, 'Is race or class more important?' *Journal of Epidemiology and Community Health, 59*(12), 1029. PMID 16286488

Braveman, P. (2006). Health Disparities and Health Equity: Concepts and Measurement. *Annual Review of Public Health, 27*, 167-194. PMID 16533114. doi:10.1146/annurev.publhealth.27.021405.102103

Briggs, D. J. (2008, November 27). A framework for integrated environmental health impact assessment of systemic risks. *Environmental Health, 7*(61), 1-35. PMID 19038020. doi:10.1186/1476-069X-7-61

Briones, T. L. (2007). Psychoneuroimmunology and Related Mechanisms in Understanding Health Disparities in Vulnerable Populations. *Annual Review of Nursing Research, 25*, 219-256. PMID 17958294

Brock, J. W., Burse, V. W., Ashley, D. L., Najam, A. R., Green, V. E., Korver, M. P., … & Needham, L. L. (1996, November-December). An improved analysis for chlorinated pesticides and polychlorinated biphenyls (PCBs) in human and bovine sera using solid-phase extraction. *Journal of Analytical Toxicology, 20*(7), 528-536. PMID 8932301

Brodaty, H., Pond, D., Kemp, N. M., Luscombe, G., Harding, L., Berman, K., & Huppert, F. A. (2002, March 4). The GPCOG: a new screening test for dementia designed for general practice. *Journal of American Geriatric Society, 50*(3), 530-534. PMID 11943052. doi:10.1046/j.1532-5415.2002.50122.x

Brody, C., & Melamed, A. (2004, April). The Precautionary Approach. *American Journal of Nursing, 104*(4), 104. PMID 15171122

Bronfenbrenner, U. (1979). *The Ecology of Human Development: experiments by nature and design*. Cambridge, MA: Harvard University Press

Bronfenbrenner, U., & Ceci, S. J. (1994, October). Nature-Nurture Reconceptualized in Developmental Perspective: A Bioecological Model. *Psychological Review, 101*(4), 568-586. PMID 7984707

Brown, V. (2004, November). *Kenneth Olden, Master Fencer*. *American Journal of Public Health, 94*(11), 1905-1907. PMID 1448557

Browne, A. J. (1997). A Concept Analysis of Respect Applying the Hybrid Model in Cross-Cultural Settings. *Western Journal of Nursing Research, 19*(6), 762-780

Browne, G. (2005, April). Housing, Social Support and People with Schizophrenia: A Grounded Theory Study. *Issues in Mental Health Nursing, 26*(3), 311-326. PMID 16020049. doi:10.1080/01612840590915694

Bruckner, J. V. (2000). Differences in Sensitivity of Children and Adults to Chemical Toxicity: The NAS Panel Report. *Regulatory Toxicology and Pharmacology, 31*, 280-285. PMID 10915586. doi:10.1006/rtph.2000.1393

Brulle, R., & Pellow, D. (2006). Environmental Justice: Human Health and Environmental Inequalities. *Annual Review of Public Health, 27*, 103-124. PMID 16533111. doi:10.1146/annurev.publhealth.27.021405.102124

Bryant, B., Hopkins, J., Arceo, S., & Leitman, S. (2009, September). Evaluation of low red blood cell mean corpuscular volume in an apheresis donor population. *Transfusion, 49*(9), 1971-1976. PMID 19453988. doi:10.1111/j.1537-2995.2009.02207.x

Bryant, B., & Mohai, P. (1992). Introduction. In B. Bryant & P. Mohai (Eds.), *Race and the Incidence of Environmental Hazards* (pp. 1-9). Boulder, CO: Westview Press.

Bryant, B., & Mohai, P. (1992, March-April). The Michigan conference: A turning point. *EPA Journal, 18*(1), 9-10

Bucher, J. R., & Lucier, G. (1998, December). Current Approaches Toward Chemical Mixture Studies at the National Institutes of Environmental Health Sciences and the U.S. National Toxicology Program. *Environmental Health Perspectives, 106*(Supplement 6), 1295-1298. PMID 9860884

Buchet, J. P., Roels, H., Hubermont, G., & Lauwerys, R. (1978). Placental Transfer of Lead, Mercury, Cadmium, and Carbon Monoxide in Women. II. Influence of

some epidemiological factors on the frequency distributions of the biological indices in maternal and umbilical cord blood. *Environmental Research, 15,* 494-503. PMID 679913

Buck, A., Vena, J., McGuinness, B., Cooney, M., & Louis, G. (2010, May). Communicating serum chemical concentrations to study participants: follow up survey. *Environmental Health, 9*(20). doi:10.1186/1476-069X-9-20

Buck, G. M., Lynch, C. D., Stanford, J. B., Sweeney, A. M., Schieve, L. A., Rockett, J. C., … Schrader, S. M. (2004, January). Prospective Pregnancy Study Designs for Assessing Reproductive and Developmental Toxicants. *Environmental Health Perspectives, 112*(1), 79-86. PMID 14698935

Buck, G. M., Mendola, P., Vena, J. E., Sever, L. E., Kostyniak, P., Greizerstein, H., … Stephen, F. D. (1999 February). Paternal Lake Ontario fish consumption and risk of conception delay, New York State Angler Cohort. *Environmental Research, 80*(2 Part 2), S13-S18. PMID 10092415. doi:10.1006/enrs.1998.3926

Buck, G. M., Sever, L. E., Mendola, P., Zielezny, M., & Vena, J. E. (1997, December 1). Consumption of contaminated sport fish from Lake Ontario and time-to-pregnancy, New York State Angler Cohort. *American Journal of Epidemiology, 146*(11), 949-954. PMID 9400336

Buck, G. M., Tee, G. P., Fitzgerald, E. F., Vena, J. E., Weiner, J. M., Swanson, M., & Msall, M. E. (2003, June 2). Maternal fish consumption and infant birth size and gestation, New York State Angler Cohort Study. *Environmental Health, 2*(1):7. PMID 12826023. doi:10.1186/1476-069X-2-7

Buck, G. M., Vena, J. E., Schisterman, E. F., Dmochowski, J., Mendola, P., Sever, L., … Olson, J. (2000, July). Parental consumption of contaminated sport fish from Lake Ontario and predicted fecundability. *Epidemiology, 11*(4), 388-393. PMID 10874544

Budtz-Jørgensen, E., Grandjean, P., & Weihe, P. (2007a). Separation of Risks and Benefits of Seafood Intake. *Environmental Health Perspectives, 115,* 323-327. PMID 17431478. doi:10.1289/ehp.9738

Budtz-Jørgensen, E., Kieding, N., & Grandjean, P. (2001, September). Benchmark Dose Calculation from Epidemiological Data. *Biometrics, 57,* 698-706. PMID 11550917

Budtz-Jørgensen, E., Keiding, N., Grandjean, P., & Weihe, P. (2002, October). Estimation of health effects of prenatal methylmercury exposure using structural equation models. *Environmental Health, 1*(2), 1-22. PMID 12513702

Budtz-Jørgensen, E., Keiding, N., Grandjean, P., & Weihe, P. (2003). Consequences of exposure measurement error for confounder identification in environmental epidemiology. *Statistics in Medicine, 22*, 3089-3100. PMID 12973789. doi:10.1002/sim.1541

Budtz-Jørgensen, E., Keiding, N., Grandjean, P., & Weihe, P. (2007b). Confounder Selection in Environmental Epidemiology: Assessment of Health Effects of Prenatal Mercury Exposure. *Annals of Epidemiology, 17,* 27-35. PMID 17027287. doi:10.1016/j.annepidem.2006.05.007

Buelke-Sam, J., & Mactutus, C. F. (1990, May-June). Workshop on the Qualitative and Quantitative Comparability of Human and Animal Developmental Neurotoxicology, Work Group II Report: Testing Methods in Developmental Neurotoxicity for Use in Human Risk Assessment. *NeuroToxicology and Teratology, 12*(3), 269-274. PMID 2196424

Buescher, P., Gizlice, Z., & Jones-Vessey, K. (2005, July-August). Discrepancies Between Published Data on Racial Classification and Self-Reported Race: Evidence from the 2002 North Carolina Live Birth Records. *Public Health Reports, 120*(4), 393-398. PMID 16025719

Bullard, R. D. (1990). *Dumping in Dixie: Race, Class and Environmental Quality*. Boulder, CO: Westview Press

Bullard, R. D. (1992). Environmental Blackmail in Minority Communities. In B. Bryant & P. Mohai (Eds.), *Race and the Incidence of Environmental Hazards* (pp. 82-95). Boulder, CO: Westview Press

Bullard, R. D., Mohai, P., Saha, R., & Wright, B. (2007, March). *Toxic Wastes and Race at Twenty (1987-2007*). Cleveland, OH: United Church of Christ.

Retrieved April 1, 2011 from http://www.ucc.org/environmental-ministries/environment/toxic-waste-20.htm

Burbacher, T. M., Rodier, P. M. & Weiss, B. (1990, May-June). Methylmercury Developmental Neurotoxicity: A Comparison of Effects in Humans and Animals. *NeuroToxicology and Teratology, 12*(3), 191-202. PMID 2196419

Burbank, P. M. (2006). *Vulnerable Older Adults: Health Care Needs and Interventions*. New York, NY: Springer Publishing Company

Burgard, S. A., Brand, J. E., & House, J. S. (2009, September). Perceived job insecurity and worker health in the United States. *Social Science & Medicine, 69*(5), 777-785. PMID 19596166. doi:10.1016/j.socscimed.2009.06.029

Burger, J. (2002). Consumption Patterns and Why People Fish. *Environmental Research Section A, 90*, 125-135. PMID 12483803

Burns, C. J., Garabrant, D., Albers, J. W., Berent, S., Giordani, B., Haidar, S., … Richardson, R. J. (2006). Chlorpyrifos exposure and biological monitoring among manufacturing workers. *Occupational and Environmental Medicine, 63,* 218-220. PMID 16497866. doi:10.1136/oem.2005.021139

Burst, H. V. (2003). *Varney's Midwifery*, 4th edition. Sudbury, MA: Jones & Bartlett Publishers, Inc.

Burstyn, I. (2009, April). Measurement Error and Model Specification in Determining How Duration of Tasks Affects Level of Occupational Exposure. *Annals of Occupational Hygiene, 53*(3), 265-270. PMID 19188265. doi:10.1093/annhyg/mep003

Butler, J., Cohen, M., Friedman, C., Scripp, R., & Watz, C. (2002, October). Collaboration between public health and law enforcement: new paradigms and partnerships for bioterrorism planning and response. *Emerging Infectious Diseases, 8*(10), 1152-1156. PMID 12396931

Butler Walker, J., Houseman, J., Seddon, L., McMullen, E., Tofflemire, K., Mills, C., … Van Oostdam, J. (2006, March). Maternal and umbilical cord blood levels

of mercury, lead, cadmium, and essential trace elements in Arctic Canada. *Environmental Research, 100*(3), 295-318. PMID 16081062. doi:10.1016/j.envres.2005.05.006

Butler Walker, J., Seddon, L., McMullen, E., Houseman, J., Tofflemire, K., Corriveau, A., … Van Oostdam, J. (2003, January 20). Organochlorine levels in maternal and umbilical cord blood plasma in Arctic Canada. *The Science of the Total Environment, 302*(1-3), 27-52. PMID 12526896. doi:10.1016/S0048-9697(02)00319-4

Butte, W., & Heinzow, B. (2002). Pollutants in house dust as indicators of indoor contamination. *Reviews of Environmental Contamination and Toxicology, 175*, 1-46. PMID 12206053.

Butterfield, P. (2002, September). Upstream Reflections on Environmental Health: An Abbreviated History and Framework for Action. *Advances In Nursing Science, 25*(1), 32-49. PMID 15920361

Butterfield, P., Hill, W., & Nelson, S. (2004, Spring). An environmental risk reduction study with rural families: linking conceptual and measurement phases of environmental health research. *Communicating Nursing Research, 37,* 131. PMID 15382321

Cade, J., Thompson, R., Burley, V., & Warm, D. (2002, August). Development, validation and utilisation of food-frequency questionnaires - a review. *Public Health Nutrition, 5*(4), 567-587. PMID 12186666. doi:10.1079/PHN2001318

Calabrese, E. J. (2005, September). Biomedical Implications of Hormesis. *Newsletter of Biological Effects of Low Level Exposures (BELLE)*, 13(2), 1

Calabrese, E. J. (2007). Threshold Dose - Response Model – RIP: 1991 - 2006. *BioEssays, 29*(7), 686-688. PMID 17563088. doi:10.1002/bies.20590

Calabrese, E. J. (2008). Hormesis: Why It Is Important to Toxicology and Toxicologists. *Environmental Toxicology and Chemistry, 27*(7), 1451-1474. PMID 18275256. doi:10.1897/07-541

Calabrese, E. J., & Baldwin, L. A. (2003, February 13). Toxicology rethinks its central belief. *Nature, 421*, 691-692. PMID 12610596. doi:10.1038/421691a

Calafat, A. M., Ye, X., Silva, M. J., Kuklenyik, Z., & Needham, L. L. (2006). Human exposure assessment to environmental chemicals using biomonitoring. *International Journal of Andrology, 29*, 166-171. PMID 16466536. doi:10.1111/j.1365-2605.2005.00570.x

Caldwell, K. L., Mortensen, M. E., Jones, R. L., Caudill, S. P., & Osterloh, J. D. (2009, May 29). Total blood mercury concentrations in the U.S. population: 1999-2006. *International Journal of Hygiene and Environmental Health, 212*(6), 588-598. PMID 19481974. doi:10.1016/j.ijheh.2009.04.004

California Office of Environmental Health Hazard Assessment. (2008, December 18). *Air Toxicology and Epidemiology.* Retrieved April 1, 2011 from http://www.oehha.ca.gov/air

Callahan, M. A., & Sexton, K. (2007, May). If Cumulative Risk Assessment Is the Answer, What is the Question? *Environmental Health Perspectives, 115*(5), 799-806. PMID 17520071. doi:10.1289/ehp.9330

Calkins, M., Szmerekovsky, J., & Biddle, S. (2007). Effect of Increased Time Spent Outdoors on Individuals with Dementia Residing in Nursing Homes. *Journal of Housing for the Elderly, 21*(3), 211-228

Calvert, G. (2007, April 27). Lead Exposure Among Females of Childbearing Age – United States, 2004. *Mortality and Morbidity Weekly Report, 56*(16), 397-400

Calvert, G., Sanderson, W., Barnett, M., Blondell, J., & Mehler, L. (2001). Surveillance of Pesticide-Related Illnesses and Injuries in Humans. In R. I. Kreiger (Ed.), *Handbook of Pesticide Toxicology* (2nd ed., Vol. I, pp. 603-641). San Diego, CA.: Academic Press

Campbell-Lendrum, D., Wilkinson, P., Kuhn, K., Kovats, R., Haines, A., & Menne, B. (2002). Monitoring the health impacts of global climate change. In P. Martens & A. J. McMichael (Eds.), *Environmental Change, Climate and Health* (pp. 253-289). Cambridge, U.K.: Cambridge University Press

Canfield, R. L., Henderson, C. R., Cory-Slechta, D. A., Cox, C., Jusko, T. A., & Lanphear, B. P. (2003, April 17). Intellectual Impairment in Children with Blood Lead Concentrations below 10 μg/dl. *The New England Journal of Medicine, 348*(16), 1517-1526. PMID 12700371. doi:10.1056/NEJMoa022848

Cantonwine, D., Hu, H., Tellez-Rojo, M., Sanchez, B. Lamadrid-Figueroa, H., Ettinger, A., … Wright, R. (2010, July). HFE gene variants modify the association between maternal lead burden and infant birthweight: a prospective birth cohort study in Mexico City, Mexico. *Environmental Health, 9*(43). doi:10.1186/1476=069X-9-43

Cao, Y., Calafat, A. M., Doerge, D. R., Umbach, D. M., Bernbaum, J. C., Twaddle, N. C., … Rogan, W. J. (2009, February). Isoflavones in urine, saliva and blood of infants: data from a pilot study on the estrogenic activity of soy formula. *Journal of Exposure Science & Environmental Epidemiology, 19*(2), 223-234. PMID 18665197. doi:10.1038/jes.2008.44

Cao, Y., Chen, A., Jones, R., Radcliffe, J., Caldwell, K., … Rogan, W. (2010, January). Does background postnatal methyl mercury exposure in toddlers affect cognition and behavior? *NeuroToxicology, 31*(1), 1-9. PMID: 19969021. doi:10.1016/j.neuro.2009.10.017

Caraccio, T., & Mofenson, H. (1993). Pharmacokinetics. In P. Viccellio (Ed.), *Handbook of Medical Toxicology* (pp. 12-27). Boston, MA: Little, Brown and Company

Carpenter, D. O. (2006). Polychlorinated Biphenyls (PCBs): Routes of Exposure and Effects on Human Health. *Reviews on Environmental Health, 21*(1), 1-23. PMID 16700427

Carpenter, D. O., Arcaro, K. F., Bush, B., Niemi, W. D., Pang, S., & Vakharia, D. D. (1998, December). Human Health and Chemical Mixtures: An Overview. *Environmental Health Perspectives Supplements, 106*(Supplement 6), S1263-S1270. PMID 9860880

Carpenter, D. O., Arcaro, K., & Spink, D. C. (2002, February). Understanding the Human Health Effects of Chemical Mixtures. *Environmental Health Perspectives, 110*(Supplement 1), S25-S42. PMID 11834461

Carpenter, D. O., Tarbell, A., Fitzgerald, E., Kadlec, M. J., O'Hehir, D., & Bush, B. (2002). University-community partnership for the study of environmental contamination at Akwesasne. In S. H. Wilson & W. A. Suk (Eds.), *Biomarkers of Environmentally Associated Disease* (pp. 507-523). Boca Raton, FL: Lewis Publishers

Carpi, A., & Chen, Y.-F. (2001). Gaseous Elemental Mercury as an Indoor Air Pollutant. *Environmental Science & Technology, 35*(21), 4170-4173. PMID 11718328

Carpy, S. A., Kobel, W., & Doe, J. (2000). Health Risk of Low-Dose Pesticides Mixtures: A Review of the 1985-1998 Literature on Combination Toxicology and Health Risk Assessment. *Journal of Toxicology and Environmental Health, 3*(Part B), 1-25. PMID 10711323

Carrier, G., Bouchard, M., Brunet, R., & Caza, M. (2001). A Toxicokinetic Model for Predicting the Tissue Distribution and Elimination of Organic and Inorganic Mercury Following Exposure to Methyl Mercury in Animals and Humans. II. Application and Validation of the Model in Humans. *Toxicology and Applied Pharmacology, 171*, 50-60. PMID 11181111. doi:10.1006/taap.2000.9113

Carrington, C., & Bolger, M. (2002, August). An exposure assessment for methylmercury from seafood for consumers in the United States. *Risk Analysis, 22*(4), 689-699. PMID 12224743

Carrizo, D., Grimalt, J. O., Ribas-Fito, N., Torrent, M., & Sunyer, J. (2007). *In utero* and post-natal accumulation of organochlorine compounds in children under different environmental conditions. *Journal of Environmental Monitoring, 9,* 523-529. PMID 17554423. doi:10.1039/b700247e

Carta, P., Flore, C., Alinovi, R., Ibba, A., Tocco, M. G., Aru, G., … Randaccio, F. S. (2003). Sub-Clinical Neurobehavioral Abnormalities Associated with Low Level of Mercury Exposure through Fish Consumption. *NeuroToxicology, 24,* 617-623. PMID 12900074. doi:10.1016/S0161-813X(03)00080-9

Carter-Pokras, O., & Bethune, L. (2009, October). Defining and Measuring Acculturation: A Systematic Review of Public Health Studies with Hispanic Populations in the United States. *Social Science & Medicine, 69*(7), 992-995. PMID 19631433. doi:10.1016/j.socscimed.2009.06.042

Cascorbi, I. (2006). Genetic basis of toxic reactions to drugs and chemicals. *Toxicology Letters, 162,* 16-28. PMID 16310984. doi:10.1016/j.toxlet.2005.10.015

Cassel, J. (1976, August). The contribution of the social environment to host resistance: The Fourth Wade Hampton Frost Lecture. *American Journal of Epidemiology, 104*(2), 107-123. PMID 782233

Castoldi, A., Blandini, F., Randine, G., Samuele, A., Manzo, L., & Coccini, T. (2006, September 27). Brain monoaminergic neurotransmission parameters in weanling rats after perinatal exposure to methylmercury and 2,2',4,4',5,5'-hexachlorobiphenyl (PCB153). *Brain Research, 1112*(1), 91-98. PMID 16904659. doi:10.1016/j.brainres.2006.07.022

Castorina, R., Bradman, A., McKone, T. E., Barr, D. B., Harnly, M. E., & Eskenazi, B. (2003, October). Cumulative Organophosphate Pesticide Exposure and Risk Assessment among Pregnant Women Living in an Agricultural Community: A Case Study from the CHAMACOS Cohort. *Environmental Health Perspectives, 111,* 1640-1648. PMID 14527844

Cave, M., Appana, S., Patel, M., Falkner, K., McClain, C., & Brock, G. (2010, December). Polychlorinated Biphenyls, Lead and Mercury are Associated with Liver Disease in American Adults: NHANES 2003-2004. *Environmental Health Perspectives, 118*(12), 1735-1742. PMID 21126940. doi:10.1289/ehp.1002720

Cecil, K. M., Brubaker, C. J., Adler, C. M., Dietrich, K. N., Altaye, M., Egelhoff, J. C., … Lanphear, B. P. (2008, May 27). Decreased Brain Volume in Adults with Childhood Lead Exposure. *PLoS Medicine, 5*(5), 741-750. PMID 18507499. doi:10.1371/journal.pmed.0050112

Cederbrant, K., Gunnarsson, L., & Marcusson, J. A. (2000). Mercury Intolerance and Lymphocyte Transformation Test with Nickel Sulfate, Palladium Chloride, Mercuric Chloride and Gold Sodium Thiosulfate. *Environmental Research, 84,* 140-144. PMID 11068927. doi:10.1006/enrs.2000.4079

Centers for Disease Control and Prevention. (1990, November 16). Notices to Readers National Minority Health Conference. *Mortality and Morbidity Weekly Report, 39*(45), 825. Retrieved April 1, 2011 from http://www.cdc.gov/mmwr/preview/mmwrhtml/00001836.htm

Centers for Disease Control and Prevention. (1998, April 3). Recommendations to Prevent and Control Iron Deficiency in the United States. *Mortality and Morbidity Weekly Report, 47*(RR-3), 1-36. PMID 9563847. Retrieved April 1, 2011 from http://www.cdc.gov/mmwr/preview/mmwrhtml/00051880.htm

Centers for Disease Control and Prevention (2001a). *Women and Smoking*. Retrieved April 1, 2011 from http://www.cdc.gov/tobacco/data_statistics/sgr/2001/complete_report/

Centers for Disease Control and Prevention (2001b). *Healthy People 2000 Final Review*. Retrieved April 1, 2011 from http://www.cdc.gov/nchs/data/hp2000/hp2k01.pdf

Centers for Disease Control and Prevention. (2004). *Pregnancy Risk Assessment and Monitoring System*. Retrieved April 1, 2011 from http://www.cdc.gov/reproductivehealth/tobaccousepregnancy/

Centers for Disease Control and Prevention. (2006, December). Advancing the Nation's Health: A Guide to Public Health Research Needs, 2006-2015. Atlanta, GA: Author

Centers for Disease Control and Prevention. (2009a, April 9). *Healthy People 2010 Impact Goals and Objectives.* Atlanta, GA: Author

Centers for Disease Control and Prevention. (2009d, October 30). *Healthy People 2020 Framework*. Retrieved April 1, 2011 from http://www.healthypeople.gov/hp2020/Objectives/TopicArea.aspx?id=20&TopicArea=Environmental+Health

Centers for Disease Control and Prevention. (2010). *Healthy People 2020 Objective Topic Areas*. Retrieved April 1, 2011 from http://www.healthypeople.gov/2020/topicsobjectives2020/default.aspx

Centers for Disease Control and Prevention, National Center for Environmental Health. (2001, March). *National Report on Human Exposure to Environmental Chemicals*. Atlanta, GA: Author

Centers for Disease Control and Prevention, National Center for Environmental Health. (2003, March). *Second National Report on Human Exposure to Environmental Chemicals (02-0716)*. Atlanta, GA: Author

Centers for Disease Control and Prevention, National Center for Environmental Health. (2005, July). *Third National Report on Human Exposure to Environmental Chemicals (05-0570).* Atlanta, GA: Author

Centers for Disease Control and Prevention, National Center for Environmental Health. (2007, March). *Chemicals measured in selected participants for NHANES 2003-2004.* Retrieved April 1, 2011 from http://oehha.ca.gov/multimedia/biomon/pdf/CDCChemicalsListHandout.pdf

Centers for Disease Control and Prevention, National Center for Environmental Health. (2009). *Fourth National Report on Human Exposure to Environmental Chemicals.* Retrieved April 1, 2011 from http://www.cdc.gov/exposurereport/

Centers for Disease Control and Prevention, National Center for Environmental Health. (2010, July). *Update for Fourth National Report on Human Exposure to Environmental Chemicals.* Atlanta, GA: Author

Centers for Disease Control and Prevention, National Center for Environmental Health. (2010, November). *Guidelines for the Identification and Management of Lead Exposure in Pregnant and Lactating Women.* Atlanta, GA: Author

Centers for Disease Control and Prevention, National Center for Health Statistics. (n.d.a). *General Information: Laboratory Methodology and Public Data Files*. Retrieved April 1, 2011 from http://www.cdc.gov/nchs/nhanes

Centers for Disease Control and Prevention, National Center for Health Statistics. (n.d.b). *National Health & Nutrition Examination Survey 1999-2010 Survey Content.* Retrieved April 1, 2011 from http://www.cdc.gov/nchs/data/nhanes/survey_content_99_10.pdf

Centers for Disease Control and Prevention, National Center for Health Statistics. (1993, September 23). *Analytical and Reporting Guidelines Appendix B: Joint Policy on Variance Estimation and Statistical Reporting Standards on NHANES III and CSFII Reports: Analytic Working Group Recommendations*. Retrieved April 1, 2011 from http://www.cdc.gov/nchs/data/nhanes/nhanes3/nh3gui.pdf

Centers for Disease Control and Prevention, National Center for Health Statistics. (2000, March). *Race and Ethnicity Code Set Version 1.0.* Retrieved April 1, 2011 from http://www.cdc.gov/phin/library/documents/pdf/Introduction_to_CDC_Race_and_Ethnicity_Code_Set_Version_1.0.pdf

Centers for Disease Control and Prevention, National Center for Health Statistics. (2003, February). *Dietary Interview Component Total Nutrient Intakes*. Retrieved April 1, 2011 from http://www.cdc.gov/nchs/data/nhanes/nhanes_03_04/dr1tot_c.pdf

Centers for Disease Control and Prevention, National Center for Health Statistics. (2004, June). *Summary of Surveys and Data Systems, National Center for Health Statistics*. Retrieved April 1, 2011 from http://www.cdc.gov/nchs/data/NCHS_Survey_Matrix.pdf

Centers for Disease Control and Prevention, National Center for Health Statistics. (2006c). *NHANES Homepage*. Retrieved April 1, 2011 from http://www.cdc.gov/nchs/mhanes.htm

Centers for Disease Control and Prevention, National Center for Health Statistics. (2006, September). *Analytic and Reporting Guidelines.* Retrieved April 1, 2011 from http://www.cdc.gov/nchs/nhanes/nhanes2003-2004/analytical_guidelines.htm

Centers for Disease Control and Prevention, National Center for Health Statistics. (2007a, September). *NHANES 1999-2000 Data Documentation: Nutritional Biochemistries*. Retrieved April 1, 2011 from http://cdc.gov/nchs/data/nhanes/nhanes_99_00/lab06_doc.pdf

Centers for Disease Control and Prevention, National Center for Health Statistics. (2007b, November). *NHANES 2003-2004 Data Documentation: Dietary*

Interview Total Nutrient Intakes (First Day). Retrieved April 1, 2011 from http://www.cdc.gov/nchs/data/nhanes/nhanes_03_04/dr1tot_c.pdf

Centers for Disease Control and Prevention, National Center for Health Statistics. (2008a, January). *NHANES Household Interview Sample Person Questionnaire: Occupation*. Retrieved April 1, 2011 from http://www.cdc.gov/nchs/data/nhanes/nhanes_03_04/ocq_c.pdf

Centers for Disease Control and Prevention, National Center for Health Statistics. (2008b, January). *NHANES 2003-2004 Documentation, Codebook and Frequencies: Occupation*. Retrieved April 1, 2011 from http://www.cdc.gov/nchs/data/nhanes/nhanes_03_04/ocq_c.pdf

Centers for Disease Control and Prevention, National Center for Health Statistics (2009a, January). *NHANES 1999-2000 Public Release Data File: Lab 28POC*. Retrieved April 1, 2011 from http://cdc.gov/nchs/data/nhanes/nhanes_99_00/l28podoc.pdf

Centers for Disease Control and Prevention, National Center for Health Statistics. (2009d, July 30). *About National Health and Nutrition Examination Survey (NHANES)*. Retrieved April 1, 2011 from http://www.cdc.gov/nchs/nhanes/about_nhanes.htm

Centers for Disease Control and Prevention, National Center for Health Statistics. (2009b, August). *Laboratory Methods 2001-2002*. Retrieved April 1, 2011 from http://www.cdc.gov/nchs/nhanes/nhanes2001-2002/lab_methods_01_02.htm

Centers for Disease Control and Prevention, National Center for Health Statistics. (2009c, September). *Data Documentation, Codebook and Frequencies: Demographic Variables*. Retrieved April 1, 2011 from http://www.cdc.gov/nchs/nhanes/nhanes2003-2004/DEMO_C.htm

Centers for Disease Control and Prevention, National Center for Health Statistics. (2009e, November). *Age Standardization and Population Counts*. Retrieved April 1, 2011 from http://www.cdc.gov/nchs/tutorials/nhanes/NHANESAnalyses/AgeStandardization/age_standardization_intro.htm

Centers for Disease Control and Prevention, National Center for Health Statistics. (2009f, November). *Hispanic Health and Nutrition Examination Survey (HHANES)*. Retrieved April 1, 2011 from http://www.cdc.gov/nchs/products/elec_prods/subject/hhanes.htm

Centers for Disease Control and Prevention, National Center for Health Statistics. (2009g, November). *NHANES Geocoding RDC Release Code Book*. Retrieved April 1, 2011 from http://www.cdc.gov/nchs/data/nhanes/nhanes_07_08/N0708_GE.pdf

Centers for Disease Control and Prevention, National Center for Health Statistics. (2009h, November). *Survey Overview and History*. Retrieved April 1, 2011 from http://www.cdc.gov/nchs/tutorials/NHANES/SurveyOrientation/SurveyOverview/intro.htm

Centers for Disease Control and Prevention, National Center for Health Statistics. (2009i, November). *Web Tutorial.* Retrieved April 1, 2011 from http://www.cdc.gov/nchs/tutorials/NHANES/index_current.htm

Centers for Disease Control and Prevention, National Center for Health Statistics. (2009j, December). *Laboratory Methods 2003-2004*. Retrieved April 1, 2011 from http://www.cdc.gov/nchs/nhanes/nhanes2003-2004/lab_methods_03_04.htm

Centers for Disease Control and Prevention, National Center for Health Statistics. (2010a, January). *Laboratory Methods 1999-2000*. Retrieved April 1, 2011 from http://www.cdc.gov/nchs/nhanes/nhanes1999-2000/lab_methods_99_00.htm

Centers for Disease Control and Prevention, National Center for Health Statistics. (2010b, January 6). *Who are Participants?* Retrieved April 1, 2011 from http://www.cdc.gov/nhanes/

Centers for Disease Control and Prevention, National Center for Health Statistics. (2010, February 4). *NCHS Research Data Center Disclosure Manual: Rules for Researchers*. Retrieved April 1, 2011 from http://www.cdc.gov/rdc/Data/B4/DisclosureManual.pdf

Centers for Disease Control and Prevention, National Center for Health Statistics. (2010c, May 21). *NCHS Research Ethics Review Board (ERB) Approval.* Retrieved April 1, 2011 from http://www.cdc.gov/nchs/nhanes/irba98.htm

Centers for Disease Control and Prevention, National Center for Health Statistics. (2010e, August 5). *Population Information: Bridged-Race Estimates.* Retrieved April 1, 2011 from http://wonder.cdc.gov

Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health (NIOSH). (2002, July). *Exposure Assessment Methods: Research Needs and Priorities*. Cincinnati, OH: National Institute for Occupational Safety and Health

Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health. (2004, December). *Mixed Exposures Research Agenda*. Cincinnati, OH: National Institute for Occupational Safety and Health

Cernichiari, E., Toribara, T. Y., Liang, L., Marsh, D. O., Berlin, M. W., Myers, G. J., … Clarkson, T. W. (1995). The Biological Monitoring of Mercury in the Seychelles Study. *NeuroToxicology, 16*(4), 613-628. PMID 8714867

Certosimo, A., Robertello, F., Dishman, M., & Bogacki, R. (2002, July-August). The effect of bleaching agents on mercury release from spherical dental amalgam. *General Dentistry, 51*(4), 356-360. PMID 15055616

Chaisson, C., & Solomon, G. (2001). Children's Exposure to Toxic Chemicals – Modeling their World to Quantify the Risks: Session V. Summary and Research Needs. *NeuroToxicology, 22*, 563-565. PMID 11770876

Challa, S. (2005). *Malnutrition in nursing home residents: differences across urban-rural continuum in a national sample*. (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 1431241)

Chalupka, S. (2002). *AAOHN/ATSDR Core Curriculum in Environmental Health.* Retrieved April 1, 2011 from https://www.aaohn.org/component/option,com_lms/Itemid,486/

Chambers, E. C., Duarte, C. S., & Yang, F. M. (2009, February). Household Instability, Area Poverty and Obesity in Urban Mothers and Their Children. *Journal of Health Care for the Poor and Underserved, 20*(1), 122-133. PMID 19202252. doi:10.1353/hpu.0.0110

Chan, M. H., Chan, I. H., Kong, A. P., Osaki, R., Cheung, R. C., Ho, C. S., … Lam, C. W. (2009, August). Cold-vapour atomic absorption spectrometry underestimates total mercury in blood and urine compared to inductively-coupled plasma mass spectrometry: an important factor for determining mercury reference intervals. *Pathology, 41*(5):467-72. PMID 19900086. doi:10.1080/00313020903041085

Chang, L. W., & Fu, C. S. (1990). Neuropathology of Heavy Metals and Its Modulation by Nutritional Influences. In E. C. Foulkes (Ed.), *Biological Effects of Heavy Metals, Volume 1* (pp. 69-96). Boca Raton, FL: CRC Press

Chang, Y., Yeh, C., & Wang, J. (1995). Subclinical Neurotoxicity of Mercury Vapor Revealed by A Multimodality Evoked Potential Study of Chloralkali Workers. *American Journal of Industrial Medicine, 27*, 271-279. PMID 7755016

Chaparro, C. M., Fornes, R., Nufeld, L. M., Alavez, G. T., Cedillo, R. E., & Dewey, K. G. (2007). Early Umbilical Cord Clamping Contributes to Elevated Blood Lead Levels Among Infants with Higher Lead Exposure. *Journal of Pediatrics, 151,* 506-512. PMID 17961694.doi:10.1016/j.jpeds.2007.04.056

Chapin, R. E., & Buck, G. M. (2004, January). Our Once-in-a-Lifetime Opportunity. *Environmental Health Perspectives, 112*(1), 67-68. PMID 14698933

Chapin, R. E., Robbins, W. A., Schieve, L. A., Sweeney, A. M., Tabacova, S. A., & Tomashek, K. M. (2004, January). Off to a Good Start: The Influence of Pre- and Periconceptual Exposures, Parental Fertility, and Nutrition on Children's Health. *Environmental Health Perspectives, 112*(1), 69-78. PMID 14698934

Charlson, M., Charlson, R., Peterson, J., Marinopoulos, S., Briggs, W., & Hollenberg, J. (2008). The Charlson comorbidity index is adapted to predict costs of chronic disease in primary care patients. *Journal of Clinical Epidemiology, 61*, 1234-1240. PMID 18619805. doi:10.1016/j.jclinepi.2008.01.006

Charlson, M., Pompei, P., Ales, K., & MacKenzie, C. (1987). A new method of classifying prognostic comorbidity in longitudinal studies: development and validation. *Journal of Chronic Diseases, 40*, 373-383. PMID 3558716

Chaudhuri, N. (2004, July-December). Interventions to improve children's health by improving the housing environment. *Reviews in Environmental Health, 19*(3-4), 197-222. PMID 15742671

Chaudry, R. V. (2008, May-June). The Precautionary Principle, Public Health and Public Health Nursing. *Public Health Nursing, 25*(3), 261-268. PMID 18477377. doi:10.1111/j.1525-1446.2008.00703.x

Checkoway, H., & Eisen, E. (2005). Epidemiology. In L. Rosenstock, M. R. Cullen, C. A. Brodkin & C. A. Redlich (Eds.), *Textbook of Clinical Occupational and Environmental Medicine* (2nd ed., pp. 120-137). Philadelphia, PA: Elsevier Saunders

Chen, H., Foo, S-H., & Ury, W. (2002, September). Recognizing dementia. *Western Journal of Medicine, 176*, 267-270

Chen, H. P., Paschal, D. C., Miller, D. T., & Morrow, J. C. (1998, September-October). Determination of Total and Inorganic Mercury in Whole Blood by On-Line Digestion with Flow Injection. *Atomic Spectroscopy, 19*(5), 176-179 doi:http://fen.selcuk.edu.tr/web/kimya/personel/ycengel/ContrAA/Atomic_Spectroscopy/AS19(5).PDF#page=36

Chen, J. J., Chen, Y.-J., Rice, G., Teuschler, L. K., Hamernik, K., Protzel, A., & Kodell, R. L. (2001). Using Dose Addition to Estimate Cumulative Risks from Exposures to Multiple Chemicals. *Regulatory Toxicology and Pharmacology, 34,* 35-41. PMID 11502154. doi:10.1006/rtph.2001.1485

Chen, J. J., Moon, H., & Kodell, R. L. (2007). A probabilistic framework for non-cancer risk assessment. *Regulatory Toxicology and Pharmacology, 48,* 45-50. PMID 17166641. doi:10.1016/j.yrtph.2006.10.008

Cheng, J., Yang, Y., Ma, J., Wang, W., Liu, X., Sakamoto, M., … Shi, W. (2009). Assessing noxious effects of dietary exposure to methylmercury, PCBs and Se

coexisting in environmentally contaminated rice in male mice. *Environment International, 35*, 619-625

Cherian, M. G., Hursh, J. B., Clarkson, T. W., & Allen, J. (1978, May-June). Radioactive Mercury Distribution in Biological Fluids and Excretion in Human Subjects after Inhalation of Mercury Vapor. *Archives of Environmental Health, 33*(3), 109-114. PMID 686833

Cherry, D., Lowry, L., Velez, L., Cotrell, C., & Keyes, D. C. (2002, January). Elemental Mercury Poisoning in a Family of Seven. *Family & Community Health, 24*(4), 1-8. PMID 11772345

Chetty, C., Rajanna, S., Hall, E., Yallapragada, P., & Rajanna, B. (1996, September). *In vitro* and *in vivo* effects of lead, methyl mercury and mercury on inositol 1,4,5-trisphosphate and 1,3,4,5-tetrakisphosphate receptor bindings in rat brain. *Toxicology Letters, 87*(1), 11-17. PMID 8701439

Child, C., & Turcotte, J. (1964). Surgery and portal hypertension. In C. Child (Ed.), *Liver and Portal Hypertension* (pp. 50-64). Phildelphia, PA: W. Saunders

Children's Health Act of 2000, Pub. L. No. 106, §310, 1004 (2000, October 17)

Chilton, M., Black, M. M., Berkowitz, C., Casey P. H., Cook, J., Cutts, D., … Frank, D. A. (2009a, March). Food insecurity and risk of poor health among U.S. born children of immigrants. *American Journal of Public Health, 99*(3), 556-562. PMID 19106417. doi:10.2105/AJPH.2008.144394

Chilton, M., & Rose, D. (2009b, July). A rights-based approach to food insecurity in the United States. *American Journal of Public Health, 99*(7), 1203-1211. PMID 19443834. doi: 10.2105/AJPH.2007.130229

Chiodo, L. M., Jacobson, S. W., & Jacobson, J. L. (2004). Neurodevelopmental effects of postnatal lead exposure at very low levels. *NeuroToxicology and Teratology, 26,* 359-371. PMID 15113598. doi:10.1016/j.ntt.2004.01.010

Chisholm, J. (1980). Lead and Other Metals: A Hypothesis of Interaction. In R. L. Singhai & J. A. Thomas (Eds.), *Lead Toxicity*. (pp. 461-482.) Baltimore, MD: Urban & Schwarzenberg

Choi, M., Afzal, B., & Sattler, B. (2006, September-October). Geographic information systems: a new tool for environmental health assessments. *Public Health Nursing, 23*(5), 381-391. PMID 16961558. doi:10.1111/j.1525-1446.2006.00577.x

Choi, S. D., Baek, S. Y., Chang, Y. S., Wania, F., Ikonomou, M. G., Yoon, Y. J., … Hong, S. (2008, October 1). Passive air sampling of polychlorinated biphenyls and organochlorine pesticides at the Korean Arctic and Antarctic research stations: implications for long-range transport and local pollution. *Environment and Science Technology, 42*(19), 7125-7131. PMID 18939536

Choi, Y., Seelbach, M., Pu, H., Eum, S., Chen, L., Zhang, B., … Toborek, M. (2010, July). Polychlorinated Biphenyls Disrupt Intestinal Integrity via NADPH Oxidase-Induced Alterations of Tight Junction Protein Expression. *Environmental Health Perspectives, 118*(7), 976-981. PMID 20299304

Chopoorian, T. J. (1986). Reconceptualizing the Environment. In P. Moccia (Ed.), *New Approaches to Theory Development* (pp. 39-54). New York, NY: National League for Nursing

Christakos, G. (2003). Critical Conceptualism in Environmental Modeling and Prediction. *Environmental Science & Technology, 37*(20), 4685-4693. PMID 14594379

Christiani, D., & Zhou, W. (2002, May). Hormesis: The New Approach in Risk Assessment? *Newsletter of Biological Effects of Low Level Exposures (BELLE)*, *10*(3), 12-13

Chromý, V., Rozkosná, K., & Sedlák, P. (2008). Determination of serum creatinine by Jaffe method and how to calibrate to eliminate matrix interference problems. *Clinical Chemistry and Laboratory Medicine, 46*(8), 1127-1133. PMID 18724810. doi:10.1515/CCLM.2008.224

Chuang, J. C., Callahan, P. J., Lyu, C. W., & Wilson, N. K. (1999). Polycyclic aromatic hydrocarbon exposures of children in low-income families. *Journal of Exposure Analysis and Environmental Epidemiology, 9*(2), 85-98. PMID 10321348

Chung, R. H., Kim, B. S., & Abreu, J. M. (2004). Asian American Multidimensional Acculturation Scale: Development, Factor Analysis, Reliability and Validity. *Cultural Diversity & Ethnic Minority Psychology, 10*(1), 66-80. PMID 14992631. doi:10.1037/1099-9809.10.1.66

Ciarapica, D., Mauro, B., Zaccaria, M., Cannella, C., & Polito, A. (2010, March-June). Validity of self-reported body weight and height among women including patients with eating disorders. *Eating and Weight Disorders, 15*(1-2), 74-80. PMID 20571324

Cicchetti, D. V., (2007, July). Prenatal Chlorpyrifos and Early Neurodevelopment: How Good is the Science? (Peer Commentary on the article "Impact of Prenatal Chlorpyrifos Exposure on Neurodevelopment in the First 3 Years of Life Among Inner-City Children" by Rauh, Garfinkel, Perera, Andrews, Hoepner, Barr, … Whyatt, 2006, December). *Pediatrics, 120*(1), 243. PMID 17606590. doi:10.1542/peds.2007-0689

Clark, M. J. (2008). *Community Health Nursing: Advocacy for Population Health* (5th ed.) Upper Saddle River, NJ: Pearson Education, Inc.

Clark, R., & Maynard, M. (1998, April). Research Methodology: Using Online Technology for Secondary Analysis of Survey Research Data. *Social Science Computer Review, 16*(1), 58-71

Clark, S. L., & Weismantle, M. (2003, August). *Employment Status: 2000*. Retrieved April 1, 2011 from http://www.census.gov/prod/2003pubs/c2kbr-18.pdf

Clarke, S. P., & Cossette, S. (2000, December). Secondary Analysis: Theoretical, Methodological and Practical Considerations. *Canadian Journal of Nursing Research, 32*(3), 109-129. PMID 11928128

Clarkson, T. W. (1997). The Toxicology of Mercury. *Critical Reviews in Clinical Laboratory Sciences, 34*(3), 369-403. PMID 9288445. doi:10.3109/10408369708998098

Clarkson, T. W., Amin-Zaki, L., & Al-Tikriti, S. K. (1976, October). An outbreak of methylmercury poisoning due to consumption of contaminated grain. *The Federation of American Societies for Experimental Biology Journal, 35*(12), 2395-2399

Clarkson, T. W. & Magos, L. (2006). The Toxicology of Mercury and its Chemical Compounds. *Critical Reviews in Toxicology, 36*, 609-662. PMID 16973445. doi:10.1080/10408440600845619

Clarkson, T. W., Magos, L., & Greenwood, M. R. (1972). The Transport of Elemental Mercury into Fetal Tissue. *Biology of the Neonate, 21,* 239-244. PMID 4656187

Clarkson, T. W., Magos, L., & Myers, G. J. (2003, October 30). The Toxicology of Mercury – Current Exposures and Clinical Manifestations. *The New England Journal of Medicine, 349*(18), 1731-1737. PMID 14585942. doi 10.1056/NEJMra022471

Clarkson, T. W., Vyas, J. B., & Ballatori, N. (2007, October). Mechanism of Mercury Disposition in the Body. *American Journal of Industrial Medicine, 50*(10), 757-764. PMID 17477364. doi 10.1002/ajim.20476

Clay, R. (1999, June). Still Moving Toward Environmental Justice. *Environmental Health Perspectives, 107*(6), A308-A310. PMID 10339458

Clayton, C. A., Pellizzari, E. D., Whitmore, R. W., Perritt, R. L., & Quackenboss, J. J. (1999). National Human Exposure Assessment Survey (NHEXAS): distributions and associations of lead, arsenic and volatile organic compounds in EPA Region 5. *Journal of Exposure Analysis and Environmental Epidemiology, 9*(5), 381-392. PMID 10554141

Clayton, C. A., Pellizzari, E. D., Whitmore, R. W., Quackenboss, J. J., Adgate, J., & Sexton, K. (2003). Distributions, associations, and partial aggregate exposure of pesticides and polynuclear aromatic hydrocarbons in the Minnesota

Children's Pesticide Exposure Study (MNCPES). *Journal of Exposure Analysis and Environmental Epidemiology, 13*(2), 100-111. PMID 12679790. doi:10.1038/sj.jea.7500261

Clayton, G. D. (1973). Introduction to Industrial Hygiene. In G. D. Clayton & W. D. Kelley (Eds.), *the Industrial Environment – its Evaluation & Control* (pp. 1-5). Washington, DC: U.S. Government Printing Office

Cleveland, L., Minter, M., Cobb, K., Scott, A., & German, V. (2008, November). Lead Hazards for Pregnant Women and Children: Part 2. *American Journal of Nursing, 108*(11), 40-47. PMID 18946264

Clougherty, J., & Kubzansky, L. (2009, September). A Framework for Examining Social Stress and Susceptibility to Air Pollution in Respiratory Health. *Environmental Health Perspectives, 117*(9), 1351-1358. PMID 19750097

Coccini, T., Manzo, L., Debes, F., Steuerwald, U., Weihe, P., & Grandjean, P. (2009, March). No changes in lymphocyte muscarinic receptors and platelet monoamine oxidase-B examined as surrogate central nervous system biomarkers in a Faroese children cohort prenatally exposed to methylmercury and polychlorinated biphenyls. *Biomarkers, 14*(2), 67-76. PMID 19330584. doi:10.1080/13547500902783739

Coccini, T., Randine, G., Castoldi, A., Grandjean, P., Ostendorp, G., Heinzow, B., & Manzo, L. (2006, July). Effects of developmental co-exposure to methylmercury and 2,2',4,4',5,5'-hexachlorobiphenyl (PCB153) on cholinergic muscarinic receptors in rat brain. *Neurotoxicology, 27*(4), 468-477. PMID 16455139. doi:10.1016/j.neuro.2005.12.004

Coccini, T., Roda, E., Castoldi, A., Goldoni, M., Poli, D., Bernocchi, G., & Manzo, L. (2007, August 16). Perinatal co-exposure to methylmercury and PCB153 or PCB126 in rats alters the cerebral cholinergic muscarinic receptors at weaning and puberty. *Toxicology, 238*(1), 34-48. PMID 17618726. doi:10.1016/j.tox.2007.05.018

Cocker, J., Mason, H. J., Garfitt, S. J., & Jones, K. (2002). Biological monitoring of exposure to organophosphate pesticides. *Toxicology Letters, 134,* 97-103. PMID 12191866

Colborn, T. (2006). A Case for Revisiting the Safety of Pesticides: A Closer Look at Neurodevelopment. *Environmental Health Perspectives, 114,* 10-17. PMID 16393651

Colborn, T., Smolen, M. J., & Rolland, R. (1998). Environmental Neurotoxic Effects: The Search for New Protocols in Functional Teratology. *Toxicology and Industrial Health, 14*(1/2), 9-23. PMID 9460167

Collaborative on Health and the Environment's Learning and Development Disabilities Initiative. (2008a, July 1). *Scientific Consensus Statement on Environmental Agents Associated with Neurodevelopmental Disorders.* Retrieved April 1, 2011 from http://www.iceh.org/pdfs/LDDI/LDDIStatement.pdf

Collaborative on Health and the Environment's Learning and Developmental Disabilities Initiative. (2008b, September 16). *Policy Implications Based on the Scientific Consensus Statement on Environmental Agents Associated with Neurodevelopmental Disorders.* Retrieved April 1, 2011 from http://www.iceh.org/pdfs/LDDI/LDDIStatement.pdf

Collins, K. (2007, November). Healthcare Workers' Perceptions of Occupational Hazards – Asking Questions to Guide Practice. *AAOHN Journal, 55*(11), 437-440. PMID 18019766

Commission of the European Communities. (2000, February 2). *Communication from the Commission on the Precautionary Principle*. Brussels, Belgium: Author

Congiu, L., Corongiu, F., Dore, M., Montaldo, C., Vargiolu, S., Casula, D., & Spiga, G. (1979, November). The effect of lead nitrate on the tissue distribution of mercury in rats treated with methylmercury chloride. *Toxicology and Applied Pharmacology, 51*(2), 363–366. PMID 531897

Connor, K. (2007). *Dementia care: classification and analysis of care management activities associated with caregiver mastery and relationship strain*. (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 3299571)

Connor, N., Monk, P., & Murray, V. (2000, June). Survey of how public health doctors in the United Kingdom and Republic of Ireland investigate the effects of long term exposure to point sources of chemicals. *Communicable Disease and Public Health, 3*(2), 127-131. PMID 10902256

Connors, S. L., Levitt, P., Matthews, S. G., Slotkin, T. A., Johnston, M. V., Kinney, H. C., … Zimmerman, A. W. (2008, March). Fetal Mechanisms in Neurodevelopmental Disorders. *Pediatric Neurology, 38*(3), 163-176. PMID 18279750. doi:10.1016/j.pediatrneurol.2007.10.009

Conrad, P., & Kern, R. (1981). *The Sociology of Health and Illness: Critical Perspectives*. New York: St. Martin's Press

Cordeiro, Q. Jr., de Araujo Medrado Faria, M., & Fraguas, R. Jr. (2003, Fall). Depression, insomnia and memory loss in a patient with chronic intoxication by inorganic mercury. *The Journal of Neuropsychiatry and Clinical Neurosciences, 15*(4), 457-458. PMID 1462777

Cory-Slechta, D. A. (1990, June 1). Lead exposure during advanced age: alterations in kinetics and biochemical effects. *Toxicology and Applied Pharmacology, 104*(1), 67-78. PMID 2360209

Cory-Slechta, D. A. (2005). Studying Toxicants as Single Chemicals: Does this Strategy Adequately Identify Neurotoxic Risk? *NeuroToxicology, 26,* 491-510. PMID 16112317. doi:10.1016/j.neuro.2004.12.007

Cory-Slechta, D. A., Virgolini, M. B., Thiruchelvam, M., Weston, D. D., & Bauter, M. R. (2004, May). Maternal Stress Modulates the Effects of Developmental Lead Exposure. *Environmental Health Perspectives, 112*(6), 717-730. PMID 15121516. doi:10.1289/ehp.6481

Costa, L. (1998). Signal transduction in environmental neurotoxicity. *Annual Review of Pharmacology & Toxicology, 38*(1), 21-43. PMID 9597147. doi:10.1146/annurev.pharmtox.38.1.21

Costa, L., Aschner, M., Vitalone, A., Syversen, T., & Soldin, O. (2004). Developmental neuropathology of environmental agents. *Annual Review of*

Pharmacology and Toxicology, 44(1), 87-110. PMID 14744240. doi:10.1146/annurev.pharmtox.44.101802.121424

Costa, L., Fattori, V., Giordano, G., & Vitalone, A. (2007, July 31). An *in vitro* approach to assess the toxicity of certain food contaminants: methylmercury and polychlorinated biphenyls. *Toxicology, 237*(1-3), 65-76. PMID 17553607. doi:10.1016/j.tox.2007.05.003

Costa, L., Guizzetti, M., Lu, H., Bordi, F., Vitalone, A., Tita, B., … Silvestrini, B. (2001, March 7). Intracellular signal transduction pathways as targets for neurotoxicants. *Toxicology, 160*(1-3), 19-26. PMID 11246120

Costa, L., Richter, R. J., Li, W., Cole, T., Guizzetti, M., & Furlong, C. E. (2003). Paraoxonase (PON1) as a biomarker of susceptibility for organophosphate toxicity. *Biomarkers, 8*(1), 1-12. PMID 12519632. doi:10.1080/13547500210148315

Counter, S. A. (2003, January). Neurophysiological Anomalies in Brainstem Responses of Mercury-Exposed Children of Andean Gold Miners. *Journal of Occupational and Environmental Medicine, 45*(1), 87-95. PMID 12553183

Courtwright, A. (2009). Justice, Stigma and the new Epidemiology of Health Disparities. *Bioethics, 23*(2), 90-96. PMID 19531162. doi:10.1111/j.1467-8519.2008.00717.x

Cox, C., Clarkson, T. W., Marsh, D. O., Amin-Zaki, L., Tikriti, S., & Myers, G. O. (1989). Dose-Response Analysis of Infants Prenatally Exposed to Methyl Mercury: An Application of a Single Compartment Model to Single-Strand Hair Analysis. *Environmental Research, 49,* 318-332. PMID 2473897

Cox, S., Niskar, A., Narayan, K., & Marcus, M. (2007, December). Prevalence of self-reported diabetes and exposure to organochlorine pesticides among Mexican Americans: Hispanic health and nutrition examination survey, 1982-1984. *Environmental Health Perspectives, 115*(12), 1747-1752. PMID 18087594. doi:10.1289/ehp.10258

Cralley, L. J., & Cralley, L. V. (1985). Rationale. In L. J. Cralley & L. V. Cralley (Eds.), *Patty's Industrial Hygiene and Toxicology* (2nd ed., Vol. 2, pp. 1-20). New York, NY: John Wiley & Sons

Cranor, C. F. (2004). Toward Understanding Aspects of the Precautionary Principle. *Journal of Medicine and Philosophy, 29*(3), 259-279

Crocetti, A., Mushak, P., & Schwartz, J. (1990, November). Determination of numbers of lead-exposed women of childbearing age and pregnant women: an integrated summary of a report to the U.S. Congress on childhood lead poisoning. *Environmental Health Perspectives, 89*, 121-124. PMID 2088737

Chromý, V., Rozkosná, K., & Sedlák, P. (2008). Determination of serum creatinine by Jaffe method and how to calibrate to eliminate matrix interference problems. *Clinical Chemistry Laboratory Medicine, 46*(8), 1127-1233. PMID 18724810

Crump, K., Chen, C., & Louis, T. (2010, October). The Future Use of *in vitro* Data in Risk Assessment to Set Human Exposure Standards: Challenging Problems and Familiar Solutions. *Environmental Health Perspectives, 118*(10), 1350-1354. PMID 20562051. doi:10.1289/ehp.1001931

Cummings, A., & Kavlock, R. (2004). Gene-Environment Interactions: A Review of Effects on Reproduction and Development. *Critical Reviews in Toxicology, 34*(6), 461-485. PMID 15609483

Cullen, M. (2005). Low-Level Environmental Exposures. In L. Rosenstock, M. R. Cullen, C. A. Brodkin & C. A. Redlich (Eds.), *Textbook of Clinical Occupational and Environmental Medicine* (2nd ed., pp. 1127-1132). Philadelphia, PA: Elsevier Saunders

Cullen, M., & Rosenstock, L. (2005). Introduction to Occupational and Environmental Medicine. In L. Rosenstock, M. R. Cullen, C. A. Brodkin, & C. A. Redlich (Eds.), *Textbook of Clinical Occupational and Environmental Medicine* (2nd ed., pp. 3-15). Philadelphia, PA: Elsevier Saunders

Curley, A., Sedlák, V. A., Girling, E. F., Hawk, R. E., Barthel, W. F., Pierce, P. E., & Likosky, W. H. (1971, April 2). Organic Mercury Identified as the Cause of Poisoning in Humans and Hogs. *Science, 172*(3978), 65-67. PMID 5102013

Cutter, S. (2001, February). A Research Agenda for Vulnerability Science and Environmental Hazards. *Newsletter of the International Human Dimensions Programme on Global Environmental Change*. Bonn, Germany: International Human Dimensions Programme on Global Environmental Change

Dai, D., & Oyana, T. J. (2008, October 21). Spatial variations in the incidence of breast cancer and potential risks associated with soil dioxin contamination in Midland, Saginaw, and Bay Counties, Michigan, USA. *Environmental Health, 7,* 1-49. PMID 18939976. doi:10.1186/1476-069X-7-49

Dales, R., Lui, L., Wheeler, A. J., & Gilbert, N. L. (2008, July 15). Quality of indoor residential air and health. *Canadian Medical Association Journal, 179*(2), 147-152. PMID 18625986. doi:10.1503/cmaj.070359

Daniels, J. L., Longnecker, M. P., Klebanoff, M. A., Gray, K. A., Brock, J. W., Zhou, H., … Needham, L. L. (2003). Prenatal Exposure to Low-Level Polychlorinated Biphenyls in Relation to Mental and Motor Development at 8 Months. *American Journal of Epidemiology, 157*(6), 485-492. PMID 12631537

Danovero-Holliday, M. C., Gordon, E. R., Woernie, C., Higginbotham, G. H., Judy, R. H., Icenogle, J. P., & Reef, S. E. (2003, February). Identifying Risk Factors for Rubella Susceptibility in a Population at Risk in the United States. *American Journal of Public Health, 93*(2), 289-291. PMID 12554588

Darga, K. (1999). *Sampling and the Census: a case against the proposed adjustments for undercount*. Washington, D.C.: The AEI Press

Darga, K. (2001). *Fixing the Census Until It Breaks: An Assessment of the Undercount Adjustment Puzzle*. Lansing, MI: Michigan Information Center

Darvill, T., Lonky, E., Reihman, J., Stewart, P., & Pagano, J. (2000, December). Prenatal exposure to PCBs and infant performance on the Fagan test of infant intelligence. *NeuroToxicology, 21*(6), 1029-1038. PMID 11233749

Davidson, P. W., Myers, G. J., Cox, C., Axtell, C., Shamlaye, C., Sloan-Reeves, J., … Clarkson, T. W. (1998, August 26). Effects of Prenatal and Postnatal Methylmercury Exposure from Fish Consumption on Neurodevelopment: outcomes at 66 months of age in the Seychelles child development study. *Journal of the American Medical Association, 280*(8), 701-707. PMID 9728641

Davidson, P. W., Myers, G. J., & Weiss, B. (2004, April). Mercury Exposure and Child Development Outcomes. *Pediatrics, 113*(4), 1023-1029. PMID 15060195

Davis, K., Crow, J., Chambers, H., Meek, E., & Chambers, J. (2009, August). Racial Differences in Paraoxonase-1 (PON1): A Factor in the Health of Southerners? *Environmental Health Perspectives, 117*(8), 1226-1231. PMID 19672401

Davis, J. M., Otto, D. A., Weil, D. E., & Grant, L. D. (1990, May-June). The Comparative Developmental Neurotoxicity of Lead in Humans and Animals. *NeuroToxicology and Teratology, 12*(3), 215-229. PMID 2196421

Day, R. D., Segars, A. L., Arendt, M. D., Lee, A. M., & Peden-Adams, M. M. (2007). Relationship of Blood Mercury Levels to Health Parameters in the Loggerhead Sea Turtle (Caretta caretta). *Environmental Health Perspectives, 115,* 1421-1428. PMID 17938730. doi: 10.1289/ehp.9918

de Burbure, C., Buchet, J., Leroyer, A., Nisse, C., Haguenoer, J., Mutti, A., … Bernard, A. (2006, April). Renal and Neurologic Effects of Toxic Metals in Children. *Environmental Health Perspectives, 114*(4), 584-590. PMID 16581550

de Chesnay, M. (2005). Vulnerable Populations: Vulnerable People. In M. de Chesnay (Ed.) *Caring for the Vulnerable: Perspectives in Nursing Theory, Practice and Research*, (pp. 3-12). Sudbury, MA: Jones and Bartlett Publishers

de Chesnay, M., Wharton, R., & Pamp, C. (2005). Cultural Competence, Resilience and Advocacy. In M. de Chesnay (Ed.) *Caring for the Vulnerable: Perspectives in Nursing Theory, Practice and Research* (pp. 31-41). Sudbury, MA: Jones and Bartlett Publishers

de Coster, S., Koppen, G., Bracke, M., Schroijen, C., Den Hond, E., Nelen, V., ... van Larebeke, N. (2008). Pollutant effects on genotoxic parameters and tumor-associated protein levels in adults: a cross sectional study. *Environmental Health 7*(26), 1-59. PMID 18522717. doi:10.1186/1476-069X-7-26

Dederick, E. J. (2008). *Methods in exposure analysis for vulnerable subpopulations* (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 3309635)

de Fur, P. L., Evans, G. W., Cohen, E. A., Kyle, A. D., Morello-Frosch, R. A., & Williams, D. R. (2007, May). Vulnerability as a Function of Individual and Group Resources in Cumulative Risk Assessment. *Environmental Health Perspectives, 115*(5), 817-824. PMID 17520073. doi:10.1289/ehp.9332

de Groot, V., Beckerman, H., Lankhorst, G., & Bouter, L. (2003). How to measure comorbidity: a critical review of available methods. *Journal of Clinical Epidemiology, 56*, 221-229. PMID 12725876

Del Bene Davis, A. (2006). *Home environmental health risks of people with developmental disabilities* (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 3247724)

Del Boca, F., & Darkes, J. (2003). The validity of self-reports of alcohol consumption: state of the science and challenges for research. *Addiction, 98*(Supplement 2), 1-12

Dellinger, J. A. (2004, July). Exposure assessment and initial intervention regarding fish consumption of tribal members of the Upper Great Lakes Region in the United States. *Environmental Research, 95*(3), 325-340. PMID 15220067. doi:10.1016/j.envres.2003.07.012

Denham, M., Schell, L. M., Deane, G., Gallo, M. V., Ravenscroft, J., DeCaprio, A. P., & the Akwesasne Task Force on the Environment. (2005, February). Relationship of lead, mercury, mirex, dichlorodiphenyldichloroethylene, hexachlorobenzene and polychlorinated biphenyls to timing of menarche among Akwesasne Mohawk girls. *Pediatrics, 115*(2), 27-34. PMID 15653789. doi:10.1542/peds.2004-1161

de Olivera Santos, E. C., de Jesus, I. M., da Silva Brabo, E., Brito Loureiro, E. C., da Silva Mascarenhas, A. F., Weirich, J., … Cleary, D. (2000). Mercury Exposures in Riverside Amazon Communities in Para, Brazil. *Environmental Research, 84*(2)*,* 100-107. PMID 11068923

de Palma, G., Mariotti, O., Lonati, D., Goldoni, M. Catalani, S., Mutti, A., … Apostoli, P. (2008). Toxicokinetics and toxicodynamics of elemental mercury following self-administration. *Clinical Toxicology, 46*(9), 869-876. PMID 18787993. doi:10.1080/15563650802136241

de Rosa, C. T., El-Masri, H. A., Pohl, H., Cibulas, W., & Mumtaz, M. M. (2004). Implications of Chemical Mixtures in Public Health Practice. *Journal of Toxicology and Environmental Health Critical Reviews Section B, 7,* 339-350. PMID 9487091

de Rosa, C. T., Stevens, Y. W., Wilson, J. D., Ademoyero, A. A., Buchanan, S. D., Cibulas, W. Jr., … Williams-Johnson, M. M. (1993, November). The Agency for Toxic Substances and Disease Registry's role in development and application of biomarkers in public health practice. *Toxicology and Industrial Health, 9*(6), 979-994. PMID 8191504

Derr, J., & Orris, P. (2005). Persistent Organic Pollutants. In L. Rosenstock, M. R. Cullen, C. A. Brodkin, & C. A. Redlich (Eds.), *Textbook of Clinical Occupational and Environmental Medicine* (2nd ed., pp. 1061-1071). Philadelphia, PA: Elsevier Saunders

Derrickson, J. P., Fisher, A. G., & Anderson, J. E. (2000, November). The core food security module scale measure is valid and reliable when used with Asian and Pacific Islanders. *The Journal of Nutrition, 130*(11), 2666-2674. PMID 11053505

Desaiah, D. (1990). Action of Metals on Calmodulin-Regulated Calcium Pump Activity in Brain. In E. C. Foulkes, (Ed.), *Biological Effects of Heavy Metals, Volume 1* (pp. 60-68). Boca Raton, FL: CRC Press

Desenclos, J.-C., Vaillant, J.-C., Astagneau, E., Campese, C., Che, B., Coignard, L., … de Valk, H. (2007). Principles of an outbreak investigation in public health practice (translated to English). *Médecine et maladies infectieuses, 37*, 77-94. PMID 17196781

Després, C., Beuter, A., Richer, F., Poitras, K., Veilleux, A., Ayotte, P., … Muckle, G. (2005, March-April). Neuromotor functions in Inuit preschool children exposed to Pb, PCBs and Hg. *Neurotoxicology and Teratology, 27*(2), 245-257. PMID 15734276. doi: 10.1016/j.ntt.2004.12.001

Detsky, A. S., McLaughlin, J. R., Baker, J. P., Johnston, N., Whittaker, S., Mendelson, R. A., & Jeejeebhoy, K. N. (1987, January-February). What is Subjective Global Assessment of Nutritional Status? *Journal of Parenteral and Enteral Nutrition, 11*(1), 8-13. PMID 3820522

Dettling, A., Witte, S., Skopp, G., Graw, M., & Haffner, H. T. (2009, September). A regression model applied to gender-specific ethanol elimination rates from blood and breath measurements in non-alcoholics. (Abstract.) *International Journal of Legal Medicine, 123*(5), 381-385. PMID 18839202. doi:10.1007/s00414-008-0282-y

Dewailly, É., Ayotte, P., Laliberte, C., Weber, J.-P., Gingras, S., & Nantel, A. J. (1996, September). Polychlorinated Biphenyl (PCB) and Dichlorodiphenyl Dichloroethylene (DDE) Concentrations in the Breast Milk of Women in Quebec. *American Journal of Public Health, 86*(9), 1241-1246. PMID 8806375

Dewailly, É., Ayotte, P., Rhainds, M., Bruneau, S., Fugal, C., Grondin, J., … Muckle, G. (2002). Application of biomarkers to population studies on food chain contaminants in Nunavik (Artic Quebec, Canada). In S. H. Wilson & W. A. Suk (Eds.), *Biomarkers of environmentally associated disease* (pp. 495-505). Boca Raton, FL: Lewis Publishers

Deyo, R. A., Cherkin, D. C., & Ciol, M. A. (1992, June). Adapting a clinical co-morbidity index for use with ICD-9-CM administrative databases. *Journal of Clinical Epidemiology, 45*(6), 613-619. PMID 1607900

Diamond, G. L. (1988). Biological Monitoring of Urine for Exposure to Toxic Metals. In T. W. Clarkson, L. Friberg, G. F. Nordberg & P. R. Sager (Eds.), *Biological Monitoring of Toxic Metals* (pp. 515-526). New York, NY: Plenum Press

Diawara, M. M., Litt, J. S., Unis, D., Alfonso, N., Martinez, L.-A., Crock, J., … Carsella, J. (2006, August). Arsenic, cadmium, lead, and mercury in surface soils, Pueblo, Colorado: implications for population health risk. *Environmental and Geochemical Health, 28*(4), 297-315. PMID 16752202. doi:10.1007/s10653-005-9000-6

Dietrich, K. N. (1999, January). Environmental chemicals and child development. *Journal of Pediatrics, 134*(1), 7-9. PMID 9880440

Dietrich, K. N., & Bellinger, D. (1994). The Assessment of Neurobehavioral Development in Studies of the Effects of Prenatal Exposure to Toxicants. In H. L. Needleman & D. Bellinger (Eds.), *Prenatal Exposure to Toxicants: Developmental Consequences* (pp. 57-85). Baltimore, MD: The Johns Hopkins University Press

Dietrich, K. N., Eskenazi, B., Schantz, S., Yolton, K., Rauh, V. A., Johnson, C. B., … Berman, R. F. (2005). Principles and Practices of Neurodevelopmental Assessment in Children: Lessons Learned from the Centers for Children's Environmental Health and Disease Prevention Research. *Environmental Health Perspectives, 113,* 1437-1446. PMID 16203260

Dietrich, K. N., Krafft, K. M., Bornschein, R. L., Succop, P. A., & Bier, M. (1987, November 5). Low-Level Fetal Lead Exposure Effect on Neurobehavioral Development in Early Infancy. *Pediatrics, 80*(5), 721-730. PMID 2444921

Dietrich, K. N., Ris, M. D., Succop, P. A., Berger, O. G., & Bornschein, R. L. (2001). Early exposure to lead and juvenile delinquency. *NeuroToxicology and Teratology, 23*, 511-518. PMID 11792521

Dietrich, K. N., Succop, P. A., Bornshein, R. L., Krafft, K. M., Berger, O., Hammond, P. B., & Buncher, C. R. (1990). Lead Exposure and Neurobehavioral Development in Later Infancy. *Environmental Health Perspectives, 89,* 13-19. PMID 2088739

DiPietro, E., Lapeza, C. R. Jr., Cash, T. P., Turner, W. E., Green, V. E., Gill, J. B., & Patterson, D. G. Jr. (1997). A fast universal automated cleanup system for the isotope-dilution high-resolution mass spectrometric analysis of PCDDs, PCDFs, coplanar PCBs, PCB congeners and persistent pesticides from the same serum sample. *Organohalogen Compounds, 31*, 26-31

Dix, K. (2001). Absorption, Distribution, and Pharmacokinetics. In R. I. Kreiger (Ed.), *Handbook of Pesticide Toxicology* (2nd ed., Vol. I, pp. 563-582). San Diego, CA: Academic Press

Dixon, S. L., Gaitens, J. M., Jacobs, D. E., Strauss, W., Nagaraja, J., Pivetz, T., … Ashley, P. J. (2009, March). Exposure of U.S. children to residential dust lead, 1999-2004: II. The contribution of lead-contaminated dust to children's blood lead levels. *Environmental Health Perspectives, 117*(3), 468-474. PMID 19337524

Dong, M., & Ross, J. (2001). Coping with Aggregate Pesticide Exposure Assessment: An Integration Approach. In R. I. Kreiger (Ed.), *Handbook of Pesticide Toxicology* (2nd ed., Vol. I, pp. 479-491). San Diego, CA: Academic Press

Donnan, P. T., McLernone, D., Steinke, D., Ryder, S., Roderick, P., Sullivan, F., … Dillon, J. F. (2007, April 16). Development of a decision support tool to facilitiate primary care management of patients with abnormal liver function tests without clinically apparent liver disease: Abnormal Liver Funtion Investigations Evaluation (ALFIE). *BMC Health Services Research*, *7*, 1-54. PMID 17437630. doi:10.1186/1472-6963-7-54

Dooley, D., Fielding, J., & Levi, L. (1996). Health and Unemployment. *Annual Review in Public Health, 17*, 449-465. PMID 8724235. doi:10.1146/annurev.pu.17.050196.002313

Dórea, J. G. (2004). Mercury and lead during breast-feeding. *British Journal of Nutrition, 92,* 21-40. PMID 15230985. doi:10.1079/BJN20041163

Dórea, J. G. (2008, October). Effects of Prenatal Exposure to Pollutants on Children's Development: Additional Issues. (Peer Commentary on article “Effects of prenatal exposure to coal-burning pollutants on children’s development in China” by Tang, Li, Liu, Zhou, Yuan, Chen, … Perera, 2008, October). *Environmental Health Perspectives, 116*(10), A418-A421. PMID 18941549. doi:10.1289/ehp.11763

Dórea, J. G., & Barbosa, A. C. (2003). Maternal Mercury Transfer. *Environmental Research, 93,* 113-114. PMID 12963394

Dos Santos, A. P., Mateus, M. L., Carvalho, C. M., & Batoreu, M. C. (2007). Biomarkers of exposure and effect as indicators of the interference of selenomethionine on methylmercury toxicity. *Toxicology Letters, 169,* 121-128. PMID 17267146. doi:10.1016/j.toxlet.2006.12.007

Dos Santos, C. R., Rodrigues, R. S., Silva, C. S., Nascimento, E. S. (2006, August). Determination of lead in whole blood by electrothermal atomic absorption spectrometry using Zeeman correction and sample stability. *Journal of Radioanalytical & Nuclear Chemistry***,** 269(2), 481-485. AN 22751169. doi:10.1007/s10967-006-0411-3

Doull, J., & Rozman, K. K. (2000, August 14). Using Haber's Law to define the margin of exposure. *Toxicology, 149*(1), 1-2. PMID 10963856

Dourson, M., Charnley, G. & Scheuplein, R. (2002). Differential Sensitivity of Children and Adults to Chemical Toxicity. II. Risk and Regulation. *Regulatory Toxicology and Pharmacology, 35*, 448-467. PMID 12202058

Downs, M. H., Kaminsky, A., & Lewis, J. (2006, September-October). Open the door whenever opportunity knocks. *Public Health Nursing, 23*(5), 433-441. PMID 16961562. doi:10.1111/j.1525-1446.2006.00581.x

Driscoll, C. D., Steissguth, P., & Riley, E. P. (1990, May-June). Prenatal Alcohol Exposure: Comparability of Effects in Humans and Animal Models. *NeuroToxicology and Teratology, 12*(3), 231-237. PMID 2196422

Drukker, M., Kaplan, C., & van Os, J. (2005, June). Residential instability in socioeconomically deprived neighbourhoods, good or bad? *Health & Place, 11*(2), 121-129. PMID 15629680. doi:10.1016/j.healthplace.2004.02.002

Drew, C., Barnes, M., Phelps, J., & Van Houten, B. (2008, April). NIEHS Extramural Global Environmental Health Portfolio: Opportunities for Collaboration. *Environmental Health Perspectives, 116*(4), 421-425. PMID 18414621. doi:10.1289/ehp.11323

Dreyling, E., Dederick, E. J., Chari, R., Resnick, B., Malecki, K. C., Burke, T., & Neff, R. (2007, December). Tracking health and the environment: a pilot test of environmental public health indicators. *Journal of Environmental Health, 70*(5), 9-16, 38, 40. PMID 18189034

Dube, S., Asman, K., Malarcher, A., & Carabollo, R. (2009, November 13). Cigarette Smoking Among Adults and Trends in Smoking Cessation – United States, 2008. *Morbidity and Mortality Weekly Report, 58*(44), 1227-1232. PMID 19910909

Dubos, R. (1977, Spring). The Despairing Optimist. *American Scholar, 46*(2), 152-158.

Dubos, R. (1980). *Man Adapting* (enlarged ed.). New Haven, CT: Yale University Press

Dubos, R. (1983a). Environment. In M. Allaby (Ed.), *A Dictionary of the Environment*, (2nd ed., pp. 208-210.) New York, NY: New York University Press

Dubos, R. (1983b). Human Adaptation. In M. Allaby (Ed.), *A Dictionary of the Environment*, (2nd ed., pp. 337-338.) New York, NY: New York University Press

Dufault, R., Schnoll, R., Lukiw, W. J., Leblanc, B., Cornett, C., Patrick, L., … Crider, R. (2009, October 27). Mercury exposure, nutritional deficiencies and metabolic disruptions may affect learning in children. *Behavior and Brain Function, 5*(44), 1-15. PMID 19860886. doi:10.1186/1744-9081-5-44

Dunn, J. R. (2002, September). Housing and inequalities in health: a study of socioeconomic dimensions of housing and self-reported health from a survey of Vancouver residents. *Journal of Epidemiology and Community Health, 56*(9), 671-681. PMID 12177083. doi:10.1136/jech.56.9.671

du Plooy, H. C. (2005). *An investigation into the degree of correlation between occupational hygiene and occupational medicine data* (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 0667980)

Dupuy, A. (2009). Determination of serum ferritin using immunoturbidimetry or chemiluminescent detection in comparison with radioimmunoassay a compendium of a methodological juxtaposition. *Clinical Laboratory, 55*(5-6), 207-215. PMID 19728554

Dwyer, J., Picciano, M.-F., Raiten, D., & Members of the Steering Committee. (2003, February). Estimation of Usual Intakes: What We Eat in America – NHANES. *The Journal of Nutrition, 133*(2), 609S-623S. PMID 12566511

Dye, B. A., Schober, S. E., Dillon, C. F., Jones, R. L., Fryar, C., McDowell, M., & Sinks, T. H. (2005). Urinary mercury concentrations associated with dental restorations in adult women aged 16-49 years: United States, 1999-2000. *Occupational and Environmental Medicine, 62,* 368-375. PMID 15901883. doi:10.1136/oem.2004.016832

Eaton, D. L. (2005). Toxicology. In L. Rosenstock, M. R. Cullen, C. A. Brodkin & C. A. Redlich (Eds.), *Textbook of Clinical Occupational and Environmental Medicine* (2nd ed., pp. 83-118). Philadelphia, PA: Elsevier Saunders

Eaton, D. L., Daroff, R. B., Autrup, H., Bridges, J., Buffler, P., Costa, L. G., … Spencer, P. S. (2008). Review of the Toxicology of Chlorpyrifos with an Emphasis on Human Exposure and Neurodevelopment. *Critical Reviews in Toxicology, 38*(1), 1-125. PMID 18726789. doi:10.1080/10408440802272158

Eaton, D. L., & Klaassen, C. D. (2003). Principles of Toxicology. In C. D. Klaassen & J. B. Watkins (Eds.), *Casarett and Doull's Essentials of Toxicology* (pp. 6-20). New York, NY: McGraw-Hill

Ebi, K., & Patz, J. (2002). Epidemiological and impacts assessment methods. In P. Martens & A. J. McMichael (Eds.), *Environmental Change, Climate and Health* (pp. 120-143). Cambridge, U.K.: Cambridge University Press

Ebmeier, K. (2010, January). Normal cognitive decline or dementia? *The Practitioner, 254*(1725), 23-27

Echeverria, D., Vasken Aposhian, H., Woods, J. S., Heyer, N. J., Aposhian, M. M., Bittner, A. C. Jr., … Cianciola, M. (1998, August). Neurobehavioral effects from exposure to dental amalgam Hg^0: new distinctions between recent exposure and Hg^0 body burden. *The Federation of American Societies for Experimental Biology Journal, 12,* 971-980. PMID 9707169

Echeverria, D., Heyer, N. J., Martin, M. D., Naleway, C. A., Woods, J. S., & Bittner, A. C. (1995). Behavioral Effects of Low-Level Exposure to Hg^0 Among Dentists. *NeuroToxicology and Teratology, 17*(2), 161-168. PMID 7760775

Editor. (1959, May). Minamata Disease. *Nutrition Reviews, 17*(5), 139-141. PMID 13644822

Editor. (1969, November). White House Conference on Food, Nutrition and Health, December 2-4, 1969, Washington, DC. *Journal of Infectious Diseases, 120*(5), 637-642. PMID 5346542

Edmondson, M. E., & Williamson, G. C. (1998, January). Environmental Health Education for Health Professionals and Communities. *AAOHN Journal, 46*(1), 14-19. PMID 9481215

Egeghy, P. P., Quackenboss, J. J., Catlin, S., & Ryan, P. B. (2005, September). Determinants of temporal variability in NHEXAS – Maryland environmental concentrations, exposures, and biomarkers. *Journal of Exposure Analysis and Environmental Epidemiology, 15*(5), 388-397. PMID 15602583. doi:10.1038/sj.jea.7500415

Eighth International Conference on Mercury as a Global Pollutant. The Madison Declaration on Mercury Pollution. *Ambio*, 36(1), 62-65. PMID 17408191

Eisenhardt, K. M. (1989, October). Building Theories from Case Study Research. *The Academy of Management Review, 14*(4), 532-550

Eknoyan, G. (2005, January). Emergence of Quantification in Clinical Investigation and the Quest for Certainty in Therapeutics: The Road From Hammurabi to Kefauver. *Advances in Chronic Kidney Disease, 12*(1), 88-95. PMID 15719339

Elberger, S. T. (1993). Cadmium, Mercury and Arsenic. In P. Viccellio (Ed.), *Handbook of Medical Toxicology*. (pp. 285-293). Boston, MA: Little Brown

Elhassani, S. B. (1982, October). The Many Faces of Methylmercury Poisoning. *Journal of Toxicology and Clinical Toxicology, 19*(8), 875-906. PMID 6763633

Elinder, C., Gerhardsson, L., & Oberdoerster, G. (1988). Overview. In T. W. Clarkson, L. Friberg, G. F. Nordberg & P. R. Sager (Eds.), *Biological Monitoring of Toxic Metals* (pp. 1-74). New York, NY: Plenum Press

Elliott, K. C. (2006, December). A Novel Account of Scientific Anomaly: Help for the Dispute Over Low-Dose Biochemical Effects. *Philosophy of Science, 73*, 790-802

Ellis, K. J. (1988). In Vivo Monitoring of Toxic Metals: Assessment of Neutron Activation and X-Ray Fluorescence Techniques. In T. W. Clarkson, L. Friberg, G. F. Nordberg & P. R. Sager (Eds.), *Biological Monitoring of Toxic Metals* (pp. 499-514). New York, NY: Plenum Press

Ellman, L., & Sunstein, C. (2004, September). Hormesis, the Precautionary Principle and Legal Regulation. *Newsletter of Biological Effects of Low Level Exposures (BELLE)*, *12*(2), 2-12

el-Masri, H., Reardon, K., & Yang, R. (1997). Integrated Approaches for the Analysis of Toxicologic Interactions of Chemical Mixtures. *Critical Reviews in Toxicology, 27*(2), 175-197. PMID 9099518. doi:10.3109/10408449709021618

Environmental Justice. (2009, February 12). Retrieved April 1, 2011 from http://www.epa.gov/compliance/environmentaljustice/index.html

Erci, B. (2005). Nursing Theories Applied to Vulnerable Populations. In M. de Chesnay (Ed.) *Caring for the Vulnerable: Perspectives in Nursing Theory, Practice and Research* (pp. 45-60). Sudbury, MA: Jones and Bartlett Publishers

Ercolano, E., Hendrickson, K. C., & Dixon, J. (2008, July-August). Talking with patients about environmental health: the "I talk" mnemonic. *Holistic Nursing Practice, 22*(4), 197-205. PMID 18607232

Eriksson, P., Ankarberg, E., Viberg, H., & Fredriksson, A. (2001, January). The developing cholinergic system as target for environmental toxicants, nicotine and polychlorinated biphenyls (PCBs): implications for neurotoxicological processes in mice. *Neurotoxicology Research, 3*(1), 37-51. PMID 15111260

Ernhart, C. B., Morrow-Tlucak, M., Marler, M. R., & Wolf, A. W. (1987, May-June). Low Level Lead Exposure in the Prenatal and Early Preschool Periods: Early Preschool Development. *NeuroToxicology and Teratology, 9*(3), 259-270. PMID 2442586

Ernhart, C. B. (1996, June 12). Bone Lead Levels and Delinquent Behavior. (Peer Commentary on article "Bone lead levels in adjudicated delinquents: A case control study" by Needleman, McFarland, Ness, Fienberg & Tobin, 2002b, November-December). *Journal of the American Medical Association, 275*(22), 1726. PMID 8637163

Ervasti, M., Kotisaari, S., Heinonen, S., & Punnonen, K. (2007). Use of advanced red blood cell and reticulocyte indices improves the accuracy in diagnosing iron deficiency in pregnant women at term. *European Journal of Haematology, 79*, 539-545. PMID 17976190. doi:10.1111/j.1600-0609.2007.00964.x

Escriba-Aguir, V., & Perez-Hoyos, S. (2007). Psychological well-being and psychosocial work environment characteristics among emergency medical and nursing staff. *Stress and Health, 23*, 153-160. AN 2009657204

Eskenazi, B., Bradman, A., & Castorina, R. (1999, June). Exposures of Children to Organophosphate Pesticides and Their Potential Adverse Health Effects. *Environmental Health Perspectives, 107*(Supplement 3), 409-419. PMID 10346990

Eskenazi, B., Harley, K., Bradman, A., Weltzien, E., Jewell, N. P., Barr, D. B., … Holland, N. T. (2004, July). Association of *in utero* Organophosphate Pesticide Exposure and Fetal Growth and Length of Gestation in an Agricultural Population. *Environmental Health Perspectives, 112*(10), 1116-1124. PMID 15238287

Eskenazi, B., Marks, A. R., Bradman, A., Harley, K., Barr, D. B., Johnson, C., … Jewell, N. P. (2007, May). Organophosphate Pesticide Exposure and

Neurodevelopment in Young Mexican-American Children. *Environmental Health Perspectives, 115*(5), 792-798. PMID 17520070. doi:10.1289/ehp.9828

Eskenazi, B., Rosas, L. G., Marks, A. R., Bradman, A., Harley, K. Holland, N., … Barr, D. B. (2008). Pesticide Toxicity and the Developing Brain. *Basic & Clinical Pharmacology & Toxicology, 102*, 228-236. PMID 18226078

Esposito, C., & Dal Canton A. (2010, September-October). Functional changes in the aging kidney. *Journal of Nephrology, 23*(Supplement 15), S41-S45. PMID 20872370

Espinoza, E. O., Mann, M., & Bleasdell, B. (1995, September 21). Arsenic and Mercury in Traditional Chinese Herbal Balls. *The New England Journal of Medicine, 333*(12), 803-804. PMID 7643901

Espinoza, E. O., Mann, M., Bleasdell, B., DeKorte, S., & Cox, M. (1996, May). Toxic Metals in Selected Traditional Chinese Medicinals. *Journal of Forensic Sciences, 41*(3), 453-456

Etkina, E. I., & Etkina, I. A. (1995, July). Chemical Mixtures Exposure and Children's Health. *Chemosphere, 31*(1), 2463-2474. PMID 7670860

Eto, K. (2000, September). Minamata disease. *Neuropathology, 20*(Supplement), 14-19. PMID 11037181

Ettinger, A. S., Lamadrid-Figueroa, H., Téllez-Rojo, M., Mercado-Garcia, A., Peterson, K. E., Schwartz, J., … Hernández-Avila, M. (2009, January). Effect of calcium supplementation on blood lead levels in pregnancy: a randomized placebo-controlled trial. *Environmental Health Perspectives, 117*(1), 26-31. PMID 19165383

Ettinger, A. S., Hu, H., & Hernandez-Avila, M. (2007, March). Dietary calcium supplementation to lower blood lead levels in pregnancy and lactation. *Journal of Nutrition and Biochemistry, 18*(3), 172-178. PMID 17296490. doi:10.1016/j.jnutbio.2006.12.007

Ettinger, A. S., Téllez-Rojo, M. M., Amarasiriwardena, C., Peterson, K. E., Schwartz, J., Aro, A., … Hernández-Avila, M. (2006, January 1). Influence of maternal bone lead burden and calcium intake on levels of lead in breast milk over the course of lactation. *American Journal of Epidemiology, 163*(1), 48-56. PMID 16282237. doi:10.1093/aje/kwj010

Ettinger, A. S., Téllez-Rojo, M. M., Amarasiriwardena, C., Bellinger, D., Peterson, K., Schwartz, J., … Hernández-Avila, M. (2004a, October). Effect of breast milk lead on infant blood lead levels at 1 month of age. *Environmental Health Perspectives, 112*(14), 1381-1385. PMID 15471729

Ettinger, A. S., Téllez-Rojo, M. M., Amarasiriwardena, C., González-Cossío, T., Peterson, K. E., Aro, A., … Hernández-Avila, M. (2004b, June). Levels of Lead in Breast Milk and Their Relation to Maternal Blood and Bone Lead Levels at One Month Postpartum. *Environmental Health Perspectives, 112*(8), 926-931. PMID 15175184

Ettinger, H. (2003, November-December). Industrial Hygienists: Who We Are, Priorities, Goals, Limitations. *AIHA Journal, 64*(6), 724-729. PMID 14696594

Eubig, P., Aguiar, A., & Schantz, S. (2010, December). Lead and PCBs as Risk Factors for Attention Deficit Hyperactivity Disorder. *Environmental Health Perspectives, 118*(12), 1654-1667. PMID 20829149. doi:10.1289/ehp.0901852

Evans, R. D., Addison, E. M., Villeneuve, J. Y., MacDonald, K. S., & Joachim, D. G. (2000, October). Distribution of Inorganic and Methylmercury among Tissues in Mink (Mustela vison) and Otter (Lutra canadensis). *Environmental Research, 84*(2), 133-139. PMID 11068926. doi:10.1006/enrs.2000.4077

Executive Order No. 12898, 3 C.F.R. 859 (1995), *reprinted as amended in* 42 U.S.C. § 4321 at 73, § 3-301(b) (1994 & Supplement VI 1998). Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations

Exposure. (n.d.). In *Oxford English Dictionary*. Retrieved April 1, 2011 from http://0-www.oed.com.helin.uri.edu

Ezzati, M., Friedman, A., Kulkarni, S., & Murray, C. (2008). The Reversal of Fortunes: Trends in County Mortality and Cross-County Mortality Disparities in the United States. *PLoS Medicine, 5*(4), 1-12. PMID 18433290. doi:10.1371/journal.pmed.0050066

Ezzati, M., Hoorn, S. V., Lopez, A. D., Danaei, G., Rodgers, A., & Mathers, C. D. (2006). Comparative Quantification of Mortality and Burden of Disease Attributable to Selected Risk Factors. In A. D. Lopez, C. D. Mathers, M. Ezzati, D. T. Jamison, & C. J. Murray (Eds.), *Global Burden of Disease and Risk Factors* (pp. 241-259). Oxford, UK: Oxford University Press

Fang, M., Boobis, A. R., & Edwards, R. J. (2007, November). Searching for novel biomarkers of centrally and peripherally-acting neurotoxicants, using surface-enhanced laser desorption/ionisation-time-of-flight mass spectrometry (SELDI-TOF MS). *Food and Chemical Toxicology, 45*(11), 2126-2137. PMID 17602814. doi:10.1016/j.fct.2007.05.007

Faria, M., Carrasco, L., Diez, S., Riva, M., Bayona, J., & Barata, C. (2009, April). Multi-biomarker responses in the freshwater mussel Dreissena polymorpha exposed to polychlorobiphenyls and metals. *Comparative Biochemistry and Physiology Part C: Toxicolgy & Pharmacology, 149*(3), 281-288. PMID 18723121. doi:10.1016/j.cbpc.2008.07.012

Faroon, O., Jones, D. & de Rosa, C. (2000). Effects of polychlorinated biphenyls on the nervous system. *Toxicology and Industrial Health, 16*, 305-333. PMID 12117298

Faupel-Badger, J., Chung-Cheng, H., Troisi, R., Lagiou, P., & Potischman, N. (2007, September). Plasma Volume Expansion in Pregnancy: Implications for Biomarkers in Population Studies. *Cancer Epidemiology, Biomarkers & Prevention, 16*(9), 1720-1723. PMID 7855687. doi:http://cebp.aacrjournals.org/lookup/doi/10.1158/1055-9965.EPI-07-0311

Faut, M., Rodríguez de Castro, C., Bietto, F. M., Castro, J. A., & Castro, G. D. (2009, September). Metabolism of ethanol to acetaldehyde and increased susceptibility to oxidative stress could play a role in the ovarian tissue cell injury promoted by alcohol drinking. *Toxicology and Industrial Health, 25*(8), 525-538. PMID 19825859. doi:10.1177/0748233709345937

Fawcett, J., & Malinski, V. M. (1996). On the requirements for a metaparadigm: a invitation to dialogue. *Nursing Science Quarterly, 9*(3), 94-97, 100-101. PMID 8850982

Feldman, D. (1990). Reconceptualizing the Nature and Consequences of Part-Time Work. *The Academy of Management Review, 15*(1), 103-112. PMID 10106335

Feldman, R. G. (1999). *Occupational and Environmental Neurotoxicology*. Philadelphia, PA: Lippincott-Raven

Felix-Ortiz, M., Newcomb, M. D., & Myers, H. (1994, May). A multidimensional measure of cultural identity for Latino and Latina adolescents. *Hispanic Journal of Behavioral Sciences, 16*(2), 99-115. doi: 10.1177/07399863940162001

Ferrari-Goelzer, B. (1998). Goals, Definitions and General Information. In J. M. Stellman (Ed.), *Encyclopedia of Occupational Health & Safety* (4th ed., Section 30). Geneva, CH: International Labour Organisation

Ferrario, J., Byrne, C., & Dupuy, A. E. (1997, June). Background Contamination by Coplanar Polychlorinated Biphenyls (PCBs) in Trace Level High Resolution Gas Chromatography/High Resolution Mass Spectrometry (HRGC/HRMS) Analytical Procedures. *Chemosphere, 34*(11), 2451-2465. PMID 9192469

Ferrier, H., Shaw, G., Nieuwenhuijsen, M., Boobis, A., & Elliott, P. (2006, June). Assessment of uncertainty in a probabilistic model of consumer exposure to pesticide residues in food. *Food Additives and Contaminants, 23*(6), 601-615. PMID 16766459. doi:10.1080/02652030600573244

Fiebig, E. W., Johnson, D. K., Hirschkorn, D. F., Knape, C. C., Webster, H. K., Lowder, J., & Busch, M. P. (1997). Lymphocyte subset analysis on frozen whole blood. *Cytometry, 29*, 340-350

Fierens, S., Mairesse, H., Heilier, J.-F., de Burbure, C., Focant, J.-F., Eppe, G., … Bernard, A. (2003, November-December). Dioxin/polychlorinated biphenyl body burden, diabetes and endometriosis: findings in a population-based study in Belgium. *Biomarkers, 8*(6), 529-534. AN 12511429

Filakti, H., & Fox, J. (1995, Autumn). Differences in mortality by housing tenure and by car access from the OPCS Longitudinal Study. *Population Trends, 81*, 27-30. PMID 8528790

First, M. W. (1978). Air Sampling and Analysis for Contaminants in Workplaces. In M. Lippmann (Ed.), *Air Sampling Instruments for evaluation of atmospheric contaminants* (5th ed., pp. A1-A14). Cincinnati, OH: American Conference of Governmental Industrial Hygienists

Fiscella, K., Franks, P., Doescher, M., & Saver, B. (2002, January). Disparities in healthcare by race, ethnicity and language among the insured: findings from a national sample. *Medical Care, 40*(1), 52-59. PMID 11748426

Fischer, C., Fredriksson, A., & Eriksson, P. (2008, February 28). Neonatal co-exposure to low doses of an ortho-PCB (PCB 153) and methylmercury exacerbate defective developmental neurobehavior in mice. *Toxicology, 244*(2-3), 157-165. PMID 18155821. doi:10.1016/j.tox.2007.11.006

Fisher, G. M. (1997, September). *The Development of the Orshansky Poverty Thresholds and Their Subsequent History as the Official U.S. Poverty Measure*. Retrieved April 1, 2011 from http://www.census.gov/hhes/www/povmeas/papers/orshansky.html#C2

Fitzpatrick-Lewis, D., Yost, J., Ciliska, D., & Krishnaratne, S. (2010, November 1). Communication about environmental health risks: a systematic review. *Environmental Health, 9*, 67. PMID 21040529. doi:10.1186/1476-069X-9-67

Flaskerud, J., & Winslow, B. (1998, March-April). Conceptualizing Vulnerable Populations Health-Related Research. *Nursing Research, 47*(2), 69-78. PMID 9536190

Fleming, D. E., Chettle, D. R., Wetmur, J. G., Desnick, R. J., Robin, J.-P, Boulay, D., … Weber, C. E. (1998). Effect of the δ–Aminolevulinate Dehydratase Polymorphism on the Accumulation of Lead in Bone and Blood in Lead Smelter Workers. *Environmental Research A, 77*, 49-61. PMID 9593628. doi:10.1006/enrs.1997.3818

Flores, G., Abreu, M., & Tomany-Korman, S. (2005, July-August). Limited English Proficiency, Primary Language at Home and Disparities in Children's Health Care: How Language Barriers are Measured Matters. *Public Health Reports, 120*(4), 418-430. PMID 16025722

Foa, V., & Alessio, L. (1998). General Principles. In J. M. Stellman (Ed.), *Encyclopedia of Occupational Health & Safety* (4th ed., Section 27.2). Geneva, CH: International Labour Organisation

Fontaine, J., Dewailly, É., Benedetti, J.-L., Pereg, D., Ayotte, P., & Déry, S. (2008, June 2). Re-evaluation of blood mercury, lead and cadmium concentrations in the Inuit population of Nunavik (Quebec): a cross-sectional study. *Environmental Health, 7*(1), 25. PMID 18518986. doi:10.1186/1476-069X-7-25

Fontana, V., Baldi, R., Franchini, M., Gridelli, P., Neri, R., Palmieri, F., … Parodi, S. (2004, March). Adverse haematological outcome and environmental lead poisoning. *Journal of Exposure Analysis and Environmental Epidemiology, 14*(2), 188-193. PMID 15014550. doi:10.1038/sj.jea.7500318

Forbes, V. E., Palmqvist, A., & Bach, L. (2006, January). The Use and Misuse of Biomarkers in Ecotoxicology. *Environmental Toxicology and Chemistry, 25*(1), 272-280. PMID 16494252

Fortin, M-C., Carrier, G., & Bouchard, M. (2008, November 4). Concentrations versus amounts of biomarkers in urine: a comparison of approaches to assess pyrethroid exposure. *Environmental Health, 7*(55), 1-45. PMID 18983658. doi:10.1186/1476-069X-7-55

Foster, S., Vaughan, R., Foster, W., & Califano, J. (2003, February 26). Alcohol Consumption and Expenditures for Underage Drinking and Adult Excessive Drinking. *Journal of the American Medical Association, 289*(8), 989-995. PMID 12597750. doi:10.1001/jama.289.8.989

Foulkes, E. C. (1990). *Biological Effects of Heavy Metals, Volume 1*. Boca Raton, FL: CRC Press

Fowle, J. R. III, & Sexton, K. (1992, November). EPA priorities for biologic markers research in environmental health. *Environmental Health Perspectives, 98*, 235-241. PMID 1486855

Fowles, E., & Gabrielson, M. (2005, Summer). First trimester predictors of diet and birth outcomes in low-income pregnant women. *Journal of Community Health Nursing, 22*(2), 117-130. PMID 15877540. doi:10.1207/s15327655jchn2202_5

Frame, G. M. (2001). The Current State-of-the-Art of Comprehensive, Quantitative, Congener-Specific PCB Analysis and What We Now Know about the Distributions of Individual Congeners in Commercial Aroclor Mixtures. In L. W. Robertson & L. G. Hansen (Eds.), *PCBs: Recent Advances in Environmental Toxicology and Health Effects*. (pp. 3-9.) Lexington, KY: The University Press of Kentucky

Francis, E. Z., Kimmel, C. A., & Rees, D. C. (1990, May-June). Workshop on the Qualitative and Quantitative Comparability of Human and Animal Developmental Neurotoxicology, Summary and Implications. *NeuroToxicology and Teratology, 12*(3), 275-280. PMID 2196427

Franzblau, A., & Fromes, M. (2005). Mercury. In L. Rosenstock, M. Cullen, C. A. Brodkin, & C. A. Redlich (Eds.), *Textbook of Clinical Occupational and Environmental Medicine* (2nd ed., pp. 979-982). Philadelphia, PA: Elsevier Saunders

Frasco, M. F., Fournier, D., Carvalho, F., & Guilhermino, L. (2005, September-October). Do metals inhibit acetylcholinesterase (AChE)? Implementation of assay conditions for the use of AChE activity as a biomarker of metal toxicity. *Biomarkers, 10*(5), 360-375. AN 19118733

Fraser, G. E., & Yan, R. (2007, January). A Multivariate Method for Measurement Error Correction Using Pairs of Concentration Biomarkers. *Annals of Epidemiology, 17*(1), 64-73. PMID 17140813. doi:10.1016/j.annepidem.2006.08.002

Fraumeni, J. F. (1974, May). Chemicals in human teratogenesis and transplacental carcinogenesis. *Pediatrics, 53*(5), 807-812. PMID 4138009

Frazis, H., Harrison-Ports, M., & Stewart, J. (1995, September). Comparing measures of educational attainment in the CPS. *Monthly Labor Review, 118*(9), 40-44

Freedman, L. S., Guenther, P. M., Dodd, K. W., Krebs-Smith, S. M., & Midthune, D. (2010, January). The population distribution of ratios of usual intakes of dietary components that are consumed every day can be estimated from repeated 24-hour recalls. *Journal of Nutrition, 140*(1), 111-116. PMID 19923394. doi:10.3945/jn.109.110254

Freeman, H. (1989, July 1). Cancer in the economically disadvantaged. *Cancer, 64*(Supplement 1), 324-334. PMID 2655875

Freeman, H. (1991, April 17). Race, poverty and cancer. *Journal of the National Cancer Institute, 83*(8), 526-527, 551-557. PMID 2005635

Freeman, R. (1963). *Public health nursing practice* (3rd ed.) Philadelphia, PA: W.B. Saunders

Friberg, L., & Vostal, J. (1972). *Mercury in the Environment: An Epidemiological and Toxicological Appraisal*. Cleveland, OH: CRC Press

Fritsche, E., Cline, J. E., Nguyen, N., Scanlan, T. S., & Abel, J. (2005, July). Polychlorinated Biphenyls Disturb Differentiation of Normal Human Neural Progenitor Cells: Clue for Involvement of Thyroid Hormone Receptors. *Environmental Health Perspectives, 113*(7), 871-876. PMID 16002375

Frongillo, E. A. Jr. (1999, February). Validation of measures of food insecurity and hunger. *The Journal of Nutrition, 129*(Supplement 2), 506S-509S. PMID 10064319

Frohlich, K. L., Dunn, J. R., McLaren, L., Shiell, A., Potvin, L., Hawe, P., … Thurston, W. E. (2007, June). Understanding place and health: A heuristic for using administrative data. *Health & Place, 13*(2), 299-309. AN 2009522475

Frustaci, A., Magnavita, N., Chimenti, C., Caldarulo, M., Sabbioni, E., Pietra, R., … Maseri, A. (1999, May). Marked Elevation of Myocardial Trace Elements in Idiopathic Dilated Cardiomyopathy Compared with Secondary Cardiac

Dysfunction. *Journal of the American College of Cardiology, 33*(6), 1578-1583. PMID 10334427

Fryar, C., Merino, M., Hirsch, R., & Porter, K. (2009, May 20). Smoking, Alcohol Use and Illicit Drug Use Reported by Adolescents Aged 12 to 17 years: United States, 1999-2004. *National Health Statistics Reports, 15*, 1-28

Fukata, H., Omori, M., Osada, H., Todaka, E., & Mori, C. (2005, March). Necessity to Measure PCBs and Organochlorine Pesticide Concentrations in Human Umbilical Cords for Fetal Exposure Assessment. *Environmental Health Perspectives, 113*(3), 297-303. PMID 15743718

Fukuda, Y., Ushijima, K., Kitano, T., Sakamoto, M., & Futatsuka, M. (1999, August). An Analysis of Subjective Complaints in a Population Living in a Methylmercury-Polluted Area. *Environmental Research A, 81*(2), 100-107. PMID 10433841. doi:10.1006/enrs.1999.3970

Fung, Y. K., Meade, A. G., Rack, E. P., & Blotcky, A. J. (1997, January). Brain mercury in neurodegenerative disorders. *Journal of Toxicology: Clinical Toxicology, 35*(1), 49-56. PMID 9022652

Furumoto-Dawson, A., Gehlert, S., Sohmer, D., Olopade, O., & Sacks, T. (2007, September-October). Early-Life Conditions and Mechanisms of Population Health Vulnerabilities. *Health Affairs, 26*(5), 1238-1248. PMID 17848432. doi:10.1377/hlthaff.26.5.1238

Futatsuka, M., Kitano, T., Shono, M., Nagano, M., Wakamiya, J., Miyamoto, K., … Osame, M. (2005). Long-Term Follow-Up Study of Health Status in Population Living in Methylmercury-Polluted Area. *Environmental Sciences, 12*(5), 239-282. PMID 16308560

Gabbe, S. G., Niebyl, J. R., & Simpson, J. L. (2007). *Obstetrics: normal and problem pregancies*, 5th edition. Philadelphia, PA: Churchill Livingstone Elsevier

Gabriel, P. E., & Schmitz, S. (2007, June). Gender differences in occupational distributions among workers. *Monthly Labor Review*, *130*(6), 19-24

Gallacher, J., Elwood, P., Phillips, K., Davies, B., & Jones, D. (1984, January). Relation between pica and blood lead in areas of differing lead exposure. *Archives of Disease in Children, 59*(1), 40-44. PMID 6696493

Gallo, L. C., Penedo, F. J., Espinosa de los Monteros, K., & Arguelles, W. (2009, December). Resiliency in the face of disadvantage: do Hispanic cultural characteristics protect health outcomes? *Journal of Personality, 77*(6), 1707-1746. PMID 19796063. doi:10.1111/j.1467-6494.2009.00598.x

Gamo, M., Oka, T., & Nakanishi, J. (2003, October). Ranking the risks of 12 major environmental pollutants that occur in Japan. *Chemosphere, 53*(4), 277-284. PMID 12946386. doi:10.1016/S0045-6535(03)00053-5

Garcia, S. J., Seidler, F. J., & Slotkin, T. A. (2003, March). Developmental Neurotoxicity Elicited by Prenatal or Postnatal Chlorpyrifos Exposure: Effects on Neurospecific Proteins Indicate Changing Vulnerabilities. *Environmental Health Perspectives, 111*(3), 297-303. PMID 12611658

Gauthier, T. W., Kable, J. A., Burwell, L., Coles, C. D., & Brown, L. A. (2010, January). Maternal Alcohol Use During Pregnancy Causes Systemic Oxidation of the Glutathione Redox System. *Alcohol Clinical and Experimental Research, 34*(1), 1-8. PMID 19860801. doi:10.1111/j.1530-0277.2009.01072.x

Gee, G. C., & Payne-Sturges, D. C. (2004, December). Environmental Health Disparities: A Framework Integrating Psychosocial and Environmental Concepts. *Environmental Health Perspectives, 112*(17), 1645-1653. PMID 15579407

Gelobter, M. (1992). Toward a Model of "Environmental Discrimination." In B. Bryant & P. Mohai (Eds.), *Race and the Incidence of Environmental Hazards* (pp. 64-81). Boulder, CO: Westview Press

George, T. S. (2001). *Minamata: Pollution and the Struggle for Democracy in Postwar Japan*. Boston, MA: Harvard University Press

Georgopoulos, P. G., & Lioy, P. J. (2006, November-December). From a Theoretical Framework of Human Exposure and Dose Assessment to Computational System Implementation: the Modeling ENvironment for TOtal Risk Studies

(MENTOR). *Journal of Toxicology and Environmental Health B Critical Reviews, 9*(6), 457-483. PMID 17090483. doi:10.1080/10937400600755929

Georgopoulos, P. G., Sasso, A. F., Sastry, S., Lioy, P. J., Vallero, D. A., Okino, M., & Reiter, L. (2009, February). Reconstructing population exposures to environmental chemicals from biomarkers: challenges and opportunities. *Journal of Exposure Science & Environmental Epidemiology, 19*(2), 149-171. PMID 18368010. doi:10.1038/jes.2008.9

Gerber, D. E., & McGuire, S. L. (1999). Teaching Students About Nursing and the Environment: Part I – Nursing Role and Basic Curricula. *Journal of Community Health Nursing, 16*(2), 69-79. PMID 10349818

Geronimus, A., & Hillemeier, M. (1992, Summer). Patterns of blood lead levels in U.S. black and white women of childbearing age. *Ethnicity & Disease, 2*(3), 222-231. PMID 1467759

Gesink Law, D., Klebanoff, M. A., Brock, J. W., Dunson, D. B., & Longnecker, M. P. (2005, September 15). Maternal Serum Levels of Polychlorinated Biphenyls and 1,1-Dichloro-2,2-bis(p-chlorophenyl)ethylene (DDE) and Time to Pregnancy. *American Journal of Epidemiology, 162*(6), 523-532. PMID 16093292. doi:10.1093/aje/kwi240

Gibičar, D. D., Logar, M., Horvat, N., Marn-Pernat, A., Ponikvar, R., & Horvat, M. (2007, May). Simultaneous determination of trace levels of ethylmercury and methylmercury in biological samples and vaccines using sodium tetra(*n*-propyl)borate as derivatizing agent. *Analytical and Bioanalytical Chemistry, 388*(2), 329-340. PMID 17340078. doi: 10.1007/s00216-007-1208-0

Gibson, M. V., Diaz, V. A., Mainous, A. G. III, & Geesey, M. E. (2005, June). Prevalence of Breastfeeding and Acculturation in Hispanics: Results from NHANES 1999-2000 Study. *Birth, 32*(2), 93-98. PMID 15918865. doi:10.1111/j.0730-7659.2005.00351.x

Ginevan, M. E., Ross, J. H., & Watkins, D. K. (2009, February). Assessing exposure to allied ground troops in the Vietnam War: A comparison of AgDRIFT and Exposure Opportunity Index models. *Journal of Exposure Science & Environmental Epidemiology, 19*(2), 187-200. PMID 18335003. doi:10.1038/jes.2008.12

Giovaniello, T. J., Bendetto, G., Palmer, D. W., & Peters, T. (1968, May). Fully and semi-automated methods for the determination of serum iron and total iron-binding capacity. *Journal of Laboratory and Clinical Medicine, 71*(5), 874-883. PMID 5647688

Gladen, B. C., Doucet, J., & Hansen, L. G. (2003, April). Assessing Human Polychlorinated Biphenyl Contamination for Epidemiologic Studies: Lessons from Patterns of Congener Concentrations in Canadians in 1992. *Environmental Health Perspectives, 111*(4), 437-443. PMID 12676596

Gladen, B. C., Ragan, N. B., & Rogan, W. J. (2000, April). Pubertal growth and development and prenatal and lactational exposure to polychlorinated biphenyls and dichlorodiphenyl dichloroethene. *Journal of Pediatrics, 136*(4), 490-496. PMID 10753247

Gluckman, P. D., & Hanson, M. A. (2004, September 17). Living with the Past: Evolution, Development and Patterns of Disease. *Science, 305,* 1733-1736. PMID 15375258. doi:10.1126/science.1095292

Gluckman, P. D., Hanson, M. A., & Beedle, A. S. (2007, January-February). Early Life Events and Their Consequences for Later Disease: A Life History and Evolutionary Perspective. *American Journal of Human Biology, 19*(1), 1-19. PMID 17160980. doi:10.1002/ajhb.20590

Gluckman, P. D., Hanson, M. A., Cooper, C., & Thornburg, K. L. (2008, July 3). Effect of *In Utero* and Early-Life Conditions on Adult Health and Disease. *The New England Journal of Medicine, 359*(1), 61-73. PMID 18596274. doi:10.1056/NEJMra0708473

Glynn, A., Atuma, S., Aune, M., Darnerud, P. O., & Cnattingius, S. (2001, July). Polychlorinated Biphenyl Congeners as Markers of Toxic Equivalents of Polychlorinated Biphenyls, Dibenzo-p-dioxins and Dibenzofurans in Breast Milk. *Environmental Research A, 86*(3), 217-228. PMID 11453672. doi:10.1006/enrs.2001.4270

Glynn, A. W., Aune, M., Darnerud, P. O., Cnattingius, S., Bjerselius, R., Becker, W., & Lignell, S. (2007, February). Determinants of serum concentrations of

organochlorine compounds in Swedish pregnant women: a cross-sectional study. *Environmental Health, 6,*1-2. PMID 17266775. doi:10.1186/1476-069X-6-2

Glynn, A. W., Thuvander, A., Johannisson, A., Ola, P., Ronquist, G. & Cnattingius, S. (2008, December 4). Immune cell counts and risks of respiratory infections among infants exposed pre- and post-natally to organochlorine compounds: a prospective study. *Environmental Health, 7*(62), 1–43. PMID 19055819. doi:10.1186/1476-069X-7-62

Glynn, A. W., Wolk, A., Aune, M., Atuma, S., Zettermark, S., Mæhle-Schmid, M., … Adami, H.-O. (2000, December 18). Serum concentrations of organochlorines in men: a search for markers of exposure. *The Science of the Total Environment, 263*(1-3), 197-208. PMID 11194153

Gochfeld, M. (2005). Community Hazardous Waste Exposures. In L. Rosenstock, M. Cullen, C. A. Brodkin & C. A. Redlich (Eds.), *Textbook of Clinical Occupational and Environmental Medicine* (2nd ed., pp. 1168-1183). Philadelphia, PA: Elsevier Saunders

Godfrey, M. E., Wojcik, D. P., & Krone, C. A. (2003, June). Apolipoprotein E genotyping as a potential biomarker for mercury neurotoxicity. *Journal of Alzheimer's Disease, 5*(3), 189-195. PMID 12897404

Gold, M., Franks, P., & Erickson, P. (1996, February). Assessing the health of the nation. The predictive validity of a preference-based measure and self-rated health. *Medical Care, 34*(2), 163-177. PMID 8632690

Goldman, L. R., Shannon, M. W., & Academy of American Pediatrics Committee on Environmental Health. (2001, July). Technical Report: mercury in the environment: implications for pediatricians. *Pediatrics, 108*(1), 197-205. PMID 11433078

Goldoni, M., Caglieri, A., Poli, D., Vettori, M., Ceccatelli, S., & Mutti, A. (2008, February). Methylmercury at low doses modulates the toxicity of PCB153 on PC12 neuronal cell line in asynchronous combination experiments. *Food and Chemical Toxicology, 46*(2), 808-811. PMID 17980472. doi:10.1016/j.fct.2007.09.104

Goldstein, B., & Gotsch, A. (2005). Communication and Assessment of Risk. In L. Rosenstock, M. Cullen, C. A. Brodkin & C. A. Redlich (Eds.), *Textbook of Clinical Occupational and Environmental Medicine* (2nd ed., pp. 1275-1283). Philadelphia, PA: Elsevier Saunders

Goldstein, D. B. (2009, April 23). Common Genetic Variation and Human Traits. *The New England Journal of Medicine, 360*(17), 1696-1698. PMID 19369660. doi:10.1056/NEJMp0806284

Gomaa, A., Hu, H., Bellinger, D., Schwartz, J., Tsaih, S.-W., Gonzalez-Cossio, T., … Hernandez-Avila, M. (2002, July). Maternal Bone Lead as an Independent Risk Factor for Fetal Neurotoxicity: A Prospective Study. *Pediatrics, 110*(1, Part 1), 110-118. PMID 12093955

Good, M. I. (1991, February 7). The Long-Term Effects of Exposure to Low Doses of Lead in Childhood. (Peer Commentary on the article "The Long-Term Effects of Exposure to Low Doses of Lead in Childhood: An 11-Year Follow-Up Report" by Needleman, Schell, Bellinger, Leviton & Allred, 1990, January 11.) *The New England Journal of Medicine, 324*(6), 415-418. PMID 1987464

Gorber, S., Scholfield-Hurwitz, S., Hardt, J., Levasseur, G., & Tremblay, M. (2009, January). The accuracy of self-reported smoking: a systematic review of the relationship between self-reported and cotinine-assessed smoking status. *Nicotine and Tobacco Research, 11*(1), 12-24. PMID 19246437. doi:10.1093/ntr/ntn010

Gordon, J. E. (1949, April). The Epidemiology of Accidents. *American Journal of Public Health, 39*(4), 504-515. PMID 18118990

Gore, K. A., Harris, R. J., & Firestone, J. M. (2004, August 14). *Differences in Male and Female Alcohol Consumption*. San Francisco, CA: American Sociological Association. Retrieved April 1, 2011 from http://www.allacademic.com//meta/p_mla_apa_research_citation/1/0/8/9/8/pages108984/p108984-1.php

Gorman, B. & Braverman, J. (2008, December). Family structure differences in health care utilization among U.S. children. *Social Science & Medicine, 67*(11), 1766-1775. PMID 18938007. doi:10.1016/j.socscimed.2008.09.034

Goyer, R. A. (1990). Transplacental Transport of Lead. *Environmental Health Perspectives, 89*(11), 101-105. PMID 2088735

Goyer, R. A. (1997). Toxic and essential metal interactions. *Annual Review of Nutrition, 17*, 37-50. PMID 9240918. doi:10.1146/annurev.nutr.17.1.37

Grady, P. A., Harden, J. T., Moritz, P., & Amende, L. M. (1997, March-April). Incorporating Environmental Sciences and Nursing Research: An NINR Initiative. *Nursing Outlook, 45,* 73-75. PMID 9364531

Graff, J., Murphy, L., Ekvall, S., & Gagnon, M. (2006, November-December). In-home toxic chemical exposures and children with intellectual and developmental disabilities. *Pediatric Nursing, 32*(6), 596-603. PMID 17256300

Graham, J., Walker, K. D., Berry, M., Bryan, E. F., Callahan, M. A., Fan, A., … Sexton, K. (1992, November-December). Role of exposure databases in risk assessment. *Archives of Environmental Health, 47*(6), 408-420. PMID 1485804

Graham, S. E., & McCurdy, T. (2004, January). Developing meaningful cohorts for human exposure models. *Journal of Exposure Analysis and Environmental Epidemiology, 14*(1), 23-43. PMID 12923556. doi:10.1038/sj.jea.7500281

Gralewicz, S., Wiaderna, D., Lutz, P., & Sitarek, K. (2009). Neurobehavioural functions in adult progeny of rat mothers exposed to methylmercury or 2,2', 4,4', 5,5'-hexachlorobiphenyl (PCB 153) alone or their combination during gestation and lactation. *International Journal of Occupational Medicine and Environmental Health, 22*(3), 277-291. PMID 19819833. doi:10.2478/v10001-009-0020-9

Grandjean, P. (1992a). Individual susceptibility to toxicity. *Toxicology Letters, 64-65*, 43-51. PMID 7618123

Grandjean, P. (1999a, November-December). Mercury Risks: Controversy or Just Uncertainty? *Public Health Reports, 114*(6), 512-515. PMID 10670618

Grandjean, P. (2008a, February). Late Insights into Early Origins of Disease. *Basic & Clinical Pharmacology & Toxicology, 102*(2), 94-99. PMID 18226061

Grandjean, P., Bellinger, D., Bergman, A., Cordier, S., Davey-Smith, G., Eskenazi, B., ... Weihe, P. (2008b, February). The Faroes Statement: Human Health Effects of Developmental Exposure to Chemicals in Our Environment. *Basic & Clinical Pharmacology & Toxicology, 102*(2), 73-75. PMID 18226057. doi:10.1111/j.1742-7843.2007.00114.x

Grandjean, P., Brown, S. S., Reavey, P., & Young, D. S. (1994, July). Biomarkers of Chemical Exposure: State of the Art. *Clinical Chemistry, 40*(7 Part 2), 1360-1362. PMID 8013119.

Grandjean, P., & Budtz-Jørgensen, E. (2007, October). Total imprecision of exposure biomarkers: implications for calculating exposure limits. *American Journal of Industrial Medicine, 50*(10), 712-719. PMID 17492658. doi:10.1002/ajim.20474

Grandjean, P., & Budtz-Jørgensen, E. (2008c, March). *Erratum*: Total imprecision of exposure biomarkers, 2007. *American Journal of Industrial Medicine, 51*(3), 229. PMID 17492658. doi:10.1002/ajim.20474

Grandjean, P., Budtz-Jørgensen, E., Jorgensen, P. J., & Weihe, P. (2005, July). Umbilical Cord Mercury Concentration as Biomarker of Prenatal Exposure to Methylmercury. *Environmental Health Perspectives, 113*(7), 905-908. PMID 16002381

Grandjean, P., Budtz-Jørgensen, E., Kieding, N., & Weihe, P. (2004a). Underestimation of Risk Due to Exposure Misclassification. *International Journal of Occupational Medicine and Environmental Health, 17*(1), 131-136. PMID 15212216

Grandjean, P., Budtz-Jørgensen, E., Steuerwald, U., Heinzow, B., Needham, L. L., Jørgensen, P. J., & Weihe, P. (2003a, April). Attenuated growth of breast-fed children exposed to increased concentrations of methylmercury and polychlorinated biphenyls. *The Federation of American Societies for Experimental Biology Journal, 17,* 699-701. PMID 12586743. doi:10.1096/fj.02-0661fje

Grandjean, P., Budtz-Jørgensen, E., White, R. F., Jørgensen, P. J., Weihe, P., Debes, F., & Keiding, N. (1999b, August 1). Methylmercury Exposure Biomarkers as Indicators of Neurotoxicity in Children Aged 7 Years. *American Journal of Epidemiology, 150*(3), 301-305. PMID 10430235

Grandjean, P., & Landrigan, P. J. (2006, December 16). Developmental neurotoxicity of industrial chemicals. *Lancet, 368,* 2167-2178. PMID 17174709. doi:10.1016/S0140-6736(06)69665-7

Grandjean, P., Murata, K., Budtz-Jørgensen, E., & Weihe, P. (2004b, February). Cardiac Autonomic Activity in Methylmercury Neurotoxicity: 14-Year Follow-Up of a Faroese Birth Cohort. *The Journal of Pediatrics, 144*(2), 169-176. PMID 14760255. doi:10.1016/j.jpeds.2003.10.058

Grandjean, P., Satoh, H., Murata, K., & Eto, K. (2010, August). Adverse Effects of Methylmercury: Environmental Health Research Implications. *Environmental Health Perspectives, 118*(8), 1137-1145. PMID 20529764. doi:10.1289/ehp.0901757

Grandjean, P., & Weihe, P. (1993, April). Neurobehavioral Effects of Intrauterine Mercury Exposure: Potential Sources of Bias. *Environmental Research, 61*(1), 176-183. PMID 8472672. doi:10.1006/enrs.1993.1062

Grandjean, P., & Weihe, P. (2003b, March). Arachidonic acid status during pregnancy is associated with polychlorinated biphenyl exposure. *American Journal of Clinical Nutrition, 77*(3), 715-719. PMID 12600866

Grandjean, P., & Weihe, P. (2008d, February). Developmental Origins of Environmentally Induced Disease and Dysfunction: International Conference on Fœtal Programming and Developmental Toxicity, 2007. *Basic & Clinical Pharmacology & Toxicology, 102*(2), 71-72. PMID 18226056

Grandjean, P., Weihe, P., Burse, V. W., Needham, L. L., Storr-Hansen, E., Heinzow, B., … White, R. F. (2001). Neurobehavioral deficits associated with PCB in 7-year-old children prenatally exposed to seafood neurotoxicants. *NeuroToxicology and Teratology, 23,* 305-317. PMID 11485834

Grandjean, P., Weihe, P., Jorgensen, P. J., Clarkson, T., Cernichiari, E., & Videro, T. (1992b, May-June). Impact of Maternal Seafood Diet on Fetal Exposure to Mercury, Selenium and Lead. *Archives of Environmental Health, 47*(3), 185-195. PMID 1596101

Grandjean, P., Weihe, P., Needham, L. L., Burse, V. W., Patterson, D. G. Jr., Sampson, E. J., … Vahter, M. (1995). Relation of a Seafood Diet to Mercury, Selenium, Arsenic and Polychlorinated Biphenyl and other Organochlorine Concentrations in Human Milk. *Environmental Research, 71,* 29-38. PMID 8757236. doi:10.1006/enrs.1995.1064

Grandjean, P., Weihe, P., White, R. F., Debes, F., Araki, S., Yokoyama, K., … Jørgensen, P. J. (1997). Cognitive Deficit in 7-Year-Old Children with Prenatal Exposure to Methylmercury. *NeuroToxicology and Teratology, 19*(6), 417-428. PMID 9392777

Grandjean, P., & White, R. F. (1999c, March 10). Effects of Methylmercury Exposure on Neurodevelopment. (Peer Commentary on article "Effects of Prenatal and Postnatal Methylmercury Exposure from Fish Consumption on Neurodevelopment: outcomes at 66 months of age in the Seychelles child development study" by Davidson, Myers, Cox, Axtell, Shamlaye, Sloan-Reeves, … Clarkson, 1998, August 26.) *Journal of the American Medical Association, 281*(10), 896-897. PMID 10078480

Grandjean, P., White, R. F., Weihe, P., & Jørgensen, P. J. (2003c, January-February). Neurotoxic Risk Caused by Stable and Variable Exposure to Methylmercury from Seafood. *Ambulatory Pediatrics, 3*(1), 18-23. PMID 12540249

Grant, N., Hamer, M., & Steptoe, A. (2009, February). Social isolation and stress-related cardiovascular, lipid and cortisol responses. *Annals of Behavioral Medicine, 37*(1), 29-37. PMID 19194770. doi:10.1007/s12160-009-9081-z

Grassman J. (1996, July 17). Obtaining information about susceptibility from the epidemiological literature. *Toxicology, 111*(1-3), 253-270. PMID 8711741. doi:10.1016/0300-483X(96)03381-1

Graubard, B. I., Sowmya Rao, R., & Gastwirth, J. L. (2005, September 15). Using the Peters-Belson method to measure health care disparities from complex survey

data. *Statistics in Medicine, 24*(17), 2659-2668. PMID 16118808. doi:10.1002/sim.2135

Gray, K. A., Klebanoff, M. A., Brock, J. W., Zhou, H., Darden, R., Needham, L., & Longnecker, M. P. (2005, July 1). *In Utero* Exposure to Background Levels of Polychlorinated Biphenyls and Cognitive Functioning among School-Age Children. *American Journal of Epidemiology, 162*(1), 17-26. PMID 15961582. doi:10.1093/aje/kwi158

Greaves, L., & Hemsing, N. (2009, August). Sex, Gender and Secondhand Smoke Policies: Implications for Disadvantaged Women. *American Journal of Preventive Medicine*, *37*(Supplement 2), S131-S137. PMID 19591752. doi:10.1016/j.amepre.2009.05.012

Green, P. M., Polk, L. V., & Slade, D. S. (2003, January-March). Environmental Nursing Diagnoses: A Proposal for Further Development of Taxonomy II. *International Journal of Nursing Terminologies and Classifications, 14*(1), 19-29. PMID 12747303

Green, P. M., & Slade, D. S. (2001, January-March). Environmental Nursing Diagnoses for Aggregates and Community. *Nursing Diagnosis, 12*(1), 5-13. AN 2001055593

Greene, T., & Ernhart, C. B. (1991). Prenatal and Preschool Age Lead Exposure: Relationship with Size. *NeuroToxicology and Teratology, 13,* 417-427. PMID 1921921

Greenland, S. (2001, September). Attributable Fractions: Bias from Broad Definition of Exposure. *Epidemiology, 12*(5), 518-520. PMID 11505170

Greenwood, M. R. (1985, June). Methylmercury Poisoning in Iraq: An Epidemiological Study of the 1971-1972 Outbreak. *Journal of Applied Toxicology, 5*(3), 148-159. PMID 4008862

Gregus, Z., & Klaassen, C. D. (2003). Mechanisms of Toxicity. In C. D. Klaassen & J. B. Watkins (Eds.), *Casarett & Doull's Essentials of Toxicology* (pp. 21-45). New York, NY: McGraw-Hill

Greizerstein, H. B., Gigliotti, P., Vena, J., Freudenheim, J., & Kostyniak, P. J. (1997, November-December). Standardization of a method for the routine analysis of polychlorinated biphenyl congeners and selected pesticides in human serum and milk. *Journal of Analytical Toxicology, 21*(7), 558-566. PMID 9399126

Greizerstein, H. B., Stinson, C., Mendola, P., Buck, G. M., Kostyniak, P. J., & Vena, J. E. (1999, April). Comparison of PCB congeners and pesticide levels between serum and milk from lactating women. *Environmental Research, 80*(3), 280-286. PMID 10092447. doi:10.1006/enrs.1999.3956

Griffin, P., Jones, K., & Cocker, J. (1997, May). Biological monitoring of polychlorinated biphenyls in plasma: a comparison of enzyme-linked immunosorbent assay and gas chromatography detection methods. *Biomarkers, 2*(3), 193-195. AN 7616057

Grimes, D. A., & Schulz, K. F. (2002, January 12). Descriptive studies: what they can and cannot do. *Lancet, 359*(9301), 145-149. PMID 11809274. doi:10.1016/S0140-6736(02)07373-7

Gross, L. (2007, February 6). Diverse Toxic Chemicals Disrupt Cell Function through a Common Path. *PLoS Biology, 5*(2), 41

Gudrais, E. (2009, March-April). The Developing Child. *Harvard Magazine, 111*(4), 34-39

Guilarte, T. R. (2009, May). Prenatal lead exposure and schizophrenia: further evidence and more neurobiological connections. (Peer Commentary on article: "Prenatal exposure to lead, delta-aminolevulinic acid, and schizophrenia: further evidence" by Opler, Buka, Groeger, McKeague, Wei, Factor-Litvak, … Susser, 2008). *Environmental Health Perspectives, 117*(5), A190-A191. PMID 19478978. doi:10.1289/ehp.0800484

Guldner, L., Monfort, C., Rouget, F., Garlantezec, R., & Cordier, S. (2007, October 24). Maternal fish and shellfish intake and pregnancy outcomes. A prospective cohort study in Britanny, France. *Environmental Health, 6*(33), PMID 17958907. doi:10.1186/1476-069X-6-33

Gulliford, M. C., Nunes, C., & Rocke, B. (2006, February 8). The 18 Household Food Security Survey items provide valid food security classifications for adults and children in the Caribbean. *BioMed Central Public Health, 6*(1), 1-26. PMID 16466571. doi:10.1186/1471-2458-6-26

Gundacker, C., Gencik, M., & Hengstchläger, M. (2010). The relevance of the individual genetic background for the toxicokinetics of two significant neurodevelopmental toxicants: mercury and lead. *Mutation Research/Reviews in Mutation Research, 705*, 130-140. PMID 20601101. doi:10.1016/j.mrrev.2010.06.003

Gundacker, C., Kormanicki, G., Zodl, B., Forster, C., Schuster, E., & Wittmann, K. (2006, December 15). Whole blood mercury and selenium concentrations in a selected Austrian population: does gender matter? *Science of the Total Environment, 372*(1), 76-86. PMID 16963109. doi:10.1016/j.scitotenv.2006.08.006

Gundacker, C., Pietschnig, B., Wittmann, K. J., Lischka, A., Salzer, H., Hohenauer, L., & Schuster, E. (2002, November). Lead and Mercury in Breast Milk. *Pediatrics, 110*(5), 873-878. PMID 12415023

Gupta, V., & Gill, K. D. (2000, January). Influence of ethanol on lead distribution and biochemical changes in rats exposed to lead. *Alcohol, 20*(1), 9-17. PMID 10680712. doi:10.1016/S0741-8329(99)00046-4

Gutknecht, W. F., Harper, S. L., Winstead, W., Sorrell, K., Binstock, D. A., Salmons, C. A., … Moore, C. (2009, January). Rapid new methods for paint collection and lead extraction. *Journal of Environmental Monitoring, 11*(1), 166-173. PMID 19137153. doi: 10.1039/b807679k

Guttormsen, B. N., Stein, J. H., McBride, P. E., Cullen, M. W., Gangnon, R., & Keevil, J. G. (2007, October 1). Rationale for Targeted Rather Than Population Based Screening with C-Reactive Protein Using the National Health and Nutrition Examination Survey (1999-2002). *American Journal of Cardiology, 100*(7), 1130-1133. PMID 17884376. doi:10.1016/j.amjcard.2007.05.037

Gwinn, M., Whipkey, D., Tennant, L., & Weston, A. (2007). Gene Expression Profiling of Di-n-Butyl Phthalate in Normal Human Mammary Epithelial

Cells. *Journal of Environmental Pathology, Toxicology and Oncology, 26*(1), 51-61. PMID 17725530

Ha, M., Lee, D., & Jacobs, D. (2007, August). Association between serum concentrations of persistent organic pollutants and self-reported cardiovascular disease prevalence: result from the National Health and Nutrition Examination Survey, 1999-2002. *Environmental Health Perspectives, 115*(8), 1204-1209. PMID 17687448. doi:10.1289/ehp.10184

Haber, L., Maier, A., & Dourson, M. (2006). *Incorporation of Mode of Action Understanding of Hormesis into Dose Response Assessment*. Cincinnati, OH: Toxicology Excellence for Risk Assessment (TERA)

Hacker, J. S. (2008, September 11). Speaking Truth to Power: the need for, and perils of health policy expertise in the White House. *The New England Journal of Medicine, 359*(11), 1085-1087. PMID 18784095. doi:10.1056/NEJMp0805482

Haddad, S., Tardif, R., Viau, C., & Krishnan, K. (1999, September 5). A modeling approach to account for toxicokinetic interactions in the calculation of biological hazard index for chemical mixtures. *Toxicology Letters, 108*(2-3), 303-308. PMID 10511275

Hagmar, L., Becher, G., Heikkilä, A., Frankman, O., Dyremark, E., Schütz, A., … Dybing, E. (1998, April 24). Consumption of fatty fish from the Baltic Sea and PCB in whole venous blood, plasma and cord blood from delivering women in the Åland/Turku Archipelago. *Journal of Toxicology and Environmental Health A, 53*(8), 581-591. PMID 9572157

Hahn, K. (2008). *The everyday life of women ages 85 and older living alone in their own residences who receive help*. (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 3347063)

Hahn, R. A. (1999, March). Why race is differentially classified on U.S. birth and infant death certficates: an examination of two hypotheses. *Epidemiology, 10*(2), 108-111. PMID 10069243

Haidet, K. (2005). *Biobehavioral responses to caregiving in very low birth weight preterm infants* (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 3261898)

Hald, A. (1952). *Statistical Theory with Engineering Applications*. New York, NY: John Wiley & Sons, Inc. (pp.144-151)

Hall, J. M., Robinson, C. H., & Broyles, T. J. (2007). Environmental Health. In M. A. Nies & M. McEwen (Eds.), *Community/Public Health Nursing: Promoting the Health of Populations* (4th ed., pp. 236-260). St. Louis, MO: Saunders Elsevier

Hall, W., Ramachndran, R., Narayan, S., Jani, A., & Vijayakumar, S. (2004, December 20). An electronic application for rapidly calculating Charlson comorbidity score. *BMC Cancer, 4*(94), 1-8. PMID 15610554. doi:10.1186/1471-2407-4-94

Halladay, A. K., Amaral, D., Aschner, M., Bolivar, V. J., Bowman, A., DiCicco-Bloom, E., … Threadgill, D. W. (2009). Animal models of autism spectrum disorders: Information for Neurotoxicologists. *NeuroToxicology, 30*, 811-821. PMID 19596370. doi:10.1016/j.neuro.2009.07.002

Ham, S. A., Yore, M. M., Kruger, J., Heath, G. W., & Moeti, R. (2007, October). Physical Activity Patterns Among Latinos in the United States: Putting the Pieces Together. *Preventing Chronic Disease, 4*(4), A92. PMID 17875267. Retrieved April 1, 2011 from http://www.cdc.gov/pcd/issues/2007/oct/06_0187.htm

Hamada, R., Arimura, K., & Osame, M. (1997). Maternal-Fetal Mercury Transport and Fetal Methylmercury Poisoning. *Metal ions in biological systems, 34,* 405-420. PMID 9046577

Hamid, A., Wani, N. A., & Kaur, J. (2009, April). New perspectives on folate transport in relation to alcoholism-induced folate malabsorption – association with epigenome stability and cancer development. *Federation of European Biochemical Societies Journal, 276*(8), 2175-2191. PMID 19292860. doi:10.1111/j.1742-4658.2009.06959.x

Han, S., Pfizenmaier, D. H., Garcia, E., Eguez, M. L., Ling, M., Kemp, F. W., & Bogden, J. D. (2000, June). Effects of Lead Exposure before Pregnancy and Dietary Calcium during Pregnancy on Fetal Development and Lead Accumulation. *Environmental Health Perspectives, 108*(6), 527-531. PMID 10856026

Hansen, H., DeRosa, C. T., Pohl, H., Fay, M., & Mumtaz, M. M. (1998, December). Public Health Challenges Posed by Chemical Mixtures. *Environmental Health Perspectives, 106*(Supplement 6), 1271-1280. PMID 9860881

Hansen, L. G. (1987). Environmental Toxicology of PCBs. In Safe, S. (Ed.) *Polychlorinated Biphenyls (PCBs): Mammalian and Environmental Toxicology* (pp. 15-48). New York, NY: Springer-Verlag

Hansen, L. G. (1998, February). Stepping Backward to Improve Assessment of PCB Congener Toxicities. *Environmental Health Perspectives, 106*(Supplement 1), 178-189

Hansen, L. G. (2001). Identification of Steady State and Episodic PCB Congeners from Multiple Pathway Exposures. In L. W. Robertson & L. G. Hansen (Eds.), *PCBs: Recent Advances in Environmental Toxicology and Health Effects*. (pp. 47-56.) Lexington, KY: The University Press of Kentucky

Hanson, D., Chu, S., Farizo, K., & Ward, J. (1995, July 24). Distribution of CD4+ T-Lymphocytes at diagnosis of Acquired Immunodeficiency Syndrome – defining and other human immunodeficiency virus-related illnesses. *Archives of Internal Medicine, 155*(14), 1537-1542. PMID 7605156

Harada, M. (1978, October). Congenital Minamata Disease: Intrauterine Methylmercury Poisoning. *Teratology, 18*(2), 285-288. PMID 362594. doi:10.1002/tera.1420180216

Harada, M. (1995). Minamata Disease: Methylmercury Poisoning in Japan Caused by Environmental Pollution. *Critical Reviews in Toxicology, 25*(1), 1-24. PMID 7734058. doi:10.3109/10408449509089885

Harada, M., & Smith, A. (1975). Minamata Disease: A Medical Report. In W. E. Smith, & A. M. Smith (Eds.), *Minamata* (pp. 180-192). New York, NY: Holt, Rinehart and Winston

Harada, M. (2004). *Minamata Disease* (English Ed.) Tokyo, Japan: Iwanami Shoten, Publishers

Hardell, E., Carlberg, M., Nordstrm, M., & van Bavel, B. (2010). Time trends of persistent organic pollutants in Sweden during 1993-2007 and relation to age, gender, body mass index, breast-feeding and parity. *Science of the Total Environment, 408*, 4412-4419. PMID 20643475. doi:http://dx.doi.org/10.1016/j.scitotenv.2010.06.029

Hardy, J., & Singleton, A. (2009, April 23). Genomewide Association Studies and Human Disease. *The New England Journal of Medicine, 360*(17), 1759-1768. PMID 19369657. doi:10.1056/NEJMra0808700

Hardy, M., & Stedeford, T. (2008, June 27). Developmental neurotoxicity in neonatal mice following co-exposure to PCB 153 and methyl mercury: interaction or false positive? *Toxicology, 248*(2-3), 160-163. PMID 18440690. doi:10.1016/j.tox.2008.03.021

Harper, M., Slaven, J. E., & Pang, T. W. (2009, February). Continue participation in an asbestos fiber-counting proficiency test with relocatable grid slides. *Journal of Environmental Monitoring, 11*(2), 434-438. PMID 19212603. doi:10.1039/b813893a

Harrad, S., Hazrati, S., & Ibarra, C. (2006, August 1). Concentrations of Polychlorinated Biphenyls in Indoor Air and Polybrominated Diphenyl Ethers in Indoor Air and Dust in Birmingham, United Kingdom: Implications for Human Exposure. *Environmental Science and Technology, 40*(15), 4633-4638. PMID 16913117

Harrell, J., Hall, S., & Taliaferro, J. (2003, February). Physiological Responses to Racism and Discrimination: An Assessment of the Evidence. *American Journal of Public Health, 93*(2), 243-248. PMID 12554577

Harremoës, P., Gee, D., MacGarvin, M., Stirling, A., Keys, J., Wynne, B., & Guedes Vaz, S. (Eds.), (2001). *Late lessons from early warnings: the precautionary principle 1896-2000*. Copenhagen, Denmark: European Environment Agency

Harville, E. W., Hertz-Picciotto, I., Schramm, M., Watt-Morse, M., Chantala, K., Osterloh, J., … Rogan, W. (2005, April). Factors influencing the difference between maternal and cord blood lead. *Occupational and Environmental Medicine, 62*(4), 263-269. PMID 15778260. doi:10.1136/oem.2003.012492

Haskell, W. L., Lee, I.-M., Pate, R. R., Powell, K. E., Blair, S. N., Franklin, B. A., … Bauman, A. (2007, August). Physical Activity and Public Health: Updated Recommendation for Adults from the American College of Sports Medicine and the American Heart Association. *Medicine and Science in Sports and Exercise, 39*(8), 1423-1434. PMID 17762377. doi:10.1249/mss.0b013e3180616b27

Hass, U. (2006). The need for developmental neurotoxicity studies in risk assessment for developmental toxicity. *Reproductive Toxicology, 22*, 148-156. PMID 16777374. doi:10.1016/j.reprotox.2006.04.009

Hassan, E. (2006). Recall bias can be a threat to retrospective and prospective research designs. *The Internet Journal of Epidemiology, 3*(2). Retrieved April 1, 2011 from http://www.ispub.com/ostia/index.php?xmlFilePath=journals/ije/vol3n2/bias.xml

Hauri, A., & Uphoff, H. (2005, September). Tasks, principles and methods of applied infectious disease epidemiology/field epidemiology. *Bundesgesundheitsblatt Gesundheitsforschung Gesundheitsshutz, 48*(9), 1013-1019. PMID 16160889. doi:10.1007/s00103-005-1119-1

Hauver, J. H., Goodman, J. A., & Grainer, M. A. (1981, June). The Federal Poverty Thresholds: Appearance and Reality. *The Journal of Consumer Research, 8*(1), 1-10.

Hawkins, N. C., Jayjock, M. A., & Lynch, J. (1982, January). A Rationale and Framework for Establishing the Quality of Human Exposure Assessments. *American Industrial Hygiene Association Journal, 53*(1), 34-41. PMID 1590219

Hayes, G., & DiMatteo, M. (2005). *Mercury, Lead and Cadmium Levels in Umbilical Cord Blood: A Pilot Study*. Providence, RI: Rhode Island Department of Health

Hays, J. C. (2005, July-August). From narrow to novice in environmental health nursing. *Public Health Nursing, 19*(4), 301-308. PMID 16150007. doi:10.1111/j.0737-1209.2005.220401.x

Hays, J. C., Schoenfeld, D., Blazer, D., & Gold, D. (1996). Global Self-Ratings of Health and Mortality: Hazard in the North Carolina Piedmont. *Journal of Clinical Epidemiology, 49*(9), 969-979. PMID 8780604

Hays, S. M., & Aylward, L. (2009, May). Using Biomonitoring Equivalents to Interpret Human Biomonitoring Data in a Public Health Risk Context. *Journal of Applied Toxicology, 29*(4), 275-288. PMID 19115313. doi:10.1002/jat.1410

He, F. (1999, September 5). Biological monitoring of exposure to pesticides: current issues. *Toxicology Letters, 108*(2-3), 277-283. PMID 10511272

Hearst, M. O., Oakes, J. M., & Johnson, P. J. (2008, December 1). The effect of racial residential segregation on black infant mortality. *American Journal of Epidemiology, 168*(11), 1247-1254. PMID 18974059. doi:10.1093/aje/kwn291

Heaven, S., Ilyushchenko, M. A., Tanton, T. W., Ullrich, S. M., & Yanin, E. P. (2000, October 9). Mercury in the River Nura and its floodplain, Central Kazakhstan: I. River sediments and water. *The Science of the Total Environment, 260*(1-3), 35-44. PMID 11032114

Heck, K., & Parker, J. (2002, February). Family structure, socioeconomic status, and access to health care for children. *Health Services Research, 37*(1), 173-186. PMID 11949919

Heindel, J. J. (2006, July). Role of exposure to environmental chemicals in the developmental basis of reproductive disease and dysfunction. *Seminars in Reproductive Medicine, 24*(3), 168-177. PMID 16804815. doi:10.1055/s-2006-944423

Heindel, J. J. (2007). Role of exposure to environmental chemicals in the developmental basis of disease and dysfunction. *Reproductive Toxicology, 23*, 257-259. PMID 17331698. doi:10.1016/j.reprotox.2007.01.006

Heindel, J. J. (2008, February). Animal models for probing the developmental basis of disease and dysfunction paradigm. *Basic and Clinical Pharmacology and Toxicology, 102*(2), 76-81. PMID 18226058

Henle, F. G. (1840). Von den Miasmen und Kontagien und von den miasmatisch-kontagiösen Krankheiten (On Miasmas and Contagions and on the Miasmatic-Contagious Diseases). *Pathologische Untersucuingen*, 1-82. in Rosen, G. (1936). Social Aspects of Jacob Henle's Medical Thought (English translation of Henle, 1840). *Bulletin of the History of Medicine*, *6*, 911-983

Henshaw, S. K. (1998, January-February). Unintended Pregnancy in the United States. *Family Planning Perspectives, 30*(1), 24-29. PMID 9494812

Herbstman, J. B., Sjödin, A., Apelberg, B. J., Witter, F. R., Patterson, D. G. Jr., Halden, R. U., … Goldman, L. R. (2007, December). Determinants of Prenatal Exposure to Polychlorinated Biphenyls and Polybrominated Diphenyl Ethers in an Urban Population. *Environmental Health Perspectives, 115*(12), 1794-1800. PMID 18087602. doi:10.1289/ehp.10333

Hernberg, S. (1980). Biochemical and Clinical Effects and Responses as Indicated by Blood Concentration. In R. L. Singhai & J. A. Thomas (Eds.), *Lead Toxicity*. (pp. 367-400.) Baltimore, MD: Urban & Schwarzenberg

Herr, C., Dostal, M., Ghosh, R., Ashwood, P., Lipsett, M., Pinkerton, K., … Hertz-Picciotto, I. (2010, August 2). Air pollution exposure during critical time periods in gestation and alterations in cord blood lymphocyte distribution: a cohort of live births. *Environmental Health, 9*(1), 46. PMID20678227. doi:10.1186/1476-069X-9-46

Herreros, M., Iñigo-Nuñez, S., Sanchex-Perez, E., Encinas, T., & Gonzalez-Bulnes, A. (2008). Contribution of fish consumption to heavy metals exposure in women of childbearing age from a Mediterranean country (Spain). *Food and Chemical Toxicology, 46*, 1591-1595. PMID 18280025. doi:10.1016/j.fct.2007.12.024

Herrick, R. F., & Dement, J. M. (2005). Principles of Industrial Hygiene. In L. Rosenstock, M. Cullen, C. A. Brodkin & C. A. Redlich (Eds.), *Textbook of Clinical Occupational and Environmental Medicine* (2nd ed., pp. 45-75). Philadelphia, PA: Elsevier Saunders

Herrick, R. F., McClean, M. D., Meeker, J. D., Baxter, L. K., & Weymouth, G. A. (2004). An Unrecognized Source of PCB Contamination in Schools and Other Buildings. *Environmental Health Perspectives, 112,* 1051-1053. PMID 15238275

Herrick, R. F., Meeker, J. D., Hauser, R., Altshul, L., & Weymouth, G. A. (2007, August 31). Serum PCB levels and Congener profiles among U.S. construction workers. *Environmental Health, 6,* 1-25. PMID 17764566. doi:10.1186/1476-069X-6-25

Hertz-Picciotto, I., Cassady, D., Lee, K., Bennett, D., Ritz, B., & Vogt, R. (2010, August 29). Study of Use of Products and Exposure-Related Behaviors (SUPERB): study design, methods, and demographic characteristics of cohorts. *Environmental Health, 9*, 54. PMID 20799988. doi:10.1186/1476-069X-9-54

Hertz-Picciotto, I., Jusko, T. A., Willman, E. J., Baker, R. J., Keller, J. A., Teplin, S. W., & Charles, M. J. (2008, July 15). A cohort study of *in utero* polychlorinated biphenyl exposures in relation to secondary sex ratio. *Environmental Health, 7,* 1-37. PMID 18627595. doi:10.1186/1476-069X-7-37

Herzstein, R. (2005). Susceptible Populations. In L. Rosenstock, M. Cullen, C. A. Brodkin & C. A. Redlich (Eds.), *Textbook of Clinical Occupational and Environmental Medicine* (2nd ed., pp. 161-181). Philadelphia, PA: Elsevier Saunders

Hightower, J. M., O'Hare, A., & Hernandez, G. T. (2006, February). Blood Mercury Reporting in NHANES: Identifying Asian, Pacific Islander, Native American and Multiracial Groups. *Environmental Health Perspectives, 114*(2), 173-175. PMID 16451850

Hill, W. (2005, Spring). The needs of rural public health workers to participate in health tracking: priority environmental health concerns for Montana public health workers. *Communicating Nursing Research, 38,* 144. AN 2009901740

Hinton, D. O. (1978). Community Air Sampling. In M. Lippmann (Ed.), *Air Sampling Instruments for evaluation of atmospheric contaminants* (5th ed., pp. C1-C6). Cincinnati OH: American Conference of Governmental Industrial Hygienists

Hirschhorn, J. N. (2009, April 23). Genomewide Association Studies: Illuminating Biologic Pathways. *The New England Journal of Medicine, 360*(17), 1699-1701. PMID 19369661. doi:10.1056/NEJMp0808934

Hisnanick, J. J., & Rogers, A. L. (2005). *Household Income Inequality Measures Based on the ACS Data: 2000-2005*. Retrieved April 1, 2011 from http://www.census.gov/hhes/www/income/publications/ACS%20inequality%20report%202000-2005_v2.pdf

Hodgson, E. (2001). Factors That Affect Pesticide Metabolism and Toxicity. In R. I. Kreiger (Ed.), *Handbook of Pesticide Toxicology* (2nd ed., Vol. I, pp. 507-513). San Diego, CA.: Academic Press

Hodgson, E., & Levy, P. (2001). Metabolism of Pesticides. In R. I. Kreiger (Ed.), *Handbook of Pesticide Toxicology Volume I: Principles* (2nd ed., pp. 531-562). San Diego, CA.: Academic Press

Hodgson, E., & Rose, R. L. (2006). Organophosphorus Chemicals: Potent Inhibitors of the Human Metabolism of Steroid Hormones and Xenobiotics. *Drug Metabolism Reviews, 38*(1-2), 149-162. PMID 16684654. doi:10.1080/03602530600569984

Hoffman, G. R. (2009). A Perspective on the Scientific, Philosophical and Policy Dimensions of Hormesis. *Dose-Response, 7*, 1-51. PMID 19343115. doi:10.2203/dose-response.08-023.Hoffmann

Höfler, M. (2005, November). The Bradford Hill considerations on causality: a counterfactual perspective. *Emerging Themes in Epidemiology, 2*(11), 1-9. PMID 16269083. doi:10.1186/1742-7622-2-11

Hogberg, H., Kinsner-Ovaskainen, A., Coecke, S., Hartung, T., & Bal-Price, A. (2010, January). mRNA expression is a relevant tool to identify developmental neurotoxicants using an in vitro approach.. *Toxicological Sciences, 113*(1), 95-115. PMID 19651682. doi:10.1093/toxsci/kfp175

Hogue, C. (2007, January 8). The Future of U.S. Chemical Regulation. *Chemical and Engineering News, 85*(2), 34-38

Hojman, D., & Fast, F. (2009, November). On the Measurement of Poverty Dynamics. *HKS Faculty Research Working Paper Series* (RWP09-035). Retrieved April 1, 2011 from http://web.hks.harvard.edu/publications/workingpapers/citation.aspx?PubId=6882

Holmberg, B., Hogberg, J., & Johanson, G. (1998). General Principles of Toxicology: Definitions and Concepts. In J. M. Stellman (Ed.), *ILO Encyclopedia of Occupational Health and Safety* (4th ed., pp. 33.3-33.7). Geneva, CH: International Labour Organisation

Holford, N. H. (1987, November). Clinical pharmacokinetics of ethanol (Abstract). *Clinical Pharmacokinetics, 13*(5), 273-292. PMID 3319346

Holmes, A. S., Blaxill, M. F., & Haley, B. E. (2003, July-August). Reduced Levels of Mercury in First Baby Haircuts of Autistic Children. *International Journal of Toxicology, 22*(4), 277-285. PMID 12933322

Hook, M. (2007). *Using the Common Sense Model to describe representations of fall risk in high-risk community dwelling older adults and to explore the relationships among representations, threat of falling, fall prevention behaviors and falling* (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 3294197)

Hooper, L., Ashton, K., Harvey, L., Decsei, T., & Fairweather-Tait, S. (2009, June). Assessing potential biomarkers of micronutrient status by using a systematic review methodology: methods. *American Journal of Clinical Nutrition, 89*(Supplement 6), S1953-S1959. PMID 19403633. doi:10.3945/ajcn.2009.27230A

Hoppin, J. A., Ryan, P. B., Hu, H., & Aro, A. C. (1996, June 12). Bone Lead Levels and Delinquent Behavior. (Peer Commentary on article "Bone lead levels in adjudicated delinquents: A case control study" by Needleman, McFarland, Ness, Fienberg & Tobin, 2002b, November-December). *Journal of the American Medical Association, 275*(22), 1727. PMID 8637163

Hornung, R., Lanphear, B., & Dietrich, K. N. (2009, August). Age of Greatest Susceptibility to Childhood Lead Exposure: A New Statistical Approach. *Environmental Health Perspectives, 117*(8), 1309-1312. PMID 19672413. doi:10.1289/ehp.0800426

Hornung, R.W., & Reed, D. L. (1990). Estimation of average concentration in the presence of nondetectable values. *Applied Occupational and Environmental Hygiene, 5*, 46-51

Horvat, M., & Hintelmann, H. (2007, May). Mercury analysis. *Analytical and Bioanalytical Chemistry, 388*(2), 315-317. PMID 17410347. doi:10.1007/s00216-007-1261-8

Hosmer, D. W., & Lemeshow, S. (2001, January). *Applied Logistic Regression*. New York, NY: John Wiley & Sons, Incorporated

Howsam, M., Grimalt, J. O., Guinó, E., Navarro, M., Marti-Ragué, J., Peinado, M. A., … Moreno, V. (2004, November). Organochlorine Exposure and Colorectal Cancer Risk. *Environmental Health Perspectives, 112*(15), 1460-1466. PMID 15531428

Hoxha, M., Dioni, L., Bonzini, M., Pesatori, A. C., Fustinoni, S., Cavallo, M., … Baccarelli, A. (2009, September 21). Association between leukocyte telomere shortening and exposure to traffic pollution: a cross-sectional study on traffic officers and indoor office workers. *Environmental Health, 8*, 1-41. PMID 19772576. doi:10.1186/1476-069X-8-41

Hrdina, P., Hanin, I., & Dubas, T. (1980). Neurochemical Correlates of Lead Toxicity. In R. L. Singhai & J. A. Thomas (Eds.), *Lead Toxicity*. (pp. 273-300). Baltimore, MD: Urban & Schwarzenberg

Hu, H. (2000, December). Exposure to Metals. *Primary Care, 27*(4), 983-996. PMID 11072295

Hu, H., Téllez-Rojo, M., Bellinger, D., Smith, D., Ettinger, A., Lamadrid-Figueroa, H., … Hernández-Avila, M. (2006, November). Fetal Lead Exposure at Each Stage of Pregnancy as a Predictor of Infant Mental Development. *Environmental Health Perspectives, 114*(11), 1730-1735. PMID 17107860. doi:10.1289/ehp.9067

Hu, Y.-C., Akland, G., Pellizzari, E., Berry, M., & Melnyk, L. (2004, December). Use of Pharmacokinetic Modeling to Design Studies for Pathway-Specific Exposure Model Evaluation. *Environmental Health Perspectives, 112*(17), 1697-1703. PMID 15579416

Huang, L.-S., Cox, C., Wilding, G. E., Myers, G. J., Davidson, P. W., Shamlaye, C. F., … Clarkson, T. W. (2003, October). Using measurement error models to assess effects of prenatal and postnatal methylmercury exposure in the Seychelles Child Development Study. *Environmental Research, 93*(2), 115-122. PMID 12963395

Huang, Y.-C., & Ghio, A. (2009, July). Controlled human exposures to ambient pollutant particles in susceptible populations. *Environmental Health, 8*(33), 1-36. PMID 19630984. doi:10.1186/1476-069X-8-33

Hughes, A. *'Can you give me respect?' Experiences of the urban poor with advanced disease* (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 3274968)

Huisman, M., Eerenstein, S. E., Koopmans-Esseboom, C., Brouwer, M., Fidler, V., Muskiet, F. A., ... Boersma, E. R. (1995a, November). Perinatal Exposure to Polychlorinated Biphenyls and Dioxins Through Dietary Intake. *Chemosphere, 31*(10), 4273-4287. PMID 8520928

Huisman, M., Koopmans-Esseboom, C., Fidler, V., Hadders-Algra, M., van der Paauw, C. G., Tuinstra, L. G., … Boersma, E. R. (1995b, April 14). Perinatal exposure to polychlorinated biphenyls and dioxins and its effect on neonatal neurological development. *Early Human Development, 41*(2), 111-127. PMID 7601016

Humbert, S., Manneh, R., Shaked, S., Wannaz, C., Horvath, A., Deschênes, L., … Margni, M. (2009, August 15). Assessing regional intake fractions in North America. *Science of the Total Environment 407*(17), 4812-4820. PMID 19535129. doi:10.1016/j.scitotenv.2009.05.024

Hunt, P. R. (2006). *Reliability and validity of an expert exposure assessment using log-linear models* (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 3221066)

Hunt, W. G., Watson, R. T., Oaks, J. L., Parish, C. N., Burnham, K. K., Tucker, R. L., … Hart, G. (2009, April 24). Lead bullet fragments in venison from rifle-killed deer: potential for human dietary exposure. *PLoS One, 4*(4), e5330. PMID 19390698. doi:10.1371/journal.pone.0005330

Hunter, D., Bomford, R. R., & Russell, D. S. (1940). Poisoning by Methyl Mercury Compounds. *The Quarterly Journal of Medicine (Br.), 9,* 193-213

Hunter, D., & Russell, D. S. (1954, November). Focal Cerebral and Cerebellar Atrophy in a Human Subject Due to Organic Mercury Compounds. *Journal of Neurology, Neurosurgery and Psychiatry, 17*(4), 235-241. PMID 13212411

Hupcey, J., & Penrod, J. (2003, Spring). Concept advancement: enhancing inductive validity. *Research and Theory in Nursing Practice: An International Journal, 17*(1), 19-30. PMID 12751883

Hursh, J. B., Clarkson, T. W., Cherian, M. G., Vostal, J. J., & Mallie, R. V. (1976, November-December). Clearance of Mercury (Hg-197, Hg-203) Vapor Inhaled by Human Subjects. *Archives of Environmental Health, 31*(6), 302-309. PMID 999343

Hussain, R. J., Gyori, J., DeCaprio, A. D., & Carpenter, D. O. (2000, September). *In Vivo* and *in Vitro* Exposure to PCB 153 Reduces Long-Term Potentiation. *Environmental Health Perspectives, 108*(9), 827-831. PMID 11017886

Hussey, L. (2002). Theories from the Biomedical Sciences. In M. McEwen & E. M. Wills (Eds.), *Theoretical Basis for Nursing* (pp. 272-296). Philadelphia, PA: Lippincott, Williams & Wilkins

Hutchinson, M., Davis, B., Jemmott, L. S., Gennaro, S., Tulman, L., Condon, E. H., … Servonsky, E. J. (2007). Promoting Research Partnerships to Reduce Health Disparities among Vulnerable Populations: sharing expertise between majority insititutions and historically black universities. *Annual Review of Nursing Research, 25*, 119-159. PMID 17958291

Hylland, K., Aspholm, O. Ø., Knutsen, J. A., & Ruus, A. (2006, March-April). Biomarkers in fish from dioxin-contaminated fjords. *Biomarkers, 11*(2), 97-117. AN 21001681

Igata, A. (1991). Epidemiological and Clinical Features of Minamata Disease. In T. Suzuki, N. Imura & T. W. Clarkson (Eds.), *Advances in Mercury Toxicology* (pp. 439-457). New York, NY: Plenum Press

Iida, T., Todaka, T., Hirakawa, H., Hori, T., Tobiishi, K., Matsueda, T., … Yamada, T. (2007, April). Concentration and distribution of dioxins and related compounds in human tissues. *Chemosphere, 67*(9), S263-S271. PMID 17215028. doi:10.1016/j.chemosphere.2006.05.107

Ikeda, M. (1988, December). Multiple Exposure to Chemicals. *Regulatory Toxicology and Pharmacology, 8*(4), 414-421. PMID 3222483

Ilacqua, V., Hanninen, O., Kuenzli, N., & Jantunen, M. (2007). Intake fraction distributions for indoor VOC sources in five European cities. *Indoor Air, 17*, 372-383. PMID 17880633. doi:10.1111/j.1600-0668.2007.00485.x

Industrial Union Department v. American Petroleum Institute, 448 U.S. 607 (1980)

Institute of Medicine. (1995). *Nursing, Health & Environment: Strengthening the Relationship to Improve the Public's Health*. Washington, DC: National Academy Press

Institute of Medicine. (2001). *Dietary Reference Intakes: Elements*. Washington, DC: National Academy Press

Institute of Medicine. (2004, January 13). *Insuring America's Health: Principles and Recommendations*. Washington, DC: National Academy Press

Institute of Medicine. (2005). *Dietary reference intakes*. Washington, DC: National Academy Press

International Council of Nurses. (1986). *The Nurse's Role in Safeguarding the Human Environment* (Position Statement). Geneva, Switzerland: International Council of Nurses

International Programme on Chemical Safety. (1983). *Guidelines on Studies in Environmental Epidemiology.* Retrieved April 1, 2011 from http://www.inchem.org/documents/ehc/ehc/ehc27.htm

International Programme on Chemical Safety. (1993). *Biomarkers and Risk Assessment: Concepts and Principles.* Retrieved April 1, 2011 from http://www.inchem.org/documents/ehc/ehc/ehc155.htm

International Programme on Chemical Safety. (2000, June 1). *Human Exposure Assessment.* Retrieved April 1, 2011 from http://www.inchem.org/documents/ehc/ehc/ehc214.htm

International Programme on Chemical Safety. (2001a). *Biomarkers in Risk Assessment: Validity and Validation.* Retrieved April 1, 2011 from http://www.inchem.org/documents/ehc/ehc/ehc222.htm

International Programme on Chemical Safety. (2001b, November 1). *Glossary of Exposure Assessment-Related Terms: A Compilation.* Retrieved April 1, 2011 from http://www.who.int/ipcs/publications/methods/harmonization/en/compilation_nov2001.pdf

International Society of Environmental Epidemiology/International Society of Exposure Analysis. (2006, November). ISEE/ISEA 2006 International Conference on Environmental Epidemiology and Exposure (September 2-6, 2006 in Paris, France) Abstracts Supplement. *Epidemiology, 17*(6), S1-S379. PMID 17315327

Irish, D. D. (1973). The significance of the occupational environment as a part of the total ecological system. In G. D. Clayton & W. D. Kelley (Eds.), *the Industrial Environment – its Evaluation & Control* (pp. 7-10). Washington, DC: U.S. Government Printing Office

JSI Center for Environmental Health Studies (2003, August 7). *Ritual Use of Mercury (Azogue) Assessment and Education Project*. Boston, MA: Massachusetts Executive Office of Environmental Affairs

Jackson, L., Zullo, M., & Goldberg, J. (2008, May). The association between heavy metals, endometriosis and uterine myomas among premenopausal women: National Health and Nutrition Examination Survey 1999-2002. *Human Reproduction, 23*(3), 679-687. PMID 18192673. doi:10.1093/humrep/dem394

Jackson, R., Rubin, C., & McGeehin, M. (2001). Sensitive Population Groups. In R.I. Kreiger (Ed.), *Handbook of Pesticide Toxicology* (2nd ed., Vol. I, pp. 783-798). San Diego, CA: Academic Press

Jackson, L., Cromer, B., & Panneerselvamm, A. (2010, November). Association between Bone Turnover, Micronutrient Intake and Blood Lead Levels in Pre- and Postmenopausal Women, NHANES 1999-2002. *Environmental Health Perspectives, 118*(11), 1590-1596. PMID 20688594. doi:10.1289/ehp.9067

Jacobs, D., Brown, M. J., Baeder, A., Sucosky, M., Margolis, S., Hershovitz, J., … Morley, R. (2010, September-October). A systematic review of housing interventions and health: introduction, methods and summary findings. *Journal of Public Health Management Practices, 16*(5 Supplement), S5-S10. PMID 20689375

Jacobs, D., Wilson, J., Dixon, S., Smith, J., & Evens, A. (2009, April). The relationship of housing and population health: a 30-year retrospective analysis. *Environmental Health Perspectives, 117*(4), 597-604. PMID 19440499. doi:10.1289/ehp.0800086

Jacobsen, M. (1976). Against Popperized Epidemiology. *International Journal of Epidemiology, 5*(1), 9-11. PMID 1262118

Jacobson, J. L., & Jacobson, S. W. (1994). The Effects of Perinatal Exposure to Polychlorinated Biphenyls and Related Contaminants. In H. L. Needleman & D. Bellinger (Eds.), *Prenatal Exposure to Toxicants: Developmental Consequences* (pp. 130-147). Baltimore, MD: The Johns Hopkins University Press

Jacobson, J. L., & Jacobson, S. W. (1996, September 12). Intellectual Impairment in Children Exposed to Polychlorinated Biphenyls *in utero*. *The New England Journal of Medicine, 335*(11), 783-789. PMID 8703183

Jacobson, J. L., & Jacobson, S. W. (2001). Developmental Effects of PCBs in the Fish Eater Cohort Studies. In L. W. Robertson & L. G. Hansen (Eds.), *PCBs: Recent Advances in Environmental Toxicology and Health Effects* (pp. 127-136). Lexington, KY: The University Press of Kentucky

Jacobson, J. L., & Jacobson, S. W. (2003, December). Prenatal exposure to polychlorinated biphenyls and attention at school age. *Journal of Pediatrics, 143*(6), 780-788. PMID 14657828. doi:10.1067/S0022-3476(03)00577-8

Jacobson, J. L., Jacobson, S. W., & Humphry, H. E. (1990, January). Effects of *in utero* exposure to polychlorinated biphenyls and related contaminants on cognitive functioning in young children. *The Journal of Pediatrics, 116*(1), 38-45. PMID 2104928

Jacobson, S. W. (1998, April). Specificity of neurobehavioral outcomes associated with prenatal alcohol exposure. *Alcohol Clinical and Experimental Research, 22*(2), 313-320. PMID 9581634

Jacobson, S. W., Chiodo, L. M., Sokol, R. J., & Jacobson, J. L. (2002, May). Validity of maternal report of prenatal alcohol, cocaine, and smoking in relation to neurobehavioral outcome. *Pediatrics, 109*(5), 815-825. PMID 11986441

Jacobziner, H. (1966, September). Lead Poisoning in Childhood: Epidemiology, Manifestations and Prevention. *Clinical Pediatrics, 5*(5), 277-286. PMID 5077325

Jaffe, M. (1886). Uber den niederschlag, welchen pikrinsaure in normalen hrn erzeugt und uber eine neue reaction des kreatinins. *Hoppe-Seyler's Zeitschrift für*

Physiologische Chemie, 10, 391-400. Retrieved April 1, 2011 from http://vlp.mpiwg-berlin.mpg.de/library/data/lit16635/index_html?pn=10&ws=1.5

Jalberet, J., Quilliam, B., & Lapane, K. (2008). A Profile of Concurrent Alcohol and Alcohol-Interactive Prescription Drug Use in the U.S. Population. *Journal of General Internal Medicine, 23*(9), 1318-1323. PMID 18575940. doi:10.1007/s11606-008-0639-4

James, T. R., Reid, H. L., & Mullings, A. M. (2008, February 28). Are published standards for haematological indices in pregnancy applicable across populations: an evaluation in healthy pregnant Jamaican women. *BioMed Central Pregnancy and Childbirth, 8*, 8. PMID 18307810. doi:10.1186/1471-2393-8-8

Jameson, R. R., Seidler, F. J., Qiao, D., & Slotkin, T. A. (2006, May). Chlorpyrifos Affects Phenotypic Outcomes in a Model of Mammalian Neurodevelopment: Critical Stages Targeting Differentiation in PC12 Cells. *Environmental Health Perspectives, 114*(5), 667-672. PMID 16675418

Jameson, R. R., Seidler, F. J., & Slotkin, T. A. (2007, January). Nonenzymatic Functions of Acetylcholinesterase Splice Variants in the Developmental Neurotoxicity of Organophosphates: Chlorpyrifos, Chlorpyrifos Oxon and Diazinon. *Environmental Health Perspectives, 115*(1), 65-70. PMID 17366821

Jameton, A. (2005). Environmental Health Ethics. In H. Frumkin (Ed.), *Environmental Health from global to local* (pp. 143 - 169). San Francisco: Jossey-Bass

Jardine, C., Hrudey, S., Shortreed, J., Craig, L., Kreweski, D., Furgal, C., & McColl, S. (2003, November-December). Risk management frameworks for human health and environmental risks. *Journal of Toxicology and Environmental Health B Critical Reviews, 6*(6), 569-720. PMID 14698953

Jaya Prasanthi, R. P., Hariprasad Reddy, G., Bhuvaneswari Devi, C., & Rajarami Reddy, G. (2005, December). Zinc and calcium reduce lead induced perturbations in the aminergic system of developing brain. *Biometals, 18*(6), 615-626. PMID 16388401. doi:10.1007/s10534-005-2993-6

Jemal, A., Graubard, B., Devesa, S., & Flegal, K. (2002, April). The association of blood lead level and cancer mortality among whites in the United States. *Environmental Health Perspectives, 110*(4), 325-329. PMID 11940448

Jenerette, C. (2004). *Testing the theory of self-care management for vulnerable populations in a sample of adults with sickle cell disease* (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 3142822)

Jeong, H., Blackmore, J., & Lewin, N. (1981). U.S. Patent No. 4,244,940. Washington, DC: U.S. Patent and Trademark Office

Jernelov, A. (1973). A New Biochemical Pathway for the Methylation of Mercury and Some Ecological Implications. In M. W. Miller & T. W. Clarkson (Eds.), *Mercury, Mercurial and Mercaptans* (pp. 315-325). Springfield, IL: Charles C. Thomas Publisher

Jiang, Y.-L., Sun, N.-H., Xiang, Y., Li, S.-L., Qi, Q.-W., Liu, J.-T., … Yang, J.-Q. (2007, July). Study on the correlation of serum folate and red blood cell folate level with birth defects and unexplained recurrent pregnancy loss. *Chinese Journal of Obstetrics and Gynecology, 42*(7), 448-452. PMID 17961332

Johansen, P., Mulvad, G., Sloth Pedersen, H., Hansen, J., & Riget, F. (2007). Human accumulation of mercury in Greenland. *Science of the Total Environment, 377*, 173-178. PMID 17368517. doi: http://dx.doi.org/10.1016/j.scitotenv.2007.02.004

Johansson, C., Castoldi, A. F., Onishchenko, N., Manzo, L., Vahter, M., & Ceccatelli, S. (2007, April). Neurobehavioural and molecular changes induced by methylmercury exposure during development. *Neurotoxicology Research, 11*(3-4), 241-260. PMID 17449462

Johansson, C., Tofighi, R., Tamm, C., Goldoni, M., Mutti, A., & Ceccatelli, S. (2006, December 15). Cell death mechanisms in AtT20 pituitary cells exposed to polychlorinated biphenyls (PCB 126 and PCB 153) and methylmercury. *Toxicology Letters, 167*(3), 183-190. PMID 17049763. doi:10.1016/j.toxlet.2006.09.006

Johnson, B. L. (2007). *Environmental Policy and Public Health*. Boca Raton, FL: CRC Press

Johnson, B. L., Harris, C. M., & Williams, R.C. (1992). *National Minority Health Conference: Focus on Environmental Contamination Proceedings, December 4-6, 1990, Atlanta, Georgia.* Atlanta, GA: Princeton Scientific Publishing Company

Johnson, D. S. (2009, Fall). Impressionistic Realism: The Europeans Focus the U.S. on Measurement. *Journal of Policy Analysis and Management, 28*(4), 725-731

Johnson, E. S. (2002). Ecological Systems and Complexity Theory: Toward an Alternative Model of Accountability in Education. *Complicity: An International Journal of Complexity and Education, 5*(1), 1-10. Retrieved April 1, 2011 from http://www.complexityandeducation.ualberta.ca/COMPLICITY5/documents/Complicity_5_1_02_Johnson.pdf

Johnson, F. O., & Atchison, W. D. (2009, September). The role of environmental mercury, lead and pesticide exposure in development of amyotrophic lateral sclerosis. *NeuroToxicology, 30*(5), 761-765. PMID 19632272. doi:10.1016/j.neuro.2009.07.010

Johnson, W., Kyvik, K. O., Mortensen, E. L., Skytthe, A., Batty, G. D., & Deary, I. J. (2010, April). Education reduces the effects of genetic susceptibilities to poor physical health. *International Journal of Epidemiology, 39*(2), 406-416. PMID 19861402. doi:10.1093/ije/dyp314

Jones, R. L., Sinks, T., Schober, S. E., & Pickett, M. (2004, November 5). Blood Mercury Levels in Young Children and Childbearing-Aged Women – United States, 1999-2002. *Morbidity and Mortality Weekly Report, 53*(43), 1018-1020. PMID 15525900

Jönsson, B. A., Rylander, L., Lindh, C., Rignell-Hydbom, A., Giwercman, A., Toft, G., … Inuendo. (2005, November 11). Inter-population variations in concentrations, determinants of and correlations between 2,2',4,4',5,5'-hexachlorobiphenyl (CB-153) and 1,1-dichloro-2,2-bis(p-chlorophenyl)-ethylene (p,p'-DDE): a cross-sectional study of 3161 men and women from

Inuit and European populations. *Environmental Health, 4,* 1-27. PMID 16283941. doi:10.1186/1476-069X-4-27

Jordan, A., & O'Riordan, T. (1999). The Precautionary Principle in Contemporary Environment Policy and Politics. In C. Raffensperger & J. Tickner (Eds.), *Protecting Public Health & the Environment: Implementing the Precautionary Principle* (pp. 15-35). Washington, DC: Island Press

Jurewicz, J., Hanke, W., Johansson, C., Lundqvist, C., Ceccatelli, S., & Van Den Hazel, P., … Zetterström, R. (2006, October). Adverse health effects of children's exposure to pesticides: What do we really know and what can be done about it? *Acta Paediatrica Supplement, 95*(453), 71-80. PMID 17000573. doi:10.1080/08035320600886489

Kacew, S., & Singhai, R. (1980). Aspects of Molecular Mechanisms Underlying the Biochemical Toxicology of Lead. In R. L. Singhai & J. A. Thomas (Eds.), *Lead Toxicity*. (pp. 43-78). Baltimore, MD: Urban & Schwarzenberg

Kaiser, R., Marcus, M. M., Blanck, H. M., Naughton, M., Zhang, R. H., Henderson, A. K., … Hertzberg, V. S. (2003, January). Polybrominated Biphenyl Exposure and Benign Breast Disease in a Cohort of U.S. Women. *Annals of Epidemiology, 13*(1), 16-23. PMID 12547481

Kales, S. N., & Goldman, R. H. (2002, February). Mercury Exposure: Current Concepts, Controversies and a Clinic's Experience. *Journal of Occupational and Environmental Medicine, 44*(2), 143-154. PMID 11851215

Kalia, M. (2008). Brain development: anatomy, connectivity, adaptive plasticity and toxicity. *Metabolism Clinical and Experimental, 57*(Supplement 2), S2-S5. PMID 18803960. doi:10.1016/j.metabol.2008.07.009

Kalamegham, R. & Ash, K. O. (2005, October 20). A simple ICP-MS procedure for the determination of total mercury in whole blood and urine. *Journal of Clinical Laboratory Analysis, 6*(4), 190-193. doi:10.1002/jcla.1860060405

Kalsbeek, W., & Heiss, G. (2000). Building bridges between populations and samples in epidemiological studies. *Annual Review in Public Health, 21*(1), 147-169. PMID 10884950. doi:10.1146/annurev.publhealth.21.1.147

Kanagawa, Y., Matsumoto, S., Koike, S., Tajima, B., Fukiwake, N., Shibata, S., … Imamura, T. (2008, October 2). Association of clinical findings in Yusho patients with serum concentrations of polychlorinate biphenyls, polycholorinated quarterphenyls and 2,3,4,7,8-pentacholorodibenzofuran more than 30 years after the poisoning event. *Environmental Health, 7*, 1-47. PMID 18831733. doi:10.1186/1476-069X-7-47

Kandula, N. R., Lauderdale, D. S., & Baker, D. W. (2007, March). Differences in self-reported health among Asians, Latinos, and non-Hispanic whites: the role of language and nativity. *Annals of Epidemiology, 17*(3), 191-198. PMID 17320786. doi:10.1016/j.annepidem.2006.10.005

Kant, A., Graubard, B., & Atchison, E. (2009, September). Intakes of plain water, moisture in foods and beverages and total water in the adult U.S. population – nutritional, meal pattern and body weight correlates: National Health and Nutrition Examination Surveys 1999-2006. *American Journal of Clinical Nutrition, 90*(3), 655-663. PMID 19640962. doi:10.3945/ajcn.2009.27749

Karmaus, W., Brooks, K. R., Nebe, T., Witten, J., Obi-Osius, N., & Kruse, H. (2005, April 14). Immune function biomarkers in children exposed to lead and organochlorine compounds: a cross-sectional study. *Environmental Health, 4*, 1-5. PMID 15831097. doi:10.1186/1476-069X-4-5

Karmaus, W., & Riebow, J. F. (2004, May). Storage of Serum in Plastic and Glass Containers May Alter the Serum Concentration of Polychlorinated Biphenyls. *Environmental Health Perspectives, 112*(6), 643-647. PMID 15121504

Karouna-Renier, N., Rao, K. R., Lanza, J., Rivers, S., Wilson, P., Hodges, D., ... Ross, G. (2008, November). Mercury levels and fish consumption practices in women of childbearing age in the Florida Panhandle. *Environmental Research, 108*(3), 320-326. PMID 18814872. doi: 10.1016/j.envres.2008.08.005

Karvetti, R., & Knuts, L. (1985, November). Validity of the 24-hour dietary recall. *Journal of American Dietetic Association, 85*(11), 1437-1442. PMID 4056262

Kasper, C. E. (2007). Genomics and Protemics Methodologies for Vulnerable Populations Research. *Annual Review of Nursing Research, 25*, 191-217. PMID 17958293

Kasperson, R. E. (2001, February). Vulnerability and Global Environmental Change. *Newsletter of the International Human Dimensions Programme on Global Environmental Change*. Bonn, Germany: International Human Dimensions Programme on Global Environmental Change

Kasperson, R. E., Kasperson, J. X., Turner, B., Dow, K. & Meyer, W. (1995). Critical environmental regions: concepts, distinctions and issues. In J. X. Kasperson, R. E. Kasperson & B. L. Turner, (Eds.), *Regions at Risk: Comparisons of Threatened Environments* (pp. 1-24). Tokyo, Japan: United Nations University

Kaufman, J. S. (1999, March). How inconsistencies in racial classification demystify the race construct in public health statistics. (Peer Commentary on the article "Why race is differentially classified on U.S. birth and infant death certficates: an examination of two hypotheses" by Hahn, 1999, March). *Epidemiology, 10*(2), 101-103. PMID 10069240

Kaushik, R., Rosenfeld, C. A., & Sultatos, L. G. (2007, June 1). Concentration-dependent interactions of the organophosphates chlorpyrifos oxon and methyl paraoxon with human recombinant acetylcholinesterase. *Toxicology and Applied Pharmacology, 221*(2), 243-250. PMID 17467020. doi:10.1016/j.taap.2007.03.013

Kavlock, R., & Perreault, S. (1994). Multiple Chemical Exposure and Risks of Adverse Reproductive Function and Outcome. In R. Yang (Ed.), *Toxicology of Chemical Mixtures*. (pp. 245-298). Burlington, MA: Academic Press

Kawahara, J., Horikoshi, R., Yamaguchi, T., Kumagai, K., & Yanagisawa, Y. (2005). Air pollution and young children's inhalation exposure to organophosphorus pesticide in an agricultural community in Japan. *Environment International, 31,* 1123-1132. PMID 15979719. doi:10.1016/j.envint.2005.04.001

Kawahara, J., Yoshinaga, J., & Yanagisawa, Y. (2007, June 1). Dietary exposure to organophosphorus pesticides for young children in Tokyo and neighboring area. *Science of the Total Environment, 378*(3), 263-268. PMID 17412399. doi:10.1016/j.scitotenv.2007.02.005

Kazantzis, G. (1988). The Use of Blood in the Biological Monitoring of Toxic Metals. In T. W. Clarkson, L. Friberg, G. F. Nordberg & P. R. Sager (Eds.), *Biological Monitoring of Toxic Metals* (pp. 547-565). New York, NY: Plenum Press

Keane, M., Stone, S., Chen, B., Slaven, J., Schwagler-Berry, D., & Antonini, J. (2009, February). Hexavalent chromium content in stainless steel welding fumes is dependent on the welding process and shield gas type. *Journal of Environmental Monitoring, 11*(2), 418-424. PMID 19212602. doi:10.1039/b814063d

Keegan, T. J., Walker, S. A., Brooks, C., Langdon, T., Linsell, L., Maconochie, N. E., … Venables, K. M. (2009, January). Exposures recorded for participants in the U.K. Chemical Warfare Agent Human Research Programme, 1941-1989. *Annals of Occupational Hygiene, 53*(1), 83-97. PMID 19131404. doi:10.1093/annhyg/men040

Keene, O. (1995). The Log Transformation is Special. *Statistics in Medicine, 14*, 811-819

Keiding, N., Budtz-Jørgensen, E., & Grandjean, P. (2003, August 23). Prenatal methylmercury exposure in the Seychelles (correspondence and replies). *The Lancet, 362*(9384), 664-665. PMID 12944071. doi:10.1016/S0140-6736(03)14166-9

Keifer, M., Wesseling, C., & McConnell, R. (2005). Pesticides and Related Compounds. In L. Rosenstock, M. Cullen, C. A. Brodkin, & C. A. Redlich (Eds.), *Textbook of Clinical Occupational and Environmental Medicine* (2nd ed., pp. 1099-1126). Philadelphia, PA: Elsevier Saunders

Kelaher, M., Paul, S., Lambert, H., Ahmad, W., & Smith, G. (2008, March 17). The impact of different measures of socioeconomic position on the relationship between ethnicity and health. *Annals of Epidemiology, 18*(5), 351-356. PMID 18346910. doi:10.1016/j.annepidem.2007.12.006

Kendall, A., Olson, C. M., & Frongillo, E. A. Jr. (1995, November). Validation of the Radimer/Cornell measures of hunger and food insecurity. *The Journal of Nutrition, 125*, 2793-2801. PMID 7472659

Khalil, N., Wilson, J. W., Talbott, E. O., Morrow, L. A., Hochberg, M. C., Hillier, T. A., … Cauley, J. A. (2009, April). Association of blood lead concentrations with mortality in older women: a prospective cohort study. *Environmental Health, 8*(15), 1-33. PMID 19344498. doi:10.1186/1476-069X-8-15

Khrisanopulo, M. (1963, August). Origin, Program and Operation of the U.S. National Health Survey. *Vital and Health Statistics, 1*(27), 1-41. PMID 14166398

Khrisanopulo, M., (1964, May). Health Survey Procedure: Concepts, Questionnaire Development, and Definitions in the Health Interview Survey. *Vital and Health Statistics Series, 1*(27), 1-66. PMID 14166399

Kieszak, S. M., Naeher, L. P., Rubin, C. S., Needham, L. L., Backer, L., Barr, D., & McGeehin, M. (2002, November). Investigation of the relation between self-reported food consumption and household chemical exposures with urinary levels of selected non-persistent pesticides. *Journal of Exposure Analysis and Environmental Epidemiology, 12*(6), 404-408. PMID 12415488. doi:10.1038/sj.jea.7500242

Kilbourne, E. M. (1994). Overview of Environmental Medicine. In L. Rosenstock & M. R. Cullen (Eds.), *Textbook of Clinical Occupational and Environmental Medicine* (pp. 41-47). Philadelphia, PA: W. B. Saunders Company

Kilpatrick, R. W. (1973, August). The Income Elasticity of the Poverty Line. *The Review of Economics and Statistics, 55*(3), 327-332

Kim, B.-M., Ha, M., Park, H.-S., Lee, B.-E., Kim, Y.-J., Hong, Y.-C., … The MOCEH Study Group. (2009, July 24). The Mothers' and Children's Environmental Health (MOCEH) Study. *European Journal of Epidemiology, 24*(9), 573-583. PMID 19629723. doi:10.1007/s10654-009-9370-7

Kim, B. S., Li, L. C., & Ng, G. F. (2005). The Asian American Values Scale – Multidimensional: Development, Reliability and Validity. *Cultural Diversity & Ethnic Minority Psychology, 11*(3), 187-201. PMID 16117587. doi:10.1037/1099-9809.11.3.187

Kim, D. Y., Staley, F., Curtis, G., & Buchanan, S. (2002, May). Relation Between Housing Age, Housing Value and Childhood Blood Lead Levels in Children in Jefferson County, KY. *American Journal of Public Health, 92*(5), 769-770. PMID 11988444

Kim, E., & Herrera, J. (2010, August 15). Characteristics of lead corrosion scales formed during drinking water distribution and their potential influence on the release of lead and other contaminants. *Environment, Science and Technology, 44*(16), 6054-6061. PMID 20704199

Kim, H. S. (2000). *The Nature of Theoretical Thinking in Nursing* (2nd ed.). New York, NY: Springer Publishing Company

Kim, J. (2008, September). Intercohort trends in the relationship between education and health: examining physical impairment and depressive symptomatology. *Journal of Aging and Health, 20*(6), 671-693. PMID 18583482. doi:10.1177/0898264308321004

Kimbrough, R. D. (1983, February). Determining Exposure and Biochemical Effects in Human Population Studies. *Environmental Health Perspectives, 48,* 77-79. PMID 6825638

Kimbrough, R. D., & Krouskas, C. A. (2003). Human Exposure to Polychlorinated Biphenyls and Health Effects: A Critical Synopsis. *Toxicological Reviews, 22*(4), 217-233. PMID 15189045

Kincl, L. D., Dietrich, K. N., & Bhattacharya, A. (2006, October). Injury Trends for Adolescents with Early Childhood Lead Exposure. *Journal of Adolescent Health, 39*(4), 604-606. PMID 16982401. doi:10.1016/j.jadohealth.2006.02.008

King, C., & Harber, P. (1998, January). Community Environmental Health Concerns and the Nursing Process. *AAOHN Journal, 46*(1), 20-27. PMID 9481216

King, D. E., Mainous, A. G., Carnemolla, M., & Everett, C. J. (2009, June). Adherence to Healthy Lifestyle Habits in U.S. Adults, 1988-2006. *The American Journal of Medicine, 122*(6), 528-534. PMID 19486715. doi:10.1016/j.amjmed.2008.11.013

King, G. (1989, February). A Seemingly Unrelated Poisson Regression Model. *Sociological Methods & Research, 17*(3), 235-255

Kinney, A. N. (1997). *Predicting body burdens of polychlorinated biphenyls by performing a comprehensive exposure assessment and pharmacokinetic modeling* (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT9809286)

Kjellström, T. (1988). Overview of Models Used in Biological Monitoring. In T. W. Clarkson, L. Friberg, G. F. Nordberg & P. R. Sager (Eds.), *Biological Monitoring of Toxic Metals* (pp. 127-145). New York, NY: Plenum Press

Kjellström, T., Kennedy, P., Wallis, S., & Mantell, C. (1986). *Physical and Mental Development of Children with Prenatal Exposure to Mercury from Fish. Stage 1: Preliminary Tests at Age 4 (Report 3080)*. Stockholm, Sweden: National Swedish Environmental Protection Board

Kjellström, T., Kennedy, P., Wallis, S., Stewart, A., Friberg, L., Lind, B., … Mantell, C. (1989). *Physical and Mental Development of Children with Prenatal Exposure to Mercury from Fish. Stage 2: Interviews and Psychological Tests at Age 6 (Report 3642)*. Stockholm, Sweden: National Swedish Environmental Protection Board

Klaassen, C. D., & Watkins, J. B. (2003). *Casarett and Doull's Essentials of Toxicology*. New York, NY: McGraw-Hill

Klein, R. J., Proctor, S. E., Boudreault, M. A., & Turczyn, K. M. (2002, July). *Healthy People 2010 Criteria for Data Suppression*. Retrieved April 1, 2011 from http://www.docstoc.com/docs/5384430/Healthy-People-2010-Criteria-for-Data-Suppression

Kleinbaum, D. G., Kupper, L. L., & Morgenstern, H. (1982). *Epidemiologic Research: Principles and Quantitative Methods*. New York, NY: John Wiley & Sons, Inc.

Klepeis, N. (1999, May). An Introduction to the Indirect Exposure Assessment Approach: Modeling Human Exposure Using Microenvironmental Measurements and the Recent National Human Activity Pattern Survey. *Environmental Health Perspectives, 107*(Supplement 2), 365-374. PMID 10350522

Klepeis, N. E., Nelson, W. C., Ott, W. R., Robinson, J. P., Tsang, A. M., Switzer, P., … Engelmann, W. H. (2001, May-June). The National Human Activity Pattern Survey (NHAPS): a resource for assessing exposure to environmental pollutants. *Journal of Exposure and Analytic Environmental Epidemiology, 11*(3), 231-352. PMID 11477521. doi:10.1038/sj.jea.7500165

Klitzman, S., Sharma, A., Nicaj, L. Vitkevich, R., & Leighton, J. (2002, June). Lead Poisoning Among Pregnant Women in New York City: Risk Factors and Screening Practices. *Journal of Urban Health, 79*(2), 225-237. PMID 12023498. doi:10.1093/jurban/79.2.225

Knaak, J., Dary, C., Power, F., Thompson, C., & Blancato, J. (2004). Physiochemical and Biological Data for the Development of Predictive Organophosphorus Pesticide QSARs and PBPK/PD Models for Human Risk Assessment. *Critical Reviews in Toxicology, 34*(2), 143-207. PMID 15112752.

Kneipp, S. M., & Yarandi, H. N. (2002, August). Complex Sampling Designs and Statistical Issues in Secondary Analysis. *Western Journal of Nursing Research, 24*(5), 552-566. PMID 12148835

Knobeloch, L., Gliori, G., & Anderson, H. (2007). Assessment of methylmercury exposure in Wisconsin. *Environmental Research, 103*, 205-210. PMID 16831413. doi:10.1016/j.envres.2006.05.012

Knol, A., Petersen, A., van der Sluijs, J., & Lebret, E. (2009, April). Dealing with uncertainties in environmental burden of disease assessment. *Environmental Health, 8*(21), 1-34. PMID 19400963. doi:10.1186/1476-069X-8-21

Kobal, A., Prezelj, M., Horvat, M., Krsnik, M., Gibičar, D., & Osredkar, J. (2008). Glutathione level after long-term occupational elemental mercury exposure. *Environmental Research, 107*, 115-123. PMID 17706633. doi:10.1016/j.envres.2007.07.001

Koch, G. G., Gillings, D. B., & Stokes, M. E. (1980). Biostatistical Implications of Design, Sampling and Measurement to Health Science Data Analysis. *Annual Review in Public Health, 1*, 163-225. PMID 6753862. doi:10.1146/annurev.pu.01.050180.001115

Koch, H. M., Hardt, J., & Angerer, J. (2001, November). Biological monitoring of exposure of the general population to the organophosphorus pesticides chlorpyrifos and chlorpyrifos-methyl by determination of their specific metabolite 3,5,6-trichloro-2-pyridinol. *International Journal of Hygiene and Environmental Health, 204*(2-3), 175-180. PMID 11759161

Kogevinas, M. (1998, October). The loss of the population approach puts epidemiology at risk. (Peer Commentary on the article "Does risk factor epidemiology put epidemiology at risk? Peering into the future" by Susser, 1998, October). *Journal of Epidemiology and Community Health, 52*(10), 615-616. PMID 10023454

Kohlhuber, M., Heinrich, J., Van Den Hazel, P., Zuurbier, M., Bistrup, M. L., Koppe, J. G., & Bolte, G. (2006, October). Children's environmental health: Why should social disparities be considered? *Acta Paediatrica Supplement, 95*(453), 26-30. PMID 17000566. doi:10.1080/08035250600885910

Kolb, S., Bruckner, U., Nowak, D., & Radon, K. (2010, August 12). Quantification of ETS exposure in hospitality workers who have never smoked. *Environmental Health, 9*, 1-49. doi:10.1186/1476-069X-9-49

Kominski, R., & Siegel, P. (1993, September). Measuring Education in the Current Population Survey. *Monthly Labor Review*, *116*(9), 34-38

Kondo, K. (2000, July). Congenital Minamata Disease: Warnings from Japan's Experience. *Journal of Child Neurology, 15*(7), 458-464. PMID 10921517

Kondrup, J., Rasmussen, H. H., Hamberg, O., Stanga, Z. & Ad Hoc ESPEN Working Group. (2003, June). Nutritional risk screening (NRS 2002): a new method based on an analysis of controlled clinical trials. *Clinical Nutrition, 22*(3), 321-336. PMID 12765673

Koopmans-Esseboom, C., Huisman, M., Weisglas-Kuperus, N., Boersma, E. R., de Ridder, M. A., Van der Paauw, C. G., ... Sauer, P. J. (1994, November-December). Dioxin and PCB levels in blood and human milk in relation to living areas in The Netherlands. *Chemosphere, 29*(9/11), 2327-1238. PMID 7850381

Koopmans-Esseboom, C., Weisglas-Kuperus, N., DeRidder, M. A., Van der Paaw, C. G., Tuinstra, L. G., & Sauer, P. J. (1996, May). Effects of Polychlorinated Biphenyl/Dioxin Exposure and Feeding Type on Infants' Mental and Psychomotor Development. *Pediatrics, 97*(5), 700-706. PMID 8628610

Koppe, J. G., Bartonova, A., Bolte, G., Bistrup, M. L., Busby, C., Butter, M., … Zuurbier, M. (2006, October). Exposure to multiple environmental agents and their effect. *Acta Paediatrica Supplement, 95*(453), 106-113. PMID 17000577. doi:10.1080/08035320600886646

Korrick, S. A., Altshul, L. M., Tolbert, P. E., Burse, V. W., Needham, L. L. & Monson, R. R. (2000, November-December). Measurement of PCBs, DDE and hexachlorobenzene in cord blood from infants born in towns adjacent to a PCB-contaminated waste site. *Journal of Exposure Analysis and Environmental Epidemiology, 10*(6, Part 2), 743-754. PMID 11138666

Korrick, S., & Sagiv, S. (2008, April). Polychlorinated biphenyls, organochlorine pesticides and neurodevelopment. *Current Opinion in Pediatrics, 20*(2), 198-204. PMID 18332718

Kostyniak, P. J., Hansen, L. G., Widholm, J. J., Fitzpatrick, R. D., Olson, J. R., Helferich, J. L., … Schantz, S. L. (2005, December). Formulation and Characterization of an Experimental PCB Mixture Designed to Mimic Human Exposure from Contaminated Fish. *Toxicological Sciences, 88*(2), 400-411. PMID 16177234. doi:10.1093/toxsci/kfi338

Kostyniak, P. J., Stinson, C., Greizerstein, H. B., Vena, J., Buck, G., & Mendola, P. (1999, February). Relation of Lake Ontario fish consumption, lifetime lactation and parity to breast milk polychlorobiphenyl and pesticide concentrations. *Environmental Research, 80*(2 Part 2), S166-S174. PMID 10092430. doi:10.1006/enrs.1998.3939

Kramer, M. S., & Kakuma, R. (2002). *The Optimal Duration of Exclusive Breastfeeding: A Systematic Review*. Geneva, Switzerland: World Health Organization Department of Nutrition for Health and Development

Krampl, V., Kontskova, M., & Kramplova, J. (1980, November). Influence of ethanol on the interaction between polychlorinated biphenyls and drug metabolism. *Bulletin of Environmental Contamination and Toxicology, 25*(5), 718-725. PMID 6781558. doi:10.1007/BF01608097

Krause, N. M., & Jay, G. M. (1994, September). What Do Global Self-Rated Health Items Measure? *Medical Care, 32*(9), 930-942. PMID 8090045

Kravets, N., & Parker, J. (2008, November). Linkage of the Third National Health and Nutrition Examination Survey to air quality data. *Vital and Health Statistics, 2*(149), 1-16. PMID 19280992

Krieger, J., & Higgins, D. (2002, May). Housing and health: time again for public health action. *American Journal of Public Health, 92*(5), 758-768. PMID 11988443

Krieger, N. (1994, October). Epidemiology and the web of causation: has anyone seen the spider? *Social Science & Medicine, 39*(7): 887-903. PMID 7992123. doi:10.1016/0277-9536(94)90202-X

Krieger, N. (2001). Theories for social epidemiology in the 21st century: an ecosocial perspective. *International Journal of Epidemiology, 30,* 668-677. PMID 11511581

Kreiger, N. (2003, February). Does Racism Harm Health? Did Child Abuse Exist Before 1962? On Explicit Questions, Critical Science and Current Controversies: An Ecosocial Perspective. *American Journal of Public Health, 93*(2), 194-199. PMID 12554569

Krieger, R. I., Bernard, C. E., Dinoff, T. M., Ross, J. H., & Williams, R. L. (2001). Biomonitoring of Persons Exposed to Insecticides Used in Residences. *Annals of Occupational Hygiene, 45*(1001), S143-S153. PMID 11290360

Kronenberg, F., Trenkwalder, E., Kronenberg, M. F., Konig, P., Utermann, G., & Dieplinger, H. (1998, October). Influence of hematocrit on the measurement of lipoproteins demonstrated by the example of lipoprotein(a). *Kidney International, 54*(4), 1385-1389. PMID 9767560. doi:10.1046/j.1523-1755.1998.00086.x

Krystek, P., & Ritsema, R. (2005, January). Mercury speciation in thawed out and refrozen fish samples by gas chromatography coupled to inductively coupled plasma mass spectrometry and atomic fluorescence spectroscopy. *Analytical & Bioanalytical Chemistry, 381*(2), 354-359. PMID 15292976. doi:10.1007/s00216-004-2740-9

Kubale, T. L. (2004). *A nested case-control study of leukemia and ionizing radiation at the Portsmouth Naval Shipyard* (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 3144513)

Kurland, L. T., Faro, S. N., & Siedler, H. (1960, November). Minamata Disease. *World Neurology, 1,* 370-395. PMID 13755288

Kutlu, T., Karagozler, A. A., & Gozurkara, E. M. (2006, Winter). Relationship among placental cadmium, lead, zinc and copper levels in smoking pregnant women. *Biological Trace Element Research, 114*(1-3), 7-17. PMID 17205983. doi:10.1385/BTER:114:1:7

Kwong, W., Friello, P., & Semba, R. (2004). Interactions between iron deficiency and lead poisoning: epidemiology and pathogenesis. *Science of the Total Environment, 330*, 21-37. PMID 15325155. doi:10.1016/j.scitotenv.2004.03.017

Laaksonen, M., Tarkiainen, L., & Martikainen, P. (2009, September). Housing wealth and mortality: A register linkage study of the Finnish population. *Social Science & Medicine, 69*(5), 754-760. PMID 19604611. doi:10.1016/j.socscimed.2009.06.035

Laden, F., Collman, G., Iwamoto, K., Alberg, A., Berkowitz, G., Freudenheim, J., … Hunter, D. (2001, May 16). 1,1-Dichloro-2,2-bis(*p*-chlorophenyl)ethylene and Polychlorinated Biphenyls and Breast Cancer: Combined Analysis of Five U.S. Studies. *Journal of the National Cancer Institute, 93*(10), 768-776. PMID 11353787

Laden, F., Neas, L., Spiegelman, D., Hankinson, S., Willett, W., Ireland, K., … Hunter, D. (1999, January). Predictors of plasma concentrations of DDE and PCBs in a group of U.S. women. *Environmental Health Perspectives, 107*(1), 75-81. PMID 9872720

Laidlaw, M. A., Mielke, H. W., Filippelli, G. M., Johnson, D. L., & Gonzales, C. R. (2005, June). Seasonality and Children's Blood Lead Levels: Developing a Predictive Model Using Climatic Variables and Blood Lead Data from Indianapolis, Indiana Syracuse, New York, and New Orleans, Lousiana. *Environmental Health Perspectives, 113*(6), 793-800. PMID 15929906

Lakind, J. S., Holgate, S. T., Ownby, D. R., Mansur, A. H., Helms, P. J., Pyatt, D., & Hays, M. (2007, September-October). A critical review of the use of Clara cell secretory protein (CC16) as a biomarker of acute or chronic pulmonary effects. *Biomarkers, 12*(5), 445-467. AN 26287809

Laks, D. R. (2009, August 21). Assessment of chronic mercury exposure within the U.S. population, National Health and Nutrition Examination Survey, 1999-2006. *Biometals.* PMID 19697139. doi:10.1007/s10534-009-9261-0

Lamb, M. R., Taylor, S., Liu, X., Wolff, M. S., Borrell, L., Matte, T. D., … Factor-Litvak, P. (2006, May). Prenatal Exposure to Polychlorinated Biphenyls and Postnatal Growth: A Structural Analysis. *Environmental Health Perspectives, 114*(5), 779-785. PMID 16675437

Lambert, J. C., & Lipscomb, J. C. (2007, December). Mode of action as a determining factor in additivity models for chemical mixture risk assessment. *Regulatory Toxicology and Pharmacology, 49*(3), 183-194. PMID 17804132. doi:10.1016/j.yrtph.2007.07.002

LaMontagne, A., Herrick, R., Van Dyke, M., Martyny, J., & Ruttenber, A. J. (2002, March-April). Exposure Databases and Exposure Surveillance: Promise and Practice. *American Industrial Hygiene Association Journal, 63*(2), 205-212. PMID 11975658

Lancisi, G. M. (1717). *De noxiis paludum effluviis eorumpque remediis, traduzione e commento a cura della dssa.* (Of the poisonous effleuvia …). Retrieved April 1, 2011 from http://lccn.loc.gov/46040766

Landrigan, P. J. (1990, November). Current Issues in the Epidemiology and Toxicology of Occupational Exposure to Lead. *Environmental Health Perspectives, 89,* 61-66. PMID 2088757

Landrigan, P. J., Schechter, C. B., Lipton, J. M., Fahs, M. C., & Schwartz, J. (2002). Environmental Pollutants and Disease in American Children: Estimates of Morbidity, Mortality, and Costs for Lead Poisoning, Asthma, Cancer and Developmental Disabilities. *Environmental Health Perspectives, 110,* 721-728. PMID 12117650

Landrigan, P. J., Nordberg, M., Lucchini, R., Nordberg, G., Grandjean, P., Iregren, A., & Alessio, L. (2007, October). The Declaration of Brescia on prevention of the neurotoxicity of metals June 18, 2006. *American Journal of Industrial Medicine, 50*(10), 709-711. PMID 17036364. doi:10.1002/ajim.20404

Langworth, S., Elinder, C., Gothe, C., & Vesterberg, O. (1991). Biological monitoring of environmental and occupational exposure to mercury. *International Archives of Occupational and Environmental Health, 63*(3), 161-167. PMID 1917065

Lanphear, B. P., Burgoon, D., Rust, S., Eberly, S., & Galke, W. (1998, February). Environmental exposures to lead and urban children's blood lead levels. *Environmental Research, 76*(2), 120-130. PMID 9515067. doi:10.1006/enrs.1997.3801

Lanphear, B. P., Dietrich, K. N., Auinger, P., & Cox, C. (2000, November-December). Cognitive Deficits Associated with Blood Lead Concentrations <10 µg/dl in U.S. Children and Adolescents. *Public Health Reports, 115,* 521-529. PMID 11354334

Lanphear, B. P., Hornung, R., Khoury, J., Yolton, K., Baghurst, P., Bellinger, D., … Roberts, R. (2005, July). Low-Level Environmental Lead Exposure and Children's Intellectual Function: An International Pooled Analysis. *Environmental Health Perspectives, 113*(7), 894-899. PMID 16002379

Laroche, M., Pons, F., & Richard, M.-O. (2009). The Role of Language in Ethnic Identity Measurement: A Multi-Trait Multimethod Approach to Construct Validation. *The Journal of Social Psychology, 149*(4), 513-539

Larson, K. (2006). *An ethnographic study of sexual risk among latino adolescents in rural North Carolina*. (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 3212468)

Larson, K., & Halfon, N. (2010, May). Family Income Gradients in the Health and Healthcare Access of U.S. Children. *Maternal and Child Health Journal*, *14*(3), 332-342. PMID 19499315. doi:10.1007/s10995-009-0477-y

Larsson, L. S., & Butterfield, P. (2002, July-August). Mapping the Future of Environmental Health and Nursing: Strategies for Integrating National Competencies into Nursing Practice. *Public Health Nursing, 19*(4), 301-308. PMID 12071904

Larsson, L., Butterfield, P., Christopher, S., & Hill, W. (2004, Spring). An environmental risk reduction study with rural families: community leaders' perceptions of environmental health risks. *Communicating Nursing Research, 37,* 133. PMID 15382321

Last, J. M. (1983). *A Dictionary of Epidemiology* (4th ed.). New York, NY: Oxford University Press

Laumbach, R., Tong, J., Zhang, L., Ohman-Strickland, P., Stern, A., Fiedler, N., … Zhang, J. (2009, January). Quantification of 1-aminopyrene in human urine after a controlled exposure to diesel exhaust. *Journal of Environmental Monitoring, 11*(1), 153-159. PMID 19137151. doi:10.1039/b810039j

Lavoué, J., & Droz, P. O. (2009, March). Multi-Model Inference and Multi-Model Averaging in Empirical Modeling of Occupational Exposure Levels. *Annals of Occupational Hygiene, 53*(2), 173-180. PMID 19174483. doi:10.1093/annhyg/men085

Lauwerys, R. R., Buchet, J. P., Roels, H., & Hubermont, G. (1978, April 15). Placental Transfer of Lead, Mercury, Cadmium and Carbon Monoxide in Women. *Environmental Research, 15*(2), 278-289. PMID 668658

Lauwerys, R. R., & Hoet, P. (1993). *Industrial Chemical Exposure: Guidelines for Biological Monitoring* (2nd ed.). Ann Arbor, MI: Lewis Publishers

Lawrence, P. S. (1959, March). Collection of Data on Accidental Injuries. *Public Health Reports, 74*(3), 195-198. PMID 13634310

Lawson, C. C., Grajewski, B., Daston, G. P., Frazier, L. M., Lynch, D., McDiarmid, M., … Whelan, E. A. (2006, March). Workgroup Report: Implementing a National Occupational Reproductive Research Agenda – Decade One and Beyond. *Environmental Health Perspectives, 114*(3), 435-441. PMID 16507468

Leavell, H. R., & Clark, E. G. (1958). *Preventive medicine for the doctor and his community*. New York, NY: McGraw-Hill

LeBlanc, G. A., & Olmstead, A. W. (2004, September). Evaluating the Toxicity of Chemical Mixtures. *Environmental Health Perspectives, 112*(13), A729-A730. PMID 15345361

LeBlanc, G. A., & Wang, G. (2006, September). Chemical Mixtures: Greater-than-Additive Effects? (Correspondence and Author's Reply). *Environmental Health Perspectives, 114*(9), A517-A518. PMID 16966072

Lee, C. (1992). Toxic Waste and Race in the United States. In B. Bryant & P. Mohai (Eds.), *Race and the Incidence of Environmental Hazards* (pp. 10-27). Boulder, CO: Westview Press

Lee, C. (2005). Environmental Justice. In H. Frumkin (Ed.), *Environmental Health: From Global to Local* (pp. 170-196). San Francisco, CA: Jossey-Bass

Lee, D., Lim, J., Song, K., Boo, Y., & Jacobs, D. (2006, March). Graded Associations of Blood Lead and Urinary Cadmium Concentrations with Oxidative-Stress-Related Markers in the U.S. Population: Results from the Third National Health and Nutrition Examination Survey. *Environmental Health Perspectives, 114*(3), 350-354. PMID 16507456

Lee, D.-H., Jacobs, D. R., & Porta, M. (2007). Association of serum concentrations of persistent organic pollutants with the prevalence of learning disability and attention deficit disorder. *Journal of Epidemiology and Community Health, 61*, 591-596. doi: 10.1136/jech.2006.054700

Lee, E. (2004). *An investigation of physical factors for estimating exposure to airborne contaminants* (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 3142826)

Lee, K., Hahn, E. J., Riker, C. A., Hoehne, A., White, A., Greenwell, D., & Thompson, D. (2007, August). Secondhand Smoke Exposure in a Rural High School. *Journal of School Nursing, 23*(4), 222-228. PMID 17676970. doi:10.1622/1059-8405(2007)23[222:SSEIAR]2.0.CO;2

Lee, M., Chun, O., & Song, W. (2005, February). Determinants of the blood lead level of U.S. women of reproductive age. *Journal of the American College of Nutrition, 24*(1), 1-9. PMID 15670978

Lee, S., Nguyen, H. A., & Tsui, J. (2011, April). Interview Language: A Proxy Measure for Acculturation Among Asian Americans in a Population-Based Survey. *Journal of Immigrant and Minority Health, 13*(2), 244-252. PMID 19639411. doi:10.1007/s10903-009-9278-z

Lee, Y. (2000). The predictive value of self-assessed general, physical and mental health on functional decline and mortality in older adults. *Journal of Epidemiology and Community Health, 54*, 123-129. PMID 10715745

Leech, J. A., Nelson, W. C., Burnett, R. T., Aaron, S., & Raizenne, M. E. (2002). It's about time: a comparison of Canadian and American time-activity patterns. *Journal of Exposure Analysis and Environmental Epidemiology, 12*, 427-432. PMID 12415491. doi:10.1038/sj.jea.7500244

Leeman, R., Heilig, M., Cunningham, C., Stephens, D., Duka, T., & O'Malley, S. (2010). Ethanol consumption: how should we measure it? Achieving consilience between human and animal phenotypes. *Addiction Biology, 15*, 109-124

Leffers, J. M., Martins, D. C., McGrath, M. M., Brown, D. G., Mercer, J., Sullivan, M. C., & Viau, P. (2004). Development of a Theoretical Construct for Risk and Vulnerability from Six Empirical Studies. *Research and Theory for Nursing Practice: An International Journal, 18*(1), 16-34. PMID 15083660

Lemus, R., Abdelghani, A., Akers, T., & Horner, W. (1996, October-December). Health risks from exposure to metals in household dusts. *Reviews in Environmental Health, 11*(4), 179-189. PMID 9085434

Lentfer, J. W., & Galster, W. A. (1987). Mercury in Polar Bears from Alaska. *Journal of Wildlife Diseases, 23*(2), 338-341. PMID 3586215

Leslie, L., & Swider, S. (1986, March). Changing factors and changing needs in women's healthcare. *Nursing Clinics of North America, 21*(1), 111-123. PMID 3513129

Lessick, M. (1986). *Development and operationalization of a vulnerability model for nursing practice based on selected behavioral genetic concepts*. (Doctoral dissertation). Retrieved from ProQuest Dissertations and Theses database. (AAT 8700231)

Levine, T. E., & Butcher, R. E. (1990, May-June). Workshop on the Qualitative and Quantitative Comparability of Human and Animal Developmental Neurotoxicology, Work Group IV Report: Triggers for Developmental Neurotoxicity Testing. *NeuroToxicology and Teratology, 12*(3), 281-284. PMID 2196426

Leviton, A., Bellinger, D., Allred, E. N., Rabinowitz, M., Needleman, H., & Shoenbaum, S. (1993, January). Pre- and Post-Natal Low-Level Lead Exposure and Children's Dysfunction in School. *Environmental Research, 60*(1), 30-43. PMID 7674348

Lewis, R. A. (1998). *Dictionary of Toxicology*. Boca Raton, FL: Lewis Publishers

Li, K.-M. (1988). Lead Values in Umbilical Cord Blood and Maternal Blood. *The Journal of the Royal Society for the Promotion of Health, 108*, 59. PMID 3131524. doi:10.1177/146642408810800210

Li, Q., Vena, J. E., & Swanson, M. K. (2005, February). Reliability of sport fish consumption in the New York State Angler cohort study. *Environmental Research, 97*(2), 142-148. PMID 15533330. doi:10.1016/j.envres.2004.01.010

Li, Z.-Z., Dong, T., Pröschel, C., & Noble, M. (2007, February). Chemically diverse toxicants converge on Fyn and c-Cbi to disrupt precursor cell function. *PLoS Biology, 5*(2), 35. PMID 17298174. doi:10.1371/journal.pbio.0050035

Lichtenwalter, S. (2005, June). Gender poverty disparity in U.S. cities: evidence exonerating female-headed families. *Journal of Sociology and Social Welfare, 32*(2), 75-96

Liden, G., & Surakka, J. (2009, March). A Headset-Mounted Mini-Sampler for Measuring Exposure to Welding Aerosol in the Breathing Zone. *Annals of Occupational Hygiene, 53*(2), 99-116. PMID 19196747. doi:10.1093/annhyg/mep001

Lieu, T. A., Newacheck, P. W., & McManus, M. A. (1993, July). Race, Ethnicity and Access to Ambulatory Care among U.S. Adolescents. *American Journal of Public Health, 83*(7), 960-965. PMID 8328617

Lilienfeld, D. E. (2007, July 1). The General Epidemiologist: Is There a Place in Today's Epidemiology? *American Journal of Epidemiology, 166*(1), 1-4. PMID 17496312. doi:10.1093/aje/kwm160

Liljelind, I. E., Hagenbjork-Gustafsson, A., & Nilsson, L. O. (2009, January). Potential dermal exposure to methyl methacrylate among dental technicians; variability and determinants in a field study. *Journal of Environmental Monitoring, 11*(1), 160-165. PMID 19137152. doi:10.1039/b810355k

Lim, K.-M., Kim, S., Noh, J.-Y., Kim, K., Jang, W.-H., Bae, O.-N., … Chung, J.-H. (2010, July). Low-Level Mercury Can Enhance Procoagulant Activity of Erythrocytes: A New Contributing Factor of Mercury-Related Thrombotic Disease. *Environmental Health Perspectives, 118*(7), 928-935. doi:10.1289/ehp.0901473

Lindberg, S., Bullock, R., Ebinghaus, R., Engstrom, D., Feng, X., Fitzgerald, W., … Panel on Source Attribution of Atmospheric Mercury. (2007, February). A

Synthesis of Progress and Uncertainties in Attributing the Sources of Mercury in Deposition. *Ambio, 36*(1), 19-32. PMID 17408188

Linder, F. E. (1958, May 30). National Health Survey. *Science, 127*(3309), 1275-1280. PMID 13555876

Linder, F. E., (1969, March). Methods for Measuring Population Change: A Systems Analysis Summary. *Vital and Health Statistics Series, 2*(32), 1-24. PMID 5313121

Lindow, S. W., Knight, R., Batty, J., & Haswell, S. J. (2003, March). Maternal and neonatal hair mercury concentrations: the effect of dental amalgam. *BJOG: an International Journal of Obstetrics and Gynaecology, 110*(3), 287-291. PMID 12628269

Links, B., & Phelan, J. (1995). Social Conditions as Fundamental Causes of Disease. *Journal of Health and Social Behavior, 35*(Extra Issue), 80-94. PMID 7560851

Links, J. M., Kensler, T. W., & Groopman, J. D. (1995). Biomarkers and Mechanistic Approaches in Environmental Epidemiology. *Annual Review of Public Health, 16*, 83-103. PMID 7639885. doi:10.1146/annurev.pu.16.050195.000503

Links, J. M., Schwartz, B. S., Simon, D., Bandeen-Roche, K., & Stewart, W. F. (2001, April). Characterization of Toxicokinetics and Toxicodynamics with Linear Systems Theory: Application to Lead-Associated Cognitive Decline. *Environmental Health Perspectives, 109*(4), 361-368. PMID 11335184

Lioy, P. J. (1990). Assessing total human exposure to contaminants. *Environmental Science & Technology, 24*(7), 938-945

Lioy, P. J., Freeman, N. C., & Millette, J. R. (2002, October). Dust: A Metric for Use in Residential and Building Exposure Assessment and Source Characterization. *Environmental Health Perspectives, 110*(10), 969-983. PMID 12361921

Lioy, P., Leaderer, B., Graham, J., Lebret, E., Sheldon, L., & Lebowitz, M. (2005a). The Major Themes from the Plenary Panel Session of the International Society of Exposure Analysis – 2004 Annual Meeting on: The Application of Exposure

Assessment to Environmental Health Science and Public Policy – what has been accomplished and what needs to happen before our 25th anniversary in 2014. *Journal of Exposure Analysis and Environmental Epidemiology, 15,* 121-122. PMID 15759011. doi:10.1038/sj.jea.7500417

Lioy, P., Lebret, E., Spengler, J., Brauer, M., Buckley, T., Freeman, N., … Zmirou-Navier, D. (2005b). Defining Exposure Science. *Journal of Exposure Analysis and Environmental Epidemiology, 15,* 463. PMID 16294192. doi:10.1038/sj.jea.7500463

Lioy, P. J., & Pellizzari, E. (1995). Conceptual Framework for Designing a National Survey of Human Exposure. *Journal of Exposure Analysis and Environmental Epidemiology, 5*(3), 425-444. PMID 8814779

Lipschitz, D., Skikne, B., & Thompson, C. (1981, May). Precision and accuracy of serum ferritin measurements. *American Journal of Clinical Nutrition, 34*(5), 951-956. PMID 7234722

Lipscomb, J. (1994, July). Environmental Health: Assuming a Leadership Role. *AAOHN Journal, 42*(7), 314-315. PMID 8060394

Lipscomb, J., & Sattler, B. (2001). Environmental Health. In M. K. Salazar (Ed.), *Core Curriculum for Occupational & Environmental Health Nursing* (2nd ed., pp. 393-410). Philadelphia, PA: W. B. Saunders Company

Lipsitz, L. A., & Goldberger, A. L. (1992, April 1). Loss of "complexity" and aging: potential applications of fractals and chaos theory to senescence. *Journal of the American Medical Association, 267*(13), 1806-1809. PMID 1482430

Liu, G., & Elsner, J. (1995, July). Review of the multiple chemical exposure factors which may disturb human behavioral development. *Sozial- und Präventivmedizin, 40*(4), 209-217. PMID 8525710

Lockitch, G. (1997, February). Clinical biochemistry of pregnancy. *Critical Review in Clinical Laboratory Sciences, 34*(1), 67-139. PMID 9055057. doi:10.3109/10408369709038216

Logroscino, G. (2005, September). The Role of Early Life Environmental Risk Factors in Parkinson Disease: What Is the Evidence? *Environmental Health Perspectives, 113*(9), 1234-1238. PMID 16140634

Logue, J. N., White, M. V., & Marchetto, D. J. (2007, October). Pennsylvania's asthma school project and descriptive pilot investigation: a focus on environmental health tracking. *Journal of Environmental Health, 70*(3), 21-27. PMID 17941399

Long, G. L., & Winefordner, J. D. (1983, June). Limit of detection: a closer look at the IUPAC definition. *Analytical Chemistry, 55*(7), 712A-724A. doi:10.1021/ac00258a001

Long, M., Deutch, B., & Bonefeld-Jørgensen, E. C. (2007, October 23). AnR transcriptional activity in serum of Inuit's across Greenlandic districts. *Environmental Health, 6,* 1-32. PMID 17956617. doi:10.1186/1476-069X-6-32

Longnecker, M. P. (2001). Endocrine and Other Human Health Effects of Environmental and Dietary Exposure to Polychlorinated Biphenyls. In L. W. Robertson & L. G. Hansen (Eds.), *PCBs: Recent Advances in Environmental Toxicology and Health Effects* (pp. 111-118). Lexington, KY: The University Press of Kentucky

Longnecker, M. P., Wolff, M. S., Gladen, B. C., Brock, J. W., Grandjean, P., Jacobson, J. L., … Jensen, A. A. (2003, January). Comparison of Polychlorinated Biphenyl Levels across Studies of Human Neurodevelopment. *Environmental Health Perspectives, 111*(1), 65-70. PMID 12515680

Longo, B. M. (2009, January-February). The Kilauea Volcano Adult Health Study. *Nursing Research, 58*(1), 23-31. PMID 19092552. doi:10.1097/NNR.0b013e3181900cc5

Looker, A. C., Dallman, P. R., Carroll, M. D., Gunter, E. W., & Johnson, C. L. (1997, March 26). Prevalence of iron deficiency in the United States. *Journal of the American Medical Association, 277*(12), 973-976. PMID 9091669

Lopez, A. D., Mathers, C. D., Ezzati, M., Jamison, D. T., & Murray, C. (2006). *Global Burden of Disease and Risk Factors*. New York, NY: Oxford University Press

Lopez, R. (2002, April). Segregation and Black/White Differences in Exposure to Air Toxics in 1990. *Environmental Health Perspectives, 100*(Supplement 2), 289-295. PMID 11929740

Lorsheider, F. L., Leong, C. C., & Syed, N. I. (2001). *How Mercury Causes Brain Neuron Degeneration*. Alberta, Canada: University of Calgary

Lu, N., Samuels, M. E., & Wilson, R. (2004, November). Socioeconomic differences in health: how much do health behaviors and health insurance coverage account for? *Journal of Health Care for the Poor and Underserved, 15*(4),618-30. PMID 15531819

Lubin, J. H., Colt, J. S., Camann, D., Davis, S., Cerhan, J. R., Severson, R. K., … Hartge, P. (2004, December). Epidemiologic Evaluation of Measurement Data in the Presence of Detection Limits. *Environmental Health Perspectives, 112*(17), 1691-1696. PMID 15579415

Lucchini, R., Calza, S., Camerino, D., Carta, P., Decarli, A., Parrinello, G., … Alessio, L. (2003, August). Application of a Latent Variable Model for a Multicenter Study on Early Effects Due to Mercury Exposure. *NeuroToxicology, 24*(4-5), 605-616. PMID 12900073. doi:10.1016/S0161-813X(03)00048-2

Luecke, C. L. (2006). *Gender differences during heat strain at critical WBGT* (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 3230392)

Lundqvist, C., Zuurbier, M., Leijs, M., Johansson, C., Ceccatelli, S., Saunders, M., … Koppe, J. G. (2006, October). The effects of PCBs and dioxins on child health. *Acta Paediatrica Supplement, 95*(453), 55-64. PMID 17000571. doi:10.1080/08035320600886257

Lusk, S., Connon, C., Dirksen, M. E., & Miller, M. (2001). Workers and Worker Populations. In M. K. Salazar (Ed.), *Core Curriculum for Occupational & Environmental Health Nursing* (2nd ed., pp. 33-69). Philadelphia, PA: W. B. Saunders Company

Lutz, R., & Dedrick, R. (1987). Physiologic Pharmacokinetic Modeling of PCBs. In Safe, S. (Ed.), *Polychlorinated Biphenyls (PCBs): Mammalian and Environmental Toxicology,* (pp. 111-132). New York, NY: Springer-Verlag

Lyerly, A., Little, M., & Faden, R. (2009, October). The National Children's Study: a golden opportunity to advance the health of pregnant women. *American Journal of Public Health, 99*(10), 1742-1745. PMID 19592606. doi:10.2105/AJPH.2009.165498

Lynch, J., Smith, G., Harper, S., & Hillemeier, M. (2004b). Is Income Inequality a Determinant of Population Health? Part 2. U.S. National and Regional Trends in Income Inequality and Age- and Cause-Specific Mortality. *The Milbank Quarterly, 82*(2), 355-400. PMID 15225332. doi:10.1111/j.0887-378X.2004.00312.x

Lynch, J., Smith, G., Harper, S., Hillemeier, M., Ross, N., Kaplan, G., & Wolfson, M. (2004a). Is Income Inequality a Determinant of Population Health? Part 1. A systematic review. *The Milbank Quarterly*, 82(1), 5-99. PMID 15016244

Lynch, S., (2003, May). Cohort and life-course patterns in the relationship between education and health: a hierarchical approach. *Demography, 40*(2), 309-331. PMID 12846134

MacDonald, M. A. (2004, July-August). From Miasma to Fractals: The Epidemiology Revolution and Public Health Nursing. *Public Health Nursing, 21*(4), 380-391. PMID 15260844. doi:10.1111/j.0737-1209.2004.21412.x

MacGregor, H. E. (2004, December 20). Toxic Levels of Metals Found in Some Ayurvedic Remedies. *Los Angeles Times,* F 3

MacIntosh, D. L., Kabiru, C., Echols, S. L., & Ryan, P. B. (2001). Dietary exposure to chlorpyrifos and levels of 3,5,6-trichloro-2-pyridinol in urine. *Journal of Exposure Analysis and Environmental Epidemiology, 11*(4), 279-285. PMID 11571607. doi:10.1038/sj.jea.7500167

MacIntosh, D. L., Needham, L. L., Hammerstrom, K. A., & Ryan, P. B. (1999, September-October). A longitudinal investigation of selected pesticide

metabolites in urine. *Journal of Exposure Analysis and Environmental Epidemiology, 9*(5), 494-501. PMID 10554151

MacIntosh, D. L., Spengler, J. D., Ozkaynak, H., Tsai, L., & Ryan, P. B. (1996, February). Dietary Exposures to Selected Metals and Pesticides. *Environmental Health Perspectives, 104*(2), 202-209. PMID 8820589

Mackenbach, J. P. (1998, October). Multi-level ecoepidemiology and parsimony. (Peer Commentary on the article "Does risk factor epidemiology put epidemiology at risk? Peering into the future" by Susser, 1998, October). *Journal of Epidemiology and Community Health, 52*(10), 614-615. PMID 10023454

Mackenbach, J. P., Stirbu, I., Roskam, A.-J., Schaap, M. M., Menvielle, G., Leinsalu, M., … the European Union Working Group on Socioeconomic Inequalities in Health. (2008, June 5). Socioeconomic Inequalities in Health in 22 European Countries. *The New England Journal of Medicine, 358*(23), 2468-2481. PMID 18525043. doi:10.1056/NEJMsa0707519

Maclure, M., & Schneeweiss, S. (2001, January). Causation of Bias: The Episcope. *Epidemiology, 12*(1), 114-122. PMID 11138805

MacMahon, B., & Pugh, T. F. (1970). *Epidemiologic Methods*. Boston, MA: Little, Brown

Mader, R., Kokalji, A., Kratochvil, E., Pilger, A., & Rudiger, H. (2008) Longitudinal biomonitoring of nurses handling antineoplastic drugs. *Journal of Clinical Nursing, 18*, 263-269. PMID 18624785. doi:10.1111/j.1365-2702.2007.02189.x

Mage, D. T., Allen, R. H., Gondy, G., Smith, W., Barr, D. B., & Needham, L. L. (2004, November). Estimating pesticide dose from urinary pesticide concentration data by creatinine correction in the Third National Health and Nutrition Examination Survey (NHANES-III). *Journal of Exposure Analysis and Environmental Epidemiology, 14*(6), 457-465. PMID 15367927. doi:10.1038/sj.jea.7500343

Magee, T., Lee, S. M., Giuliano, K. K., & Munro, B. (2006, March-April). Generating New Knowledge from Existing Data: The Use of Large Data Sets for Nursing Research. *Nursing Research, 55*(2), S50-S56. PMID 16601635

Mahaffey, K. R. (1977, August). Quantities of lead producing health effects in humans: sources and bioavailability. *Environmental Health Perspectives, 19*, 285-295. PMID 908307

Mahaffey, K. R. (1980). Nutrient-Lead Interactions. In R. L. Singhai & J. A. Thomas (Eds.), *Lead Toxicity*. (pp. 425-460.) Baltimore, MD: Urban & Schwarzenberg

Mahaffey, K. R. (1998, August 26). Methylmercury Exposure and Neurotoxicity. *Journal of the American Medical Association, 280*(8), 737-738. PMID 9728648

Mahaffey, K. R. (1999, September-October). Methylmercury: A New Look at the Risks. *Public Health Reports, 114*(5), 396-399, 402-413. PMID 10590759

Mahaffey, K. R. (2005). Mercury Exposure: Medical and Public Health Issues. *Transactions of the American Clinical and Climatological Association, 116,* 127-154. PMID 16555611

Mahaffey, K. R., Clickner, R. P., & Bodurow, C. C. (2004, April). Blood Organic Mercury and Dietary Mercury Intake: National Health and Nutrition Examination Survey, 1999 and 2000. *Environmental Health Perspectives, 112*(5), 562-570. PMID 15064162

Mahaffey, K. R., Clickner, R. P., & Jeffries, R. A. (2009, January). Adult Women's Blood Mercury Concentrations Vary Regionally in the United States: Association with Patterns of Fish Consumption (NHANES 1999-2004). *Environmental Health Perspectives, 117*(1), 47-53. PMID 19165386

Maia, C. S., Ferreira, V. M., Kahwage, R. L., do Amaral, M. N., Serra, R. B., dos Santos, S. N., … Diniz, C.W. (2010, November). Adult brain nitrergic activity after concomitant prenatal exposure to ethanol and methylmercury. *Acta Histochemica, 112*(6), 583-591. PMID 19748654. doi:10.1016/j.acthis.2009.06.004

Maia, C. S., Lucena, G. M., Corrêa, P. B., Serra, R. B., Matos, R. W., Menezes, F. C., … Ferreira, V. M. (2009a, January). Interference of ethanol and methylmercury in the developing central nervous system. *NeuroToxicology, 30*(1), 23-30. PMID 19100288.doi:10.1016/j.neuro.2008.11.008

Mancinelli, R., Vitali, M., Ceccanti, M. (2009, April-June). Women, alcohol and the environment: an update and perspectives in neuroscience. *Functional Neurology, 24*(2), 77-81. PMID 19775534

Manufactured Home Construction and Safety Standards. 42 U.S.C. §70, 1976

Mapel, D. W., Coultas, D. B., James, D. S., Hunt, W. C., Stidley, C. A., & Gilliland, F. D. (1997, May). Ethnic differences in the prevalence of nonmalignant respiratory disease among uranium miners. *American Journal of Public Health, 87*(5), 833-838. PMID 9184515

Mariën, K., & Patrick, G. M. (2001, May-June). Exposure analysis of five fish-consuming populations for overexposure to methylmercury. *Journal of Exposure Analysis and Environmental Epidemiology, 11*(3), 193-206. PMID 11477517. doi:10.1038/sj.jea.7500160

Marin, G., Sabogal, F., Marin, B., Otero-Sabogal, R., & Paerez-Stable, E. (1987). Development of a Short Acculturation Scale for Hispanics. *Hispanic Journal of Behavioral Sciences, 9*, 183-205. PMID 12267282

Markides, K. S., & Coreil, J. (1986, May-June). The health of Hispanics in the southwestern United States: an epidemiologic paradox. *Public Health Reports,101*(3), 253-265. PMID 3086917

Marsh, D. O., Clarkson, T. W., Myers, G. J., Davidson, P. W., Cox, C., Cernichiari, E., … Berlin, M. (1995, Winter). The Seychelles Study of Fetal Methylmercury Exposure and Child Development: Introduction. *NeuroToxicology, 16*(4), 583-596. PMID 8714865

Marsh, D. O., Myers, G. J., Clarkson, T. W., Amin-Zaki, L., Tikriti, S., Majeed, M. A., & Dabbaqh, A. R. (1981, November). Dose-Response Relationship for Human Fetal Exposure to Methylmercury. *Clinical Toxicology, 18*(11), 1311-1318. PMID 7341057. doi:10.3109/00099308109035071

Marshall, W. A., Clough, R., & Gehrels, W. R. (2009, April 1). The isotopic record of atmospheric lead fall-out on an Icelandic salt marsh since AD 50. *Science and*

the Total Environment,407(8), 2734-2748. PMID 19157518. doi:10.1016/j.scitotenv.2008.12.009

Martens, P., Rotmans, J., & Rothman, D. (2002). Integrated assessment modeling of human health impacts. In P. Martens & A. J. McMichael (Eds.), *Environmental Change, Climate and Health* (pp. 197-225). Cambridge, U.K.: Cambridge University Press

Marsit, C., LaMontagne, A., & Kelsey, K. (2005). Biologic Markers in Occupational and Environmental Medicine. In L. Rosenstock, M. R. Cullen, C. A. Brodkin & C. A. Redlich (Eds.), *Textbook of Clinical Occupational and Environmental Medicine* (2nd ed., pp. 139-145). Philadelphia, PA: Elsevier Saunders

Martin, M., & Naleway, C. (2004). The inhibition of mercury absorption by dietary ethanol in humans: cross-sectional and case-control studies. *Occupational and Environmental Medicine, 61*(8), 1-3. PMID 14739392

Mason, H. J., Hindell, P., & Williams, N. R. (2001, February). Biological monitoring and exposure to mercury. *Occupational Medicine (London), 51*(1), 2-11. PMID 11235823

Mason, H. J., & Wilson, K. (1999, March-April). Biological Monitoring: The Role of Toxicokinetics and Physiologically-Based Pharmacokinetic Modeling. *American Industrial Hygiene Association Journal 60*(2), 237-242. PMID 10222574

Massoudi, M. S. (2004). *Field Investigations: CDC Perspective on Outbreak Investigations*. Atlanta, GA: Centers for Disease Control and Prevention

Mather, F. J., White, L.-A., Langlois, E. C., Shorter, C. F., Swalm, C. M., Shaffer, J. G., & Hartley, W. R. (2004, October). Statistical Methods for Linking Health, Exposure, and Hazards. *Environmental Health Perspectives, 112*(14), 1440-1445. PMID 15471740

Matsuyama, A., Yasuda, Y., Yasutake, A., Xiaojie, L., Pin, J., Li, L., … Liya, Q. (2005, December). Relationship Between Leached Total Mercury and Leached Methylmercury from Soil Polluted by Mercury in Wastewater from an Organic Chemical Factory in the People's Republic of China. *Bulletin of Environmental Contamination Toxicology, 75*(6), 1234-1240. PMID 16402317. doi:10.1007/s00128-005-0881-y

Mauderly, J., & Samet, J. (2009, January). Is there Evidence for Synergy among Air Pollutants in Causing Health Effects? *Environmental Health Perspectives, 117*(1), 1-6. PMID 19165380. doi:10.1289/ehp.11654

Mavreas, V., Bebbinton, P., & Der, G. (1989). The structure and validity of acculturation. *Social Psychiatry and Psychiatric Epidemiology. 24*, 233-240

Maxwell, A. (2009). Acculturation. In L. Breslow (Ed.) *Encyclopedia of Public Health*. Retrieved April 1, 2011 from http://www.enotes.com/public-health-encyclopedia/acculturation

Mayberry, R., Mili, F., & Ofili, E. (2000). Racial and Ethnic Differences in Access to Medical Care. *Medical Care Research and Review, 57*(Supplement 1), 108-145. PMID 11092160

McAlpine, D., & Araki, S. (1958, September 20). Minamata Disease: an unusual neurological disorder caused by contaminated fish. *The Lancet II (Br.), 272*(7047), 629-631. PMID 13588955

McCauley, L. (1998, January). Chemical Mixtures in the Workplace. *Association of American Occupational Health Nurses Journal, 46*(1), 29-40. PMID 9481217

McCauley, L. (2002, August). Environmental Health Nursing Research. In Nastoff, T., Drew, D., Wigington, P., Wakefield, J., Phillips, J. & O'Fallon, L. (Eds.), *Final Report of Nursing and Environmental Health Roundtable*. Research Triangle Park, N.C.: Agency for Toxic Substances and Disease Registry

McComb, D. (1997, May-June). Occupational Exposure to Mercury in Dentistry and Dentist Mortality. *Journal of the Canadian Dental Association, 63*(5), 372-376. PMID 9170753

McCurdy, T., Glen, G., Smith, L., & Lakkadi, Y. (2000). The National Exposure Research Laboratory's Consolidated Human Activity Database. *Journal of Exposure Analysis and Environmental Epidemiology, 10*, 566-578. PMID 11140440

McDiarmid, M. A., & Gehle, K. (2006, December). Preconception Brief: Occupational and Environmental Exposures. *Maternal and Child Health Journal, 10,* S123-S128. AN 22344075

McDowell, M. A., Dillon, C. F., Osterloh, J., Bolger, P. M., Pellizzari, E., Fernando, R., … Mahaffey, K. R. (2004, August). Hair Mercury Levels in U.S. Children and Women of Childbearing Age: Reference Range Data from NHANES 1999-2000. *Environmental Health Perspectives, 112*(11), 1165-1171. PMID 15289161

McEwen, B.S. (1998, January 15). Protective and damaging effects of stress mediators. *The New England Journal of Medicine, 338*(3), 171-179, PMID 9428819

McGee, D., Liao, Y., Cao, G., & Cooper, R. (1999). Self-Reported Health Status and Mortality in a Multi-Ethnic U.S. Cohort. *American Journal of Epidemiology, 149*(1), 41-46. PMID 9883792

McGlynn, K., Guo, X., Graubard, B., Brock, J., Klebanoff, M., & Longnecker, M. (2009, September). Maternal Pregnancy Levels of Polychlorinated Biphenyls and Risk of Hypospadias and Cryptorchidism in Male Offspring. *Environmental Health Perspectives, 117*(9), 1472-1476. PMID 19750116. doi:10.1289/ehp.0800389

McGovern, V. (2003, January). Sex Matters: Exploring Differences in Responses to Exposures. *Environmental Health Perspectives, 111*(1), A24-A25. PMID 12515697

McGovern, V. (2009, July). Smoking Gain? Secondhand Smoke Exposure Influences Body Weight, Lipid Profiles in Offspring. *Environmental Health Perspectives, 117*(7), A310. PMID 19654904

McGuinness, B. M., Buck, G. M., Mendola, P., Sever, L. E., & Vena, J. E. (2001, May-June). Infecundity and consumption of polychlorinated biphenyl-contaminated fish. *Archives of Environmental Health, 56*(3), 250-253. PMID 11480501

McGuire, S. L., & Gerber, D. E. (1999b). Teaching Students about Nursing and the Environment: Part 2: Legislation and Resources. *Journal of Community Health Nursing, 16*(2), 81-94. PMID 10349819

McIntire, M. S., & Angle, C. R. (1972, August 11). Air Lead in Relation to Lead in Blood of Black School Children Deficient in Glucose-6-Phosphate Dehydrogenase. *Science, 177*(4048), 520-522. PMID 5077325

McKelvey, W., Gwynn, R. C., Jeffery, N., Kass, D., Thorpe, L. E., Garg, R. K., … Parsons, P. J. (2007, October). A Biomonitoring Study of Lead, Cadmium and Mercury in the Blood of New York City Adults. *Environmental Health Perspectives, 115*(10), 1435-1441. PMID 17938732. doi:10.1289/ehp.10056

McKernan, J. L. (2006). *Development and evaluation of proposed equations for improved exothermic process control* (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 3217236)

McKeown, T. (1991). *The Origins of Human Disease*. Oxford, U.K.: Wiley-Blackwell

McKeown-Eyssen, G. E., & Ruedy, J. (1983a, October). Methyl Mercury Exposure in Northern Quebec. I. Neurologic Findings in Adults. *American Journal of Epidemiology, 118*(4), 461-469. PMID 6637973

McKeown-Eyssen, G. E., Ruedy, J., & Neims, A. (1983b, October). Methyl Mercury Exposure in Northern Quebec. II. Neurologic Findings in Children. *American Journal of Epidemiology, 118*(4), 470-479. PMID 6637974

McKone, T. E., Ryan, P. B., & Ozkaynak, H. (2009, January). Exposure information in environmental health research: current opportunities and future directions for particulate matter, ozone and toxic air pollutants. *Journal of Exposure Science and Environmental Epidemiology, 19*(1), 30-44. PMID 18385670. doi:10.1038/jes.2008.3

McMichael, A. J. (1999, May 15). Prisoners of the proximate: loosening the constraints on epidemiology in an age of change. *American Journal of Epidemiology, 149*(10), 887-897. PMID 10342797

McMichael, A. J., & Martins, P. (2002). Global environmental changes: anticipating and assessing risks to health. In P. Martens & A. J. McMichael (Eds.), *Environmental Change, Climate and Health* (pp. 1-17). Cambridge, UK: Cambridge University Press

McMichael, A. J., Vimpani, G. V., Robertson, E. F., Baghurst, P. A., & Clark, P. D. (1986). The Port Pirie cohort study: maternal blood lead and pregnancy outcome. *Journal of Epidemiology and Community Health, 40,* 18-25. PMID 3711766

McPhaul, K. M., & Lipscomb, J. A. (2005, January). Incorporating Environmental Health into Practice. *Association of American Occupational Health Nurses Journal, 53*(1), 31-36. PMID 15675155

McPherson, K. (1998, October). Wider "causal thinking in the health sciences." (Peer Commentary on the article "Does risk factor epidemiology put epidemiology at risk? Peering into the future" by Susser, 1998, October). *Journal of Epidemiology and Community Health, 52*(10), 612-613. PMID 10023453

McRill, C., Boyer, L. V., Flood, T. J., & Ortega, L. (2000, January). Mercury toxicity due to use of a cosmetic cream. *Journal of Occupational and Environmental Medicine (JOEM), 42*(1), 4-7. PMID 10652682

Meacham, C., Freudenrich, T., Anderson, W., Sui, L., Lyons-Darden, T., Barone, S., … Shafer, T. (2005, June 1). Accumulation of methylmercury or polychlorinated biphenyls in *in vitro* models of rat neuronal tissue. *Toxicology and Applied Pharmacology, 205*(2),177-187. PMID 15893545. doi:10.1016/j.taap.2004.08.024

Mechanic, D., & Tanner, J. (2007, September-October). Vulnerable People, Groups and Populations: Societal View. *Health Affairs, 26*(5), 1220-1230. PMID 17848429. doi:10.1377/hlthaff.26.5.1220

Medinsky, M. A., & Valentine, J. L. (2003). Toxicokinetics. In C. D. Klaassen & J. B. Watkins (Eds.), *Casarett and Doull's Essentials of Toxicology* (pp. 98-107). New York, NY: McGraw-Hill

Meeker, J. D., Barr, D. B., & Hauser, R. (2006, October). Thyroid hormones in relation to urinary metabolites of non-persistent insecticides in men of reproductive age. *Reproductive Toxicology, 22*(3), 437-442. PMID 16584866. doi:10.1016/j.reprotox.2006.02.005

Meeker, J., Missmer, S., Altshul, L., Vitonis, A., Ryan, L., Cramer, D., & Hauser, R. (2009, July). Serum and follicular fluid organochlorine concentrations among women undergoing assisted reproduction technologies. *Environmental Health, 8*(32), 1-30. PMID 19594949. doi:10.1186/1476-069X-8-32

Meggs, W. J. (2003). Permanent Paralysis at Sites of Dermal Exposure to Chlorpyrifos. *Journal of Toxicology and Clinical Toxicology, 41*(6), 883-886. PMID 14677802

Mei, Z., Parvanta, I., Cogswell, M. E., Gunter, E. W., & Grummer-Strawn, L. M. (2003, May). Erthrocyte protoporphyrin or hemoglobin: which is a better screening test for iron deficiency in children and women? *American Journal of Clinical Nutrition, 77*(5), 1229-1233. PMID 12716676

Meinders, A.-J. & Meinders, A. E. (2010, March 16). How much water do we really need to drink? *Nederlands tijdschrift voor geneeskunde, 154*, A1757. PMID 20356431. (Translated from Dutch.)

Mendola, P., Buck, G. M., Vena, J. E., Zielezny, M., Sever, L. E. (1995, May). Consumption of PCB-contaminated sport fish and risk of spontaneous fetal death. *Environmental Health Perspectives, 103*(5), 498-502. PMID 7656880

Mendola, P., Selevan, S. G., Gutter, S., & Rice, D. (2002). Environmental Factors Associated with a Spectrum of Neurodevelopmental Deficits. *Mental Retardation and Developmental Disabilities Research Reviews, 8,* 188-197. PMID 12216063. doi:10.1002/mrdd.10033

Mendoza, F. S. (2009, November). Health disparities and children in immigrant families: a research agenda. *Pediatrics, 124*(Supplement 3), S187-S195. PMID 19861469. doi:10.1542/peds.2009-1100F

Menke, A., Muntner, P., Batuman, V., Silbergeld, E. K., & Guallar, E. (2006, September 26). Blood Lead Below 0.48 μmol/l (10 μg/dl) and Mortality Among U.S. Adults. *Circulation, 114*(13), 1388-1394. PMID 16982939

Menvielle, G., Boshuizen, H., Kunst, A. E., Dalton, S. O., Vineis, P., Bergmann, M. M., … Bas Bueno-de-Mesquita, H. (2009, March 4). The Role of Smoking and Diet in Explaining Educational Inequalities in Lung Cancer Incidence. *Journal of the National Cancer Institute, 101*(5), 321-330. PMID 19244178. doi:10.1093/jnci/djn513

Menzie, C. A., MacDonell, M. M., & Mumtaz, M. (2007, May). A Phased Approach for Assessing Combined Effects from Multiple Stressors. *Environmental Health Perspectives, 115*(5), 807-816. PMID 17520072. doi:10.1289/ehp.9331

Mergler, D., Anderson, H., Chan, L., Mahaffey, K., Murray, M., Sakamoto, M., … Panel on Health Risks and Toxicological Effects of Methylmercury. (2007, February). Methylmercury Exposure and Health Effects in Humans: A Worldwide Concern. *Ambio, 36*(1), 3-11. PMID 17408186

Merkin, S., Basurto-Dávila, R., Karlamangla, A., Bird, C. E., Lurie, N., Escarce, J., & Seeman, T. (2009, March). Neighborhoods and Cumulative Biological Risk Profiles by Race-Ethnicity in a National Sample of U.S. Adults: NHANES III. *Annals of Epidemiology, 19*(3), 194-201. PMID 19217002. doi:10.1016/j.annepidem.2008.12.006

Merletti, F., Soskolne, C., & Vineis, P. (1998). Epidemiological Method Applied to Occupational Health and Safety. In J. M. Stellman (Ed.), *ILO Encyclopedia of Occupational Health and Safety* (4th ed., pp. 28.2-28.6). Geneva, CH: International Labour Organisation

Merlo, J., Chaix, B., Yang, M., Lynch, J., & Råstam, L. (2005, December). A brief conceptual tutorial on multilevel analysis in social epidemiology: interpreting neighbourhood differences and the effect of neighbourhood characteristics on individual health. *Journal of Epidemiology and Community Health, 59*(12), 1022-1028. PMID 16286487. doi:10.1136/jech.2004.028035

Merne Smaldone, A., & Connor, J. A. (2003, August). The Use of Large Administrative Data Sets in Nursing Research. *Applied Nursing Research, 16*(3), 206-207. PMID 12931336

Metcalf, S. W., & Orloff, K. G. (2004, April 23-May 28). Biomarkers of Exposure in Community Settings. *Journal of Toxicology and Environmental Health, A, 67*(8-10), 715-726. PMID 15192864

Metzgar, C. (2009). Writing Worth Reading: Hormesis – Why It Is Important to Toxicology & Toxicologists. *Professional Safety, 54*(3), 41

Meyer, J., Warren N., & Reisine S. (2010, February). Racial and ethnic disparities in low birth weight delivery associated with maternal occupational

characteristics. *American Journal of Industrial Medicine, 53*(2), 153-162. PMID 19444807. doi:10.1002/ajim.20706

Michigan Department of Environmental Quality. (1998, April 20). *Mercury Use Tree.* Retrieved April 1, 2011 from http://www.deq.state.mi.us/documents/deq-ead-p2-mercury-mercusetree.pdf

Miles, L. (1977). Measurement of serum ferritin by a 2-site immunoradiometric assay. In G. Abraham (Ed.), *Handbook of Radioimmunoassay* (Chapter Four). New York, NY: Marcel Dekker, Inc.

Miller, D. T.. Paschal, D. C.. Gunter, E. W., Stroud, P. E. & D'Angelo, J. (1987, December). Determination of Lead in Blood Using Electrothermal Atomisation Atomic Absorption Spectrometry with a L'vov Platform and Matrix Modifier. *The Analyst, 112*(12), 1701-1704. doi:10.1039/AN9871201701

Miller, H. W. (1973, February). Plan and Operation of the Health and Nutrition Examination Survey (1971-1973). *Vital and Health Statistics Series, 1*(10b), 1-10. PMID 4540157

Miller, R. K., & Bellinger, D. (1993). Metals. In M. Paul (Ed.), *Occupational and Environmental Hazards: A Guide for Clinicians* (pp. 233-252). Baltimore, MD: Williams & Wilkins

Mills, A. L., Messer, K., Gilpin, E. A., & Pierce, J. P. (2009, October). The effect of smoke-free homes on adult smoking behavior: a review. *Nicotine and Tobacco Research, 11*(10), 1131-1141. PMID 19633273. doi:10.1093/ntr/ntp122

Mills, R. J. (2001, September). *Health Insurance Coverage: 2000*. Retrieved April 1, 2011 from http://www.census.gov/prod/2001pubs/p60-215.pdf

Milman, N., Bergholt, T., Byg, K.-E., Eriksen, L., & Hvas, A.-M. (2007, July). Reference intervals for haematological variables during normal pregnancy and postpartum in 434 healthy Danish women. *European Journal of Haematology, 79*(1), 39-46. PMID 17598837. doi:10.1111/j.1600-0609.2007.00873.x

Mina, K., Fritschi, L., & Knuiman, M. (2008, February). Do aggregates of multiple questions better capture overall fish consumption than summary questions? *Public Health Nutrition, 11*(2), 196-202. PMID 17610758. doi:10.1017/S1368980007000468

Minnema, D., & Cooper, G. (1990). Assessment of the Effects of Lead and Mercury *in vitro* on Neurotransmitter Release. In E. C. Foulkes (Ed.), *Biological Effects of Heavy Metals,* (Vol. 1, pp. 19-58). Boca Raton, FL: CRC Press

Miodovnik, A., & Landrigan, P. (2009, July). The U.S. Food and Drug Administration Risk Assessment on Lead in Women's and Children's Vitamins is Based on Outdated Assumptions. *Environmental Health Perspectives, 117*(7), 1021-1022. PMID 19654907

Miranda, M. L., Kim, D., Reiter, J., Overstreet Galeano, M. A., & Maxson P. (2009, November). Environmental contributors to the achievement gap. *NeuroToxicology,30*(6), 1019-1024. PMID 19643133. doi:10.1016/j.neuro.2009.07.012

Misener, T., Watkins, J., & Ossege, J. (1994, April). Public Health Nursing Research Priorities: A Collaborative Delphi Study. *Public Health Nursing, 11*(2), 66-74. PMID 8029183

Mitchell, D., Haan, M., Steinberg, F., & Visser, M. (2003). Body composition in the elderly: the influence of nutritional factors and physical activity. *The Journal of Nutrition, Health & Aging, 7*(3), 130-139. PMID 12766789

Moffitt, T. E. (1996, February 7). Measuring Children's Antisocial Behaviors. *Journal of the American Medical Association, 275*(5), 403-404. PMID 8569022

Mohai, P., & Bryant, B. (1992a). Environmental Racism: Reviewing the Evidence. In B. Bryant & P. Mohai (Eds.), *Race and the Incidence of Environmental Hazards* (pp. 163-176). Boulder, CO: Westview Press

Mohai, P., & Bryant, B. (1992b, March-April). Race, poverty and the environment. *EPA Journal, 18*(1), 6-9. AN 9609101474

Moline, J., & Landrigan, P. (2005). Lead. In L. Rosenstock, M. Cullen, C. A. Brodkin & C. A. Redlich (Eds.), *Textbook of Clinical Occupational and Environmental Medicine* (2nd ed., pp. 967-978). Philadelphia, PA: Elsevier Saunders

Molloy, A., Kirke, P., Brody, L., Scott, J., & Mills, J. (2008, June). Effects of folate and vitamin B12 deficiencies during pregnancy on fetal, infant and child development. *Food and Nutrition Bulletin, 29*(Supplement 2), S101-S115. PMID 18709885

Molnar, S. (1998). *Human Variation: Races, Types and Ethnic Groups*. Upper Saddle River, NJ: Prentice Hall

Monosson, E. (2005, April). Chemical Mixtures: Considering the Evolution of Toxicology and Chemical Assessment. *Environmental Health Perspectives, 113*(4), 383-390. PMID 15811826

Monroe, C. T., Pezzino, G., Knoche, L. L., Henning, L., & Belt, P. (1999, November). Public Health Response to Metallic Mercury Spills in Kansas. *Journal of Public Health Management Practice, 5*(6), 13-17. PMID 10662059

Moore, M. N., Depledge, M., Readman, J., & Leonard, D. (2004), An integrated biomarker-based strategy for ecotoxicological evaluation of risk in environmental management. *Mutation Research, 552*, 247-268. PMID 15288556. doi:10.1016/j.mrfmmm.2004.06.028

Moore, M. R., Hughes, M. A., & Goldberg, D. J. (1979). Lead Absorption in Man from Dietary Sources. *International Archives of Occupational and Environmental Health, 44*(2), 81-90. PMID 521168

Monson, R. R. (1980). *Occupational Epidemiology*. Boca Raton, FL.: CRC Press

Montez, J., Angel, J., & Angel, R. (2009, June). Employment, marriage, and inequality in health insurance for Mexican-origin women. *Journal of Health and Social Behavior, 50*(2), 132-148. PMID 19537456

Montgomery, L., & Carter-Pokras, O. (1993, September). Health Status by Social Class and/or Minority Status: Implications for Environmental Equity Research. *Toxicology and Industrial Health, 9*(5), 729-773. PMID 8184442

Montoya, M. (2007, February). Bioethnic Conscription: Genes, Race and Mexicana/o Ethnicity in Diabetes Research. *Cultural Anthropology, 22*(1), 94-128

Mood, L. (2002, August). Translating Environmental Knowledge Into Nursing Practice. In Nastoff, T., Drew, D., Wigington, P., Wakefield, J., Phillips, J. & O'Fallon, L. (Eds.), *Final Report of Nursing and Environmental Health Roundtable*. Research Triangle Park, N.C.: Agency for Toxic Substances and Disease Registry

Morabia, A. (1998, October). Epidemiology and bacteriology in 1900: who is the handmaid of whom? (Peer Commentary on the article "Does risk factor epidemiology put epidemiology at risk? Peering into the future" by Susser, 1998, October). *Journal of Epidemiology and Community Health, 52*(10), 617-618. PMID 10023453

Moralez, L., Gutierrez, P., & Escarce, J. (2005, July-August). Demographic and socioeconomic factors associated with blood lead levels among Mexican-American children and adolescents in the United States. *Public Health Reports, 120*(4), 448-454. PMID 16025725

Morello-Frosch, R., Brody, J., Brown, P., Altman, R., Rudel, R., & Perez, C. (2009, February 28). Toxic ignorance and right-to-know in biomonitoring results

communication: a survey of scientists and study participants. *Environmental Health, 8*(6), 1-13. PMID 19250551. doi:10.1186/1476-069X-8-6

Morello-Frosch, R., & Lopez, R. (2006a, October). The riskscape and the color line: examining the role of segregation in environmental health disparities. *Environmental Research, 102*(2), 181-196. PMID 16828737. doi:10.1016/j.envres.2006.05.007

Morello-Frosch, R., & Shenassa, E. D. (2006b, August). The Environmental 'Riskscape' and Social Inequality: Implications for Explaining Maternal and Child Health Disparities. *Environmental Health Perspectives, 114*(8), 1150-1153. PMID 16882517. doi:10.1289/ehp.8930

Morgenstern, H., & Thomas, D. (1993, December). Principles of Study Design in Environmental Epidemiology. *Environmental Health Perspectives, 101*(Supplement 4), 23-38. PMID 8206038

Mori, C., Komiyama, M., Adachi, T., Sakurai, K., Nishimura, D., Takashima, K., & Todaka, E. (2003, May). Application of Toxicogenomic Analysis to Risk Assessment of Delayed Long-Term Effects of Multiple Chemicals Including Endocrine Disruptors in Human Fetuses. *Environmental Health Perspectives Toxicogenomics, 111*(6), 803-809. PMID 12735105

Morón, C., & Viteri, F. (2009). Update on common indicators of nutritional status: food access, food consumption and biochemical measures of iron and anemia. *Nutrition Reviews, 67*(Supplement 1), S31-S35. PMID 19453675. doi:10.1111/j.1753-4887.2009.00156.x

Morrison, V. (2005). *A contemporary analysis of the smallpox vaccination program*. (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 3172761)

Morton, J., Mason, H. J., Ritchie, K. A., & White, M. (2004, January-February). Comparison of hair, nails and urine for biological monitoring of low level inorganic mercury exposure in dental workers. *Biomarkers, 9*(1), 47-55. AN 13006651

Mosher, W., Deang, L., & Bramlett M. (2003, April). Community environment and women's health outcomes: contextual data from National Center for Health Statistics. *Vital Health Statistics, 23*(23), 1-81. PMID 12762084

Mosquin, P., Whitmore, R., Suerken, C., & Quackenboss, J. (2005, September). Population coverage and nonresponse bias in a large-scale human exposure study. *Journal of Exposure Analysis and Environmental Epidemiology, 15*(5), 431-438. PMID15674317. doi:10.1038/sj.jea.7500421

Muckle, G., Ayotte, P., Dewailly, E., Jacobson, S. W., & Jacobson, J. L. (2001a, September). Determinants of polychlorinated biphenyls and methylmercury exposure in Inuit women of childbearing age. *Environmental Health Perspectives, 109*(9), 957-963. PMID 11673127

Muckle, G., Ayotte, P., Dewailly, E. E., Jacobson, S.W., & Jacobson, J. L. (2001b, December). Prenatal exposure of the northern Québec Inuit infants to environmental contaminants. *Environmental Health Perspectives, 109*(12), 1291-1299. PMID 11748038

Mueller, B. A., Kuehn, C. M., Shapiro-Mendoza, C. K., & Tomashek, K. M. (2007, May). Fetal Deaths and Proximity to Hazardous Waste Sites in Washington State. *Environmental Health Perspectives, 115*(5), 776-780. PMID 17520067. doi:10.1289/ehp.9750

Muir, D. (1995). Cause of occupational disease. *Occupational and Environmental Medicine, 52*, 289-293. PMID 7795749

Multigner, L., Kadhei, P., Pascal, M., Huc-Terki, F., Kercret, H., & Massart, C. (2008). Parallel assessment of male reproductive function in workers and wild rats exposed to pesticides in banana plantations in Guadeloupe. *Environmental Health, 7*(40), 1-30. PMID 18667078. doi:10.1186/1476-069X-7-40

Mujuru, P., & Niezen, C. (2004, October). Evaluation of an environmental health education program: assessing changes in knowledge of health professionals. *Association of American Occupational Health Nurses Journal, 52*(10), 436-441. PMID 15508858

Mulry, M. (2006, February 28). *Summary of Accuracy and Coverage Evaluation for Census 2000*. Washington, DC: Statistical Research Division, U.S. Census Bureau

Mumenthaler, M. S., Taylor, J. L., & Yesavage, J. A. (2000, September). Ethanol pharmacokinetics in white women: nonlinear model fitting versus zero-order elimination analyses (Abstract). *Alcohol Clinical and Experimental Research, 24*(9), 1353-1362. PMID 11003200

Mumtaz, M. M., Cibulas, W., & de Rosa, C. T. (1995, July). An Integrated Framework to Identify Significant Human Exposures. *Chemosphere, 31*(1), 2485-2498. PMID 7670861

Mumtaz, M. M., DeRosa, C., & Durkin, P. (1994). Approaches and challenges in risk assessments of chemical mixtures. In Yang, R. (Ed.), *Toxicology of Chemical Mixtures: Case Studies, Mechanisms and Novel Approaches*, (pp. 565-597). New York, NY: Academic Press

Mumtaz, M. M., DeRosa, C. T., Groten, J., Feron, V. J., Hansen, H., & Durkin, P. R. (1998, December). Estimation of Toxicity of Chemical Mixtures through Modeling of Chemical Interactions. *Environmental Health Perspectives, 106*(Supplement 6), 1353-1360. PMID 9860892

Mumtaz, M. M., & Durkin, P. R. (1992, November-December). A Weight-of-Evidence Approach for Assessing Interactions in Chemical Mixtures. *Toxicology and Industrial Health, 8*(6), 377-406. PMID 7570620

Mumtaz, M. M., Ruiz, P., & de Rosa, C. T. (2007, September 1). Toxicity assessment of unintentional exposure to multiple chemicals. *Toxicology and Applied Pharmacology, 223*(2), 104-113. PMID 17599373. doi:10.1016/j.taap.2007.04.015

Murata, K., Grandjean, P., & Dakeishi, M. (2007, October). Neurophysiological Evidence of Methylmercury Neurotoxicity. *American Journal of Industrial Medicine, 50*(10), 765-771. PMID 17450510. doi:10.1002/ajim.20471

Murata, K., Weihe, P., Budtz-Jørgensen, E., & Grandjean, P. (1999a, July-August). Evoked Potentials in Faroese Children Prenatally Exposed to Methylmercury. *NeuroToxicology and Teratology, 21*(4), 471-472. PMID 10440491

Murata, K., Weihe, P., Budtz-Jørgensen, E., Jørgensen, P. J., & Grandjean, P. (2004, February). Delayed Brainstem Auditory Evoked Potential Latencies in 14-Year-Old Children Exposed to Methylmercury. *The Journal of Pediatrics, 144*(2), 177-183. PMID 14760257. doi:10.1016/j.jpeds.2003.10.059

Murata, K., Weihe, P., Budtz-Jørgensen, E., Jørgensen, P. J., & Grandjean, P. (2006, October). Correction for "Delayed brainstem auditory evoked potential latencies in 14-year-old children exposed to methylmercury" by Murata, Weihe, Budtz-Jørgensen, Jørgensen, & Grandjean, 2004. *The Journal of Pediatrics, 149*(4), 583-584. PMID 17131521

Murata, K., Weihe, P., Renzoni, A., Debes, F., Vasconcelos, R., Zino, F., … Grandjean, P. (1999b, July-August). Delayed Evoked Potentials in Children Exposed to Methylmercury from Seafood. *NeuroToxicology and Teratology, 21*(4), 343-348. PMID 10440477.

Murray, C. J., & Lopez, A. D. (1999, September). On the Comparable Quantification of Health Risks: Lessons from the Global Burden of Disease Study. *Epidemiology, 10*(5), 594-605. PMID 10468439

Murray, L. R. (2003, February). Sick and Tired of Being Sick and Tired: Scientific Evidence, Methods and Research Implications for Racial and Ethnic Disparities in Occupational Health. *American Journal of Public Health, 93*(2), 221-226. PMID 12554573

Mushak, P. (1998, December). Uses and Limits of Empirical Data in Measuring and Modeling Human Lead Exposure. *Environmental Health Perspectives Supplement, 106*(S6), 1467-1484. PMID 9860906

Mushak, P. (2007, April). Hormesis and Its Place in Non-Monotonic Dose-Response Relationships: Some Scientific Reality Checks. *Environmental Health Perspectives, 115*(4), 500-506. PMID 17450215

Musiek, F. E., & Hanlon, D. P. (1999, June). Neuroaudiological Effects in a Case of Fatal Dimethylmercury Poisoning. *Ear and Hearing, 20*(3), 271-275. PMID 10386853

Mutch, E., & Williams, F. M. (2006, July 5). Diazinon, chlorpyrifos and parathion are metabolised by multiple cytochromes P450 in human liver. *Toxicology, 224*(1-2), 22-32. PMID 16757081. doi:10.1016/j.tox.2006.04.024

Myers, G. J., Thurston, S. W., Pearson, A. T., Davidson, P. W., Cox, C., Shamlaye, C. F., … Clarkson, T. W. (2009, May). Postnatal exposure to methylmercury from fish consumption: a review and new data from the Seychelles Child Development Study. *NeuroToxicology, 30*(3), 338-349. PMID 19442817. doi:10.1016/j.neuro.2009.01.005

Nagahawatte, N., & Goldenberg, R. (2008, June). Poverty, maternal health and adverse pregnancy outcomes. *Annals of New York Academy of Science, 1136*(1), 80-85. PMID 17954684. doi:10.1196/annals.1425.016

Nagy, J. (2000, March). Alcohol dependence at the cellular level: effects of ethanol on calcium homeostasis of IM-9 human lymphoblast cells. *Journal of Studies on Alcohol, 61*(2), 225-231. PMID 10757132

Naimi, T., Brewer, R., Mokdad, A., Denny, C., Serdula, M., & Marks, J. (2003, January 1). Binge Drinking Among U.S. Adults. *Journal of the American Medical Association, 289*(1), 70-75. PMID12503979

Nakagawa, M., Kodama, T., Akiba, S., Arimura, K., Wakamiya, J., Futatsuka, M., … Osame, M. (2002, January). Logistic Model Analysis of Neurological Findings in Minamata Disease and the Predicting Index. *Internal Medicine, 41*(1), 14-19. PMID 11838584

Nakai, K., & Satoh, H. (2002, February). Developmental Neurotoxicity Following Prenatal Exposures to Methylmercury and PCBs in Humans from Epidemiological Studies. *Tohoku Journal of Experimental Medicine, 196*(2), 89-98. PMID 12498320

Nastoff, T., Drew, D., Wigington, P., Wakefield, J., Phillips, J., & O'Fallon, L. (Eds.) (2002, August). *Final Report of Nursing and Environmental Health Roundtable*. Research Triangle Park, N.C.: Agency for Toxic Substances and Disease Registry

National Committee for Clinical Laboratory Standards. (1985). *Procedure for determining packed cell volume was by the micro hematocrit method (H7-A)*. Villanova, PA: Author

National Health Survey Act, Pub. L. No. 652, §510, 489 - 490 (1956, July 3)

National Research Council. (1983, March). *Risk Assessment in the Federal Government: Managing the Process*. Washington, DC: National Academy Press

National Research Council. (1986). *Nutrient adequacy: assessment using food consumption surveys*. Washington, DC: National Academy Press

National Research Council. (1991). *Environmental Epidemiology, Volume 1: Public Health and Hazardous Wastes*. Washington, DC: National Academy Press. Retrieved April 1, 2011 from http://books.nap.edu/openbook.php?record_id=1802

National Research Council. (2000a). *Scientific Frontiers in Developmental Toxicity and Risk Assessment*. Washington, DC: National Academy Press

National Research Council. (2000b). *Toxicological Effects of Methylmercury*. Washington, DC: National Academy Press

National Research Council. (2005). *Measuring Food Insecurity and Hunger: Phase I Report*. Washington, DC: The National Academies Press

National Research Council. (2006). *Human Biomonitoring for Environmental Chemicals*. Washington, DC: The National Academies Press

Navas-Acien, A., Selvin, E., Sharrett, R., Calderon-Aranda, E., Silbergeld, E., & Guallar, E. (2004, June 29). Lead, Cadmium, Smoking and Increased Risk of

Peripheral Arterial Disease. *Circulation, 109*(25), 3196-3201. PMID 15184277. doi:10.1161/01.CIR.0000130848.18636.B2

Navas-Acien, A., Silbergeld, E., Sharrett, R., Calderon-Aranda, E., Selvin, E., & Guallar, E. (2005, February). Metals in Urine and Peripheral Arterial Disease. *Environmental Health Perspectives, 113*(2), 164-169. PMID 15687053

Navratil, T., Skrivan, P., Vach, M., Dobesova, I., & Langrova, A. (2004, May 31). *The biogeochemical cycle of lead in an acidified forested catchment in central Czech Republic.* Retrieved April 1, 2011 from http://www.gli.cas.cz/lesnipotok/tommy/documents/Pb%20poster%20final.pdf

Nawrot, T. S., & Staessen, J. A. (2006, September 26). Low-Level Environmental Exposure to Lead Unmasked as Silent Killer. *Circulation, 114,* 1347-1349. PMID 17000919. doi:10.1161/CIRCULATIONAHA.106.650440

Nawrot, T. S., Staessen, J. A., Den Hond, E. M., Koppen, G., Schoeters, G., Fagard, R., … Roels, H. A. (2002, June). Host and Environmental Determinants of Polychlorinated Aromatic Hydrocarbons in Serum of Adolescents. *Environmental Health Perspectives, 110*(6), 583-589. PMID 12055049

Needham, L. L. (2005a, April). Assessing Exposure to Organophosphorus Pesticides by Biomonitoring in Epidemiologic Studies of Birth Outcomes. *Environmental Health Perspectives, 113*(4), 494-498. PMID 15811842

Needham, L. L., Barr, D. B., Caudill, S. P., Pirkle, J. L., Turner, W. E., Osterloh, J., … Sampson, E. J. (2005b, August). Concentrations of Environmental Chemicals Associated with Neurodevelopmental Effects in U.S. Population. *NeuroToxicology, 26*(4), 531-545. PMID 16112319. doi:10.1016/j.neuro.2004.09.005

Needham, L. L., Calafat, A. M., & Barr, D. B. (2008, February). Assessing Developmental Toxicant Exposures via Biomonitoring. *Basic & Clinical Pharmacology & Toxicology, 102*(2), 100-108. PMID 18226062

Needham, L. L., Ozkaynak, H., Whyatt, R. M., Barr, D. B., Wang, R. Y., … Zartarian, V. (2005c, August). Exposure Assessment in the National Children's Study:

Introduction. *Environmental Health Perspectives, 113*(8), 1076-1082. PMID 16079082

Needleman, H. L. (1973a, February). Lead Poisoning in Children: Neurologic Implications of Widespread Subclinical Intoxication. *Seminars in Psychiatry, 5*(1), 47-54. PMID 4807814

Needleman, H. L. (1980). Human Exposure: Difficulties and Strategies in the Assessment of Neuropsychological Impact. In R. L. Singhai & J. A. Thomas (Eds.), *Lead Toxicity*. (pp. 1-18.) Baltimore, MD: Urban & Schwarzenberg

Needleman, H. L. (1989, May). The Persistent Threat of Lead: A Singular Opportunity. *American Journal of Public Health, 79*(5), 643-645. PMID 2650573

Needleman, H. L. (2002a, July-August). What is not found in the spreadsheets. *NeuroToxicology and Teratology, 24*(4), 459-461. PMID 12127887

Needleman, H. L. (2006a, March). The Costs of Mercury Exposure. (Peer Commentary on the article "Mental retardation and prenatal methylmercury toxicity" by Trasande, Schecher, Haynes & Landrigan, 2006, March). *American Journal of Industrial Medicine, 49*(3), 221. PMID 16470546. doi:10.1002/ajim.20269

Needleman, H. L., & Bellinger, D. (1981a). The Epidemiology of Low-Level Lead Exposure in Childhood. *Journal of the American Academy of Child Psychiatry, 20*(3), 496-512. PMID 7310019

Needleman, H. L., & Bellinger, D. (1994). *Prenatal Exposure to Toxicants: Developmental Consequences*. Baltimore, MD: The Johns Hopkins University Press

Needleman, H. L., Bellinger, D., & Leviton, A. (1981b, December). Does Lead at Low Dose Affect Intelligence in Children? *Pediatrics, 68*(6), 894-896. PMID 7322729

Needleman, H. L., Davidson, I., Sewell, E. M., & Shapiro, I. M. (1974a, January 31). Subclinical Lead Exposure in Philadelphia Schoolchildren: Identification by Dentine Lead Analysis. *The New England Journal of Medicine, 290*(5), 245-248. PMID 4808928

Needleman, H. L., McFarland, C., Ness, R. B., Fienberg, S. E., & Tobin, M. J. (2002b, November-December). Bone lead levels in adjudicated delinquents: A case control study. *NeuroToxicology and Teratology, 24*(6), 711-717. PMID 12460653

Needleman, H. L., Rabinowitz, M., Leviton, A., Linn, S., & Schoenbaum, S. (1984, June 8). The Relationship Between Prenatal Exposure to Lead and Congenital Anomalies. *Journal of American Medical Association, 251*(22), 2956-2959. PMID 6716624

Needleman, H. L., Reiss, J. A., Tobin, M. J., Biesecker, G. E., & Greenhouse, J. B. (1996, February 7). Bone Lead Levels and Delinquent Behavior. *Journal of American Medical Association, 275*(5), 363-369. PMID 8569015

Needleman, H. L., Reiss, J. A., Tobin, M. J., & Biesecker, G. E. (1996, June 12). Bone Lead Levels and Delinquent Behavior. (Peer Commentary on article "Bone lead levels in adjudicated delinquents: A case control study." by Needleman, McFarland, Ness, Fienberg & Tobin, 2002b, November-December). *Journal of American Medical Association, 275*(22), 1728. PMID 8637163

Needleman, H. L., & Scanlon, J. (1973b, March 1). Getting the Lead Out. *The New England Journal of Medicine, 288*(9), 466-467. PMID 4683921

Needleman, H. L., Schell, A., Bellinger, D., Leviton, A., & Allred, E. N. (1990, January 11). The Long-Term Effects of Exposure to Low Doses of Lead in Childhood: An 11-Year Follow-Up Report. *The New England Journal of Medicine, 322*(2), 83-88. PMID 2294437

Needleman, H. L., & Shapiro, I. M. (1974b, May). Dentine Lead Levels in Asymptomatic Philadelphia School Children: Subclinical Exposure in High and Low Risk Groups. *Environmental Health Perspectives, 7*, 27-31. PMID 4831144

Needleman, H. L., Tuncay, O. C., & Shapiro, I. M. (1972, January 14). Lead Levels in Deciduous Teeth of Urban and Suburban American Children. *Nature, 235,* 111-112. PMID 4550400

Neel, J. V. (1962, December). Diabetes Mellitus: A "Thrifty" Genotype Rendered Detrimental by Progress? *American Journal of Human Genetics, 14*, 353-362. PMID 13937884

Neel, J. V. (1999, May). The "Thrifty" Genotype in 1998. *Nutrition Reviews, 57*(5, Part 2), S2-S9. PMID 10391020

Negy, C., Schwartz, S., & Reig-Ferrer, A. (2009, July). Violated Expectations and Acculturative Stress Among U.S. Hispanic Immigrants. *Cultural Diversity and Ethnic Minority Psychology, 15*(3), 225-264. PMID 19594254. doi:10.1037/a0015109

Nehls, G. & Ackland, G. (1973, March). Procedures for Handling Aerometric Data. *Journal of Air Pollution Control Association, 23*(3), 180-184

Nelson, M., Dick, K., & Holmes, B. (2002, November). Food budget standards and dietary adequacy in low-income families. *Proceedings of the Nutrition Society, 61*(4), 569-577. PMID 12691187

Neri, M., Bonassi, S., Knudsen, L. E., Šrám, R. J., Holland, N., Ugolini, D., & Merlo, D. F. (2006, January). Children's exposure to environmental pollutants and biomarkers of genetic damage. I. Overview and critical issues. *Mutation Research, 612*(1), 1-13. PMID 16002329. doi:10.1016/j.mrrev.2005.04.001

Neri, M., Ugolini, D., Bonassi, S., Fucic, A., Holland, N., Knudsen, L. E., … Merlo, D. F. (2006, January). Children's exposure to environmental pollutants and biomarkers of genetic damage. II. Results of a comprehensive literature search and meta-analysis. *Mutation Research, 612*(1), 14-39. PMID 16027031. doi:10.1016/j.mrrev.2005.04.003

Neufeld, A., & Harrison, M. J. (1994, October-December). Use of nursing diagnosis with population groups. *Nursing Diagnosis, 5*(4), 165-171. PMID 7826721

Neufer, L. (1994, June). The Role of the Community Health Nurse in Environmental Health. *Public Health Nursing, 11*(3), 155-162. PMID 8898554

Neufer, L., & Narkunas, D. (1994, July). Hazardous Substance Releases at the Community Level. *Association of American Occupational Health Nurses Journal, 42*(7), 329-335. PMID 8060397

Newbold, R., Padilla-Banks, E., & Jefferson, W. (2006, June). Adverse effects of the model environmental estrogen diethylstilbestrol are transmitted to subsequent generations. *Endocrinology, 147*(Supplement 6), S11-S17. PMID 16690809. doi:10.1210/en.2005-1164

Newland, M. C., & Paletz, E. M. (2000, December). Animal Studies of Methylmercury and PCBs: What do they tell us about expected effects in humans? *NeuroToxicology, 21*(6), 1003-1028. PMID 11233748

Newman, S. H., & Howell, M. A. (1953, December). Research Preferences and Activities of Public Health Service Officers. *Public Health Reports, 68*(12), 1183-1191. PMID 13121179

Ng, S. P., Conkline, D., Bhatnagar, A., Bolanowski, D., Lyon, J., & Zelikoff, J. (2009, July). Prenatal Exposure to Cigarette Smoke Induces Diet- and Sex-Dependent Dyslipidemia and Weight Gain in Adult Murine Offspring. *Environmental Health Perspectives, 117*(7), 1042-1048. PMID 19654910

Nicas, M., & Jayjock, M. (2002, May-June). Uncertainty in Exposure Estimates Made by Modeling Versus Monitoring. *American Industrial Hygiene Association Journal, 63*(3), 275-283. PMID 12173176

Nielsen-Menicucci, K. (2004). *Keeping safe: field public health nurses' perceptions* (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 3131498)

Nightingale, F. (1860). *Notes on Nursing* (1969 ed.). Mineola, NY: Dover Publications, Inc.

Ninomiya, T., Imamura, K., Kuwahata, M., Kindaichi, M., Susa, M., & Ekino, S. (2005, July-August). Reappraisal of somatosensory disorders in methylmercury poisoning. *NeuroToxicology and Teratology, 27*(4), 643-653. PMID 16087068. doi:10.1016/j.ntt.2005.03.008

Ninomiya, T., Ohmori, H., Hashimoto, K., Tsuruta, K., & Ekino, S. (1995, July). Expansion of Methylmercury Poisoning outside of Minamata: An Epidemiological Study on Chronic Methylmercury Poisoning outside of Minamata. *Environmental Research, 70*(1), 47-50. PMID 8603658. doi:10.1006/enrs.1995.1045

Nixon, D. E., Mussmann, G. V., & Moyer, T. P. (1996, January-February). Inorganic, organic and total mercury in blood and urine: cold vapor analysis with automated flow injection sample delivery. *Journal of Analytical Toxicology, 20*(1), 17-22. PMID 8837946

Noakes, P. S., Taylor, P., Wilkinson, S., & Prescott, S. L. (2006, May). The relationship between persistent organic pollutants in maternal and neonatal tissues and immune responses to allergens: A novel exploratory story. *Chemosphere, 63*(8), 1304-1311. PMID 16289241. doi:10.1016/j.chemosphere.2005.09.008

Norman, M. W. (2005). *Hearing sensitivity among Anniston Army Depot workers* (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 3187880)

Norström**,** K., Czub, G., McLachlan, M., Hu, D., Thorne, P., & Hornbuckle, K. (2010, November). External exposure and bioaccumulation of PCBs in humans living in a contaminated urban environment. *Environment International, 36*(8), 855-861. PMID 19394084. doi:10.1016/j.envint.2009.03.005

Northeast Waste Management Officials' Association (NEWMOA). (2001, October). *Reported Mercury Spills in the Northeast States*. Retrieved April 1, 2011 from http://www.newmoa.org/prevention/mercury/SpillReport.pdf

Northridge, M. E., & Shepard, P. M. (1997, May). Environmental racism and public health. *American Journal of Public Health, 87*(5), 730-732. PMID 9184496

Northridge, M. E., Stover, G., Rosenthal, J., & Sherard, D. (2003, February). Environmental Equity and Health: Understanding Complexity and Moving Forward. *American Journal of Public Health, 93*(2), 209-214. PMID 12554571

Novo, M., Hammarström, A., & Janlert, U. (2000, November). Smoking habits – a question of trend or unemployment? A comparison of young men and women between boom and recession. *Public Health*, *114*(6), 460-463. PMID 11114757. doi:10.1038/sj.ph.1900704

Nurminen, M., Nurminen, T., & Corvalan, C. F. (1999, September). Methodologic Issues in Epidemiologic Risk Assessment. *Epidemiology, 10*(5), 585-593. PMID 10468438

Nyamathi, A., Koniak-Griffin, D., & Greengold, B. A. (2007). Development of Nursing Theory and Science in Vulnerable Populations Research. *Annual Review of Nursing Research, 25*, 3-25. PMID 17958287

Odierna, D., & Schmidt, L. (2009, August). The effects of failing to include hard-to-reach respondents in longitudinal surveys. *American Journal of Public Health, 99*(8), 1515-1521. PMID 19008525. doi:10.2105/AJPH.2007.111138

Offit, P. A. (2007, September 27). Thimerosal and Vaccines – A Cautionary Tale (Perspective). *New England Journal of Medicine, 357*(13), 1278-1279. PMID 17898096. doi:10.1056/NEJMp078187

Ogata, M., & Oiwa, K. (2001). *Rowing the Eternal Sea: The Story of a Minamata Fisherman*. Lanham, MD: Rowman & Littlefield Publishers, Inc.

Ohba, T., Kurokawa, N., Nakai, K., Shimada, M., Suzuki, K., Sugawara, N., … Satoh, H. (2008, January). Permanent waving does not change mercury concentration in the proximal segment of hair close to scalp. *Tohoku Journal of Experimental Medicine, 214*(1), 69-78. PMID 18212489

Oken, E., Wright, R. O., Kleinman, K. P., Bellinger, D., Amarasiriwardena, C. J., Hu, H., … Gillman, M. W. (2005, October). Maternal Fish Consumption, Hair Mercury, and Infant Cognition in a U.S. Cohort. *Environmental Health Perspectives, 113*(10), 1376-1380. PMID 16203250

Oksanen, T., Kivimäki, M., Pentti, J., Virtanen, M., Klaukka, T., & Vahtera, J. (2010, July). Self-Report as an Indicator of Incident Disease. *Annals of Epidemiology, 20*(7), 547-554. PMID 20538198. doi:10.1016/j.annepidem.2010.03.017

Oliver, T. (1911, May 13). A lecture on lead poisoning and the race. *British Medical Journal, 1*(2628), 1096-1098

Olson, D. K., Lohman, W. H., Brosseau, L. M., Fredrickson, A. L., McGovern, P. M., Gerberich, S. G., & Nachreiner, N. M. (2005). Crosscutting Competencies for Occupational Health and Safety Professionals. *Journal of Public Health Management Practice, 11*(3), 235-243. PMID 15829837

Opler, M. G., Brown, A. S., Graziano, J., Desai, M., Zheng, W., Schaefer, C., … Susser, E. S. (2004, April). Prenatal Lead Exposure, δ-Aminolevulinic Acid, and Schizophrenia. *Environmental Health Perspectives, 112*(5), 548-552. PMID 15064159

Opler, M. G., Buka, S. L., Groeger, J., McKeague, I., Wei, C., Factor-Litvak, P., … Susser, E. S. (2008, November). Prenatal exposure to lead, δ-aminolevulinic acid, and schizophrenia: further evidence. *Environmental Health Perspectives, 116*(11), 1586-1590. PMID 19057716. doi:10.1289/ehp.10464

O'Riordon, T., & McMichael, A. (2002). Dealing with scientific uncertainties. In P. Martens, & A. J. McMichael (Eds.), *Environmental Change, Climate and Health* (pp. 311-332). Cambridge, UK: Cambridge University Press

O'Rourke, M. K., Van De Water, P. K., Jin, S., Rogan, S. P., Weiss, A. D., Gordon, S. M., … Lebowitz, M. D. (1999, September-October). Evaluations of primary metals from NHEXAS Arizona: distributions and preliminary exposures. *Journal of Exposure Analysis and Environmental Epidemiology, 9*(5), 435-445. PMID 10554146

Orsi, A. J., Grey, M., Mahon, M. M., Moriarty, H. J., Shepard, M. P., & Carroll, R. M. (1999, April). Conceptual and Technical Considerations when Combining Large Data Sets. *Western Journal of Nursing Research, 21*(2), 130-142. PMID 11512172

Ortega Garcia, J. A., Carrizo Gallardo, D., Ferris i Tortajada, J., Garcia, M. M., & Grimalt, J. O. (2006, August). Meconium and neurotoxicants: searching for a prenatal exposure timing. *Archives of Disease in Childhood, 91*(8), 642-646. PMID 16624883. doi:10.1136/adc.2005.084129

Ortiz-Roque, C., & Lopez-Rivera, Y. (2004, September). Mercury contamination in reproductive age women in a Caribbean island: Vieques. *Journal of epidemiology and community health, 58*(9), 756-757. PMID 15310801. doi:10.1136/jech.2003.019224

Ostrea, E. M. Jr., Villanueva-Uy, E., Bielawski, D. M., Posecion, N. C. Jr., Corrion, M. L., Jin, Y., … Ager, J. W. (2006, July). Maternal hair – An appropriate matrix for detecting maternal exposure to pesticides during pregnancy. *Environmental Research, 101*(3), 312-322. PMID 16584725. doi:10.1016/j.envres.2006.02.006

Otero-Sabogal, R., Sabogal, F., Paerez-Stable, E., & Hiatt, R. (1995). Dietary Practices, Alcohol Consumption and Smoking Behavior: Ethnic, Sex, and Acculturation Differences. *Journal of the National Cancer Institute, Monographs, 18*, 73-82. PMID 8562225

Ott, W. R. (1985, October). Total Human Exposure: An Emerging Science Focuses on Humans as Receptors of Environmental Pollution. *Environmental Science & Technology, 19*(10), 880-886. doi:10.1021/es00140a001

Ott, W. R. (2007). Exposure Analysis: A Receptor-Oriented Science. In W. R. Ott, A. C. Steinemann & L. A. Wallace (Eds.), *Exposure Analysis* (pp. 3-32). Boca Raton, FL: CRC Taylor & Francis Group

Ott, W. R., Wallace, L., Mage, D., Akland, G., Lewis, R., Sauls, H., … Morehouse, K. (1986). The Environmental Protection Agency's Research Program on Total Human Exposure. *Environment International, 12*, 475-494

Owen, C. V., Acquavella, J., Lynch, J., & Bird, M. (1992, September). An Industrial Hygiene Methodology Developed in Support of a Retrospective Morbidity Case-Control Study. *American Industrial Hygiene Association Journal, 53*(9), 540-547. PMID 1524029

Owen, R., Buxton, L., Sarkis, S., Toaspern, M., Knap, A., & Depledge, M. (2002, October). An evaluation of hemolymph cholinesterase activities in the tropical scallop, Euvola (Pecten) ziczac, for the rapid assessment of pesticide exposure. *Marine Pollution Bulletin, 44*(10), 1010-1017. PMID 12474960

Ozkalkanli, M. Y., Ozkalkanli, D. T., Katircioglu, K., & Savaci, S. (2009). Comparison of Tools for Nutrition Assessment and Screening for Predicting

the Development of Complications in Orthopedic Surgery. *Nutrition in Clinical Practice, 24*(2), 274-280. PMID 19321901. doi:10.1177/0884533609332087

Ozuah, P. O. (2000, March). Mercury Poisoning. *Current Problems in Pediatrics, 30*(3), 91-99. PMID 10742922

Packard, M. (2004). Unfolding the blanket of understanding in the listening space: a phenomenological exploration of "being-with"in the nursing student-teacher relationship (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 3125440)

Padhi, B., Pelletier, G., Williams, A., Berndt-Weis, L., Yauk, C., Bowers, W., & Chu, I. (2008, January 30). Gene expression profiling in rat cerebellum following *in utero* and lactational exposure to mixtures of methylmercury, polychlorinated biphenyls and organochlorine pesticides. *Toxicology Letters, 176*(2), 93-103. PMID 18077114. doi:10.1016/j.toxlet.2007.08.016

Pagana, K. D., & Pagana, T. J. (2008). *Mosby's Diagnostic and Laboratory Test Reference*, 9th edition. St. Louis, MO: Mosby Elsevier

Page, R. L. (2006, December). Acculturation in Mexican Immigrants: A Concept Analysis. *Journal of Holistic Nursing, 24*(4), 270-278. PMID 17098881. doi:10.1177/0898010106289839

Page, W. F., & Kuntz, A. J. (1980, March). Racial and Socioeconomic Factors in Cancer Survival. *Cancer,* 1029-1040. PMID 7020913

Palmer, P., Wilson, L., Casey, A., & Wagner, R. (2011, April). Occurrence of PCBs in raw and finished drinking water at seven public water systems along the Hudson River. *Environmental Monitoring and Assessment, 175*(1-4), 487-499. PMID 20556645.

Palumbo, D. R., Cox, C., Davidson, P. W., Myers, G. J., Choi, A., Shamlaye, C., … Clarkson, T. W. (2000, October). Association between Prenatal Exposure to Methylmercury and Cognitive Functioning in Seychellois Children: A Reanalysis of the McCarthy Scales of Children's Ability from the Main Cohort

Study. *Environmental Research, 84*(Section A 2), 81-88. PMID 11068921. doi:10.1006/enrs.2000.4095

Pang, Y., Macintosh, D. L., Camann, D. E., & Ryan, P. B. (2002, March). Analysis of Aggregate Exposure to Chlorpyrifos in the NHEXAS: Maryland Investigation. *Environmental Health Perspectives, 110*(3), 235-240. PMID 11882473

Paranzino, G. K., Butterfield, P., Nastoff, T., & Ranger, C. (2005, January). I PREPARE: development and clinical utility of an environmental exposure history mnemonic. *Association of American Occupational Health Nurses Journal, 53*(1), 37-42. PMID 15675156

Park, H.-Y., Hertz-Picciotto, I., Sovcikova, E., Kocan, A., Drobna, B., & Trnovec, T. (2010, August 23). Neurodevelopmental toxicity of prenatal polychlorinated biphenyls by chemical structure and activity: a birth cohort study. *Environmental Health, 9*, 51. PMID 20731829. doi: 10.1186/1476-069X-9-51

Park, J. (2005). *Assessment of suspended dust from pipe rattling operations* (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 3231570)

Park, J., Linderholm, L., Charles, M. J., Athanasiadou, M., Petrik, J., Kocan, A., … Hertz-Picciotto, I. (2007, January). Polychlorinated Biphenyls and Their Hydroxylated Metabolites (OH-PCBs) in Pregnant Women from Eastern Slovakia. *Environmental Health Perspectives, 115*(1), 20-27. PMID 17366814

Parker, J., Kravets, N., & Woodruff, T. (2008a, February). Linkage of the National Health Interview Survey to Air Quality Data. *Vital and Health Statistics, 2*(145). PMID 18351156

Parkes, M., Panelli, R., & Weinstein, P. (2003, May). Converging Paradigms for Environmental Health Theory and Practice. *Environmental Health Perspectives, 111*(5), 669-75. PMID 12727592

Parkinson, A., & Safe, S. (1987). Mammalian Biologic and Toxic Effects of PCBs. In Safe, S. (Ed.) *Polychlorinated Biphenyls: Mammalian and Environmental Toxicology*, (pp. 49-76). New York, NY: Springer-Verlag

Parse, R. R. (1981). *Man-Living-Health: A Theory of Nursing*. New York, NY: John Wiley & Sons

Parsons, P. J., Chisolm, J. J. Jr., Delves, H. T., Griffin, R., Gunters, E. W., Slavin, W., … Vocke, R. (1993). *Analytical Procedures for the Determination of Lead in Blood and Urine; Approved Guideline* C40-A. 21(9). Wayne, PA: Clinical and Laboratory Standards Institute

Parsons, P. J. & Slavin, W. A. (1993, May-June). A rapid Zeeman graphite furnace atomic-absorption spectrometric method for the determination of lead in blood. *Spectrochimica Acta, Part B: Atomic Spectroscopy, 48*(6-7), 925-939. doi:10.1016/0584-8547(93)80094-B

Passos, C. J., Mergler, D., Gaspar, E., Morais, S., Lucotte, M., Larribe, F., ... de Grosbois, S. (2003, October). Eating tropical fruit reduces mercury exposure from fish consumption in the Brazilian Amazon. *Environmental Research, 93*(2), 123-130. PMID 12963396

Pastor, M., Sadd, J., & Hipp, J. (2001). Which came first? Toxic Facilities, Minority Move-In and Environmental Justice. *Journal of Urban Affairs, 23*(1), 1-21

Patandin, S., Dagnelie, P. C., Mulder, P. G., Op de Coul, E., van der Veen, J. E., Weisglas-Kuperus, N., & Sauer, P. J. (1999a, January). Dietary Exposure to Polychlorinated Biphenyls and Dioxins from Infancy until Adulthood: A Comparison between Breastfeeding, Toddler and Long-Term Exposure. *Environmental Health Perspectives, 107*(1), 45-51. PMID 9872716

Patandin, S., Lanting, C., Mulder, P., Boersma, E. R., Sauer, P., & Weisglas-Kuperus, N. (1999b, January). Effects of environmental exposure to polychlorinated biphenyls and dioxins on cognitive abilities in Dutch children at 42 months of age. *Journal of Pediatrics, 134*(1), 33-41. PMID 9880446

Pate, R. R., Pratt, M., Blair, S. N., Haskell, W. L., Macera, C. A., Bouchard, C., … Wilmore, J. H. (1995, February 1). Physical activity and public health. A recommendation from the Centers for Disease Control and Prevention and the American College of Sports Medicine. *Journal of the American Medical Association, 273*(5), 402-407. PMID 7823386

Patterson, D. G. Jr., Todd, G. D., Turner, W. E., Maggio, V., Alexander, L. R., & Needham, L. L. (1994). Levels of Non-*ortho*-Substituted (Coplanar), Mono- and Di-*ortho*-Substituted Polychlorinated Biphenyls, Dibenzo-*p*-Dioxins and Dibenzofurans in Human Serum and Adipose Tissue. *Environmental Health Perspectives, 102*(Supplement 1), 195-204

Paustenbach, D., & Galbraith, D. (2006a, April). Biomonitoring: Is body burden relevant to public health? *Regulatory Toxicology and Pharmacology, 44*(3), 249-261. PMID 16473444. doi:10.1016/j.yrtph.2006.01.005

Paustenbach, D., & Galbraith, D. (2006b, August). Biomonitoring and Biomarkers: Exposure Assessment Will Never Be the Same. *Environmental Health Perspectives, 114*(8), 1143-1149. PMID 16882516

Payne-Sturges, D., & Gee, G. C. (2006). National environmental health measures for minority and low-income populations: tracking social disparities in environmental health. *Environmental Research, 102*, 154-171. PMID 16875687. doi:10.1016/j.envres.2006.05.014

Peake, M., & Whiting, M. (2006, November). Measurement of serum creatinine - current status and future goals. *Clinical Biochemistry Reviews, 27*(4),173-184. PMID 17581641

Pearce, N. (1996, May). Traditional Epidemiology, Modern Epidemiology and Public Health. *American Journal of Public Health, 86*(5), 678-683. PMID 8629719

Pearce, N. (2004, July). Effect Measures in Prevalence Studies. *Environmental Health Perspectives, 112*(10), 1047-1050. PMID 15238274

Peek, M. K., Cutchin, M. P., Salinas, J. J., Sheffield, K. M., Eschbach, K., Stowe, R. P., & Goodwin, J.S. (2010, May). Allostatic Load Among Non-Hispanic Whites, Non-Hispanic Blacks and People of Mexican Origin: Effects of Ethnicity, Nativity and Acculturation. *American Journal of Public Health*, *100*(5), 940-946. PMID 19834005. doi:10.2105/AJPH.2007.129312

Peeples, E. S., Schopfer, L. M., Duysen, E. G., Spaulding, R., Voelker, T., Thompson, C. M., & Lockridge, O. (2005, February). Albumin, a new biomarker of organophosphorus toxicant exposure, identified by mass spectrometry.

Toxicological Sciences, 83(2), 303-312. PMID 15525694. doi:10.1093/toxsci/kfi023

Pennington, D., Margni, M., Ammann, C., & Joliet, O. (2005, February 15). Multimedia Fate and Human Intake Modeling: Spatial versus Nonspatial Insights for Chemical Emissions in Western Europe. *Environmental Science & Technology, 39*(4), 1119-1128. PMID 15773485

Pereg, D., Lagueux, J., DeWailly, E., Poirier, G. G., & Ayotte, P. (2001, March). Cigarette smoking during pregnancy: comparison of biomarkers for inclusion in epidemiological studies. *Biomarkers, 6*(2), 161-173. AN 4318695

Perera, F. P., Rauh, V., Tsai, W.-Y., Kinney, P., Camann, D., Barr, D., … Whyatt, R. M. (2003, February). Effects of Transplacental Exposure to Environmental Pollutants on Birth Outcomes in a Multiethnic Population. *Environmental Health Perspectives, 111*(2), 201-205. PMID 12573906

Perera, F. P., Rauh, V., Whyatt, R. M., Tang, D., Tsai, W. Y., Bernert, J. T., … Kinney, P. L. (2005, August). A Summary of Recent Findings on Birth Outcomes and Developmental Effects of Prenatal ETS, PAH, and Pesticide Exposures. *NeuroToxicology, 26*(4), 573-587. PMID 16112323. doi:10.1016/j.neuro.2004.07.007

Pereira, F., Yassuda, M., Oliveira, A., Diniz, B., Radanovic, M., Talib, L., … Forlenza, O. (2010). Profiles of functional deficits in mild cognitive impairment and dementia: benefits from objective measurement. *Journal of the International Neuropsychological Society, 16*, 297-305. doi:10.1017/S1355617709991330

Perles Rosello, M., Vias Martinez, J., & Andreo Navarro, B. (2009, February). Vulnerability of human environment to risk: case of groundwater contamination risk. *Environment International, 35*(2), 325-335. PMID 18845340. doi:10.1016/j.envint.2008.08.005

Perlin, S., Wong, D., & Sexton, K. (2001, March). Residential proximity to industrial sources of air pollution: interrelationships among race, poverty, and age. *Journal of the Air & Waste Management Association, 51*(3), 406-421. PMID 11266104

Pessah, I., & Wong, P. (2001). Etiology of PCB Neurotoxicity: From Molecules to Cellular Dysfunction. In L. W. Robertson & L. G. Hansen (Eds.), *PCBs: Recent Advances in Environmental Toxicology and Health Effects* (pp. 179-184). Lexington, KY: The University Press of Kentucky

Peterson, B., Youngren, S., & Walls, C. (2001). Modeling Dietary Exposure with Special Sections on Modeling Aggregate and Cumulative Exposures. In R. I. Kreiger (Ed.), *Handbook of Pesticide Toxicology* (2nd ed., Vol. I, pp. 443-455). San Diego, CA: Academic Press

Petkeviciene, J., Simila, M., Becker, W., Kriaucioniene, V., & Valsta, L. M. (2009, January). Validity and reproducibility of the NORBAGREEN food frequency questionnaire. *European Journal of Clinical Nutrition, 63*(1), 141-149. PMID 17805226. http://dx.doi.org/10.1038/sj.ejcn.1602893

Phelps, J. (2004, June). MeHg/PCB Combination Impairs Motor Skills in Young Rats. *Environmental Health Perspectives, 112*(8), A471

Phelps, J. (2007, May). Headliners in Molecular Biology: Lead, Paraquat, and Methylmercury Disrupt Neuronal Stem Cells by a Common Mechanism. *Environmental Health Perspectives, 115*(5), A248

Phillips, D. L., Pirkle, J. L., Burse, V. W., Bernert, J. T. Jr., Henderson, L. O., & Needham, L. L. (1989, July-August). Chlorinated hydrocarbon levels in human serum: effects of fasting and feeding. *Archives of Environmental Contaminant Toxicology, 18*(4), 495-500. PMID 2505694

Piccardo, M. T., Stella, A., & Valerio, F. (2010, January 29). Is the smoker's exposure to environmental tobacco smoke negligible? *Environmental Health, 9*, 5. PMID 20113464. doi:10.1186/1476-069X-9-5

Pickett, K., & Pearl, M. (2001, February). Multilevel analyses of neighbourhood socioeconomic context and health outcomes: a critical review. *Journal of Epidemiology and Community Health, 55*(2), 111-122. PMID 11154250

Picton, T. W., Taylor, M. J., & Durieux-Smith, A. (1992). Brainstem Auditory Evoked Potentials in Pediatrics. In M. J. Aminoff (Ed.), *Electrodiagnosis in Clinical Neurology* (3rd ed., pp. 537-569). New York, NY: Churchill Livingstone

Piedrafita, B., Erceg, S., Cauli, O., & Felipo, V. (2008, July). Developmental exposure to polychlorinated biphenyls or methylmercury, but not to its combination, impairs the glutamate-nitric oxide-cyclic GMP pathway and learning in 3-month-old rats. *Neuroscience, 154*(4), 1408-1416. PMID 18556134. doi:10.1016/j.neuroscience.2008.05.013

Piikivi, L., & Ruokonen, A. (1989, May/June). Renal Function and Long-Term Low Mercury Vapor Exposure. *Archives of Environmental Health, 44*(3), 146-149. PMID 2787621

Pilsner, J. R., Hu, H., Ettinger, A., Sanchez, B. N., Wright, R. O., Cantonwine, D., … Hernández-Avila, M. (2009, September). Influence of Prenatal Lead Exposure on Genomic Methylation of Cord Blood DNA. *Environmental Health Perspectives, 117*(9), 1466-1471. PMID 19750115. doi:10.1289/ehp.0800497

Piotrowski, J. K., Trojanowska, B., & Mogilnicka, E. M. (1975, September 19). Excretion Kinetics and Variability of Urinary Mercury in Workers Exposed to Mercury Vapor. *International Archives of Occupational and Environmental Health, 35*(3-4), 245-256. PMID 1236835

Pirkle, J. L., Needham, L. L., & Sexton, K. (1995, July-September). Improving Exposure Assessment by Monitoring Human Tissues for Toxic Chemicals. *Journal of Exposure Analysis and Environmental Epidemiology, 5*(3), 405-424. PMID 8814778

Pirkle, J. L., Osterloh, J., Needham, L. L., & Sampson, E. J. (2005). National exposure measurements for decisions to protect public health from environmental exposures. *International Journal of Hygiene and Environmental Health, 208*(1-2), 1-5. PMID 15881972

Pizzol, M., Thomsen, M., & Skou Andersen, M. (2010, October 15). Long-term human exposure to lead from different media and intake pathways. *Science of the Total Environment, 408*(22), 5478-5488. PMID 20797773. doi:10.1016/j.scitotenv.2010.07.077

Pless, R., & Risher, J. F. (2000, May). Mercury, infant neurodevelopment and vaccination. *The Journal of Pediatrics, 136*(5), 571-573. PMID 10802484. doi:10.1067/mpd.2000.106797

Pless-Mulloli, T., Edwards, R., Howel, D., Wood, R., Paepke, O., & Herrmann, T. (2005, December). Does long term residency near industry have an impact on the body burden of polychlorinated dibenzo-p-dioxins, furans, and polychlorinated biphenyls in older women? *Occupational and Environmental Medicine, 62*(12), 895-901. PMID 16299100. doi:10.1136/oem.2004.018754

Plunkett, L. M. (2007, March 10). Developmental neurotoxicity of industrial chemicals. *The Lancet, 369*(9564), 821-822. PMID 17350441. doi:10.1016/S0140-6736(07)60396-1

Plusquellec, P., Muckle, G., Dewailly, E., Ayotte, P., Bégin, G., Desrosiers, C., … Poitras, K. (2010, January). The relation of environmental contaminants exposure to behavioral indicators in Inuit preschoolers in Arctic Quebec. *NeuroToxicology, 31*(1), 17-25. PMID 19854214. doi:10.1016/j.neuro.2009.10.008

Pohl, H. R., Roney, N., Wilbur, S., Hansen, H., & De Rosa, C. T. (2003, October). Six interaction profiles for simple mixtures. *Chemosphere, 53*(2), 183-197. PMID 12892681. doi:10.1016/S0045-6535(03)00436-3

Poissant, L., Zhang, H. H., Canario, J., & Constant, P. (2008, August). Critical review of mercury fates and contamination in the Arctic tundra ecosystem. *Science and the Total Environment, 400*(1-3), 173-211. PMID 18707754. doi:10.1016/j.scitotenv.2008.06.050

Polednak, A. P. (1989). *Racial and Ethnic Differences in Disease*. New York NY: Oxford University Press

Polk, L., & Green, P. (2007, April-June). Contamination: Nursing Diagnoses with Outcome and Intervention Linkages. *International Journal of Nursing Terminologies and Classifications, 18*(2), 37-44. PMID 17542859. doi:10.1111/j.1744-618X.2007.00048.x

Pollack, C. D. (1999, October-December). Methodological considerations with secondary analysis. *Outcomes Management for Nursing Practice, 3*(4), 147-152. PMID 10876539

Pollack, C. E., von dem Knesebeck, O., & Siegrist, J. (2004, March). Housing and Health in Germany. *Journal of Epidemiology and Community Health, 58*(3), 216-222. PMID 14966234. doi:10.1136/jech.2003.012781

Poole, C., & Rothman, K. J. (1998, October). Our conscientious objection to the epidemiology wars. (Peer Commentary on the article "Does risk factor epidemiology put epidemiology at risk? Peering into the future" by Susser, 1998, October). *Journal of Epidemiology and Community Health, 52*(10), 613-614. PMID 10023453

Pope, C. (2001). The Influence of Age on Pesticide Toxicity. In R. I. Kreiger (Ed.), *Handbook of Pesticide Toxicology* (2nd ed., Vol. I, pp. 873-885). San Diego, CA.: Academic Press

Porta, M. (2006, August 12). Persistent organic pollutants and the burden of diabetes. *The Lancet (Br.), 368*(9535), 558-559. PMID 16905002. doi:10.1016/S0140-6736(06)69174-5

Porterfield, S. P., & Hendrich, C. E. (1993, February). The Role of Thyroid Hormones in Prenatal and Neonatal Neurological Development – Current Perspectives. *Endocrine Reviews, 14*(1), 94-106. PMID 8491157

Prasada-Rao, P., Jordan, S., & Bhatnagar, M. (1989). Combined nephrotoxicity of methylmercury, lead, and cadmium in Pekin ducks: metallothionein, metal interactions, and histopathology. *Toxicology and Environmental Health, 26*(3), 327-348. PMID 2926832

Prendergast, M., Terry, A., & Buccafusco, J. (1998). Effects of Chronic, Low-Level Organophosphate Exposure on Delayed Recall, Discrimination and Spatial Learning in Monkeys and Rats. *NeuroToxicology and Teratology, 20*(2), 115-122. PMID 9536457

Price, P. S., & Chaisson, C. F. (2005). A conceptual framework for modeling aggregate and cumulative exposures to chemicals. *Journal of Exposure*

Analysis and Environmental Epidemiology, 15, 473-481. PMID 15856075. doi:10.1038/sj.jea.7500425

Price, P. S., Chaisson, C. F., Koontz, M., Wilkes, C., Ryan, B., Macintosh, D., & Georgopoulos, P. (2003a, June 20). *Construction of a Comprehensive Chemical Exposure Framework Using Person-Oriented Modeling*. Annandale, VA: The LifeLine Group

Price, P. S., Conolly, R. B., Chaisson, C. F., Gross, E. A., Young, J. S., Mathis, E. T., & Tedder, D. R. (2003b). Modeling Inter-individual Variation in Physiological Factors Used in PBPK Models of Humans. *Critical Reviews in Toxicology, 33*(5), 469-503. PMID 14594104

Priftis, K., Mantzouranis, E., & Anthracopoulos, M. (2009, April). Asthma symptoms and airway narrowing in children growing up in an urban versus rural environment. *Journal of Asthma, 46*(3), 244-251. PMID 19373631. doi:10.1080/02770900802647516

Primbs, T., Simonich, S., Schmedding, D., Wilson, G., Jaffe, D., Takami, A., … Kajii, Y. (2007, May 15). Atmospheric Outflow of Anthropogenic Semi-Volatile Organic Compounds from East Asia in Spring 2004. *Environmental Science and Technology, 41*(10), 3551-3558. PMID 17547177

Protection of Human Subjects, Additional Protections for Children Involved as Subjects in Research, 48 Fed. Reg. §9818 (1983, March 8)

Protection of Human Subjects, Additional Protections for Pregnant Women, Human Fetuses and Neonates Involved in Research, 66 Fed. Reg. §56778 (2001, November 13)

Protection of Human Subjects, Additional Protections Pertaining to Biomedical and Behavioral Research Involving Prisoners as Subjects, 43 Fed. Reg. §53655 (1978, November 16)

Protection of Human Subjects, Basic Health and Human Services Policy for Protection of Human Research Subjects, 56 Fed. Reg. §28012, §28022 (1991, June 18)

Pruss-Ustun, A., Bonjour, S., & Corvalan, C. (2008). The impact of the environment on health by country: a meta-synthesis. *Environmental Health, 7*(7), 1-25. PMID 18298819. doi:10.1186/1476-069X-7-7

Pryor, J. L., Hughes, C., Foster, W., Hales, B. F., & Robaire, B. (2000, June). Critical Windows of Exposure for Children's Health: The Reproductive System in Animals and Humans. *Environmental Health Perspectives, 108*(Supplement 3), 491-503. PMID 10852850

Pugh, R. N., Murray-Lyon, I. M., Dawson, J. L., Pietroni, M. C., & Williams, R. (1973, August). Transection of the oesophagus for bleeding oesophageal varices. *The British Journal of Surgery, 60*(8), 646-649. PMID 4541913

Qazi, Q. H., Madahar, C., & Yuceoglu, A. M. (1980, February). Temporary Increase in Chromosome Breakage in an Infant Prenatally Exposed to Lead. *Human Genetics, 53*(2), 201-203. PMID 6766900

Quandt, S. A., Jones, B. T., Talton, J. W., Whalley, L. E., Galván, L., Vallejos,. Q. M., … Arcury, T. A. (2010, January). Heavy metals exposures among Mexican farmworkers in eastern North Carolina. *Environmental Research, 110*(1), 83-88. PMID 19818439. doi:10.1016/j.envres.2009.09.007

Qin, Y. Y., Leung, C. K., Leung, A. O., Wu, S. C., Zheng, J. S., & Wong, M. H. (2010, January). Persistent organic pollutants and heavy metals in adipose tissues of patients with uterine leiomyomas and the association of these pollutants with seafood diet, BMI and age. *Environmental Science and Pollution Research International*, *17*(1), 229-240. doi:10.1007/s11356-009-0251-0

Quinn, L. (2004, October). *Assumptions and Limitations of the Census Bureau Methodology Ranking Racial and Ethnic Residential Segregation in Cities and Metro Area*. Retrieved, April 1, 2011 from http://www4.uwm.edu/eti/integration/QuinnCensus.pdf

Quinn, M. M. (2002, July-August). Exposure assessment in epidemiology and practice: Mind the Gap! *AIHA Journal, 63*(4), 384-389. PMID 12486771

Radimer, K. L., Olson, C. M., & Campbell, C. C. (1990, November). Development of indicators to assess hunger. *The Journal of Nutrition, 120*(Supplement 11), 1544-1548. PMID 2243303

Radio, N., Freudenrich, T., Robinette, B., Crofton, K., & Mundy, W. (2010, January-February). Comparison of PC12 and cerebellar granule cell cultures for evaluating neurite outgrowth using high content analysis. *Neurotoxicology and Teratology, 3*2(1), 25-35. PMID 19559085. doi:10.1016/j.ntt.2009.06.003

Ragas, A. M., Brouwer, F. P., Buchner, F. L., Hendriks, H. W., & Huijbregts, M. A. (2009, February). Separation of uncertainty and inter-individual variability in human exposure modeling. *Journal of Exposure Science & Environmental Epidemiology, 19*(2), 201-212. PMID 18398446. doi:10.1038/jes.2008.13

Rajanna, B., Rajanna, S., Hall, E., & Yallapragada, P. (1997, February-May). *In vitro* metal inhibition of N-methyl-D-aspartate specific glutamate receptor binding in neonatal and adult rat brain. *Drug and Chemical Toxicology, 20*(1-2), 21-29. PMID 9183560. doi:10.3109/01480549709011076

Ralston, N., Ralston, C., Blackwell, J., & Raymond, L. (2008, September). Dietary and tissue selenium in relation to methylmercury toxicity. *NeuroToxicology, 29*(5), 802-811. PMID 18761370. doi:10.1016/j.neuro.2008.07.007

Ramirez, G. B., Cruz, C. V., Pagulayan, O., Ostrea, E., & Dalisay, C. (2000, October). The Tagum Study I: Analysis and Clinical Correlates of Mercury in Maternal and Cord Blood, Breast Milk, Meconium, and Infants' Hair. *Pediatrics, 106*(4), 774-781. PMID 11015522

Ramirez, G. B., Pagulayan, O., Akagi, H., Rivera, A. F., Lee, L. V., Berroya, A., … Casintahan, D. (2003, March). Tagum Study II: Follow-Up Study at Two Years of Age After Prenatal Exposure to Mercury. *Pediatrics, 111*(3), e289-295. PMID 12612286

Ramsey, W. N. (1957, June). The determination of the total iron-binding capacity of serum. *Clinica Chimica Acta, 2*(3), 221-226. PMID 13461309

Randall, B., Tedrow, M. P., & Van Landingham, J. (1984). *Adaptation Nursing: The Roy Conceptual Model Applied*. St. Louis, MO: C. V. Mosby, Inc.

Rastogi, S., Clausen, J., & Srivastava, K. (1976). Selenium and Lead: Mutual Detoxifying Effects. *Toxicology, 6*, 377-388. PMID 996880

Rastogi, S., Nandlike, K., & Fenster, W. (2007). Elevated blood lead levels in pregnant women: identification of a high-risk population and interventions. *Journal of Perinatal Medicine, 35*(6), 492-496. PMID 18052836. doi:10.1515/JPM.2007.131

Rauh, V. A., Garfinkel, R., Perera, F. P., Andrews, H. F., Hoepner, L., Barr, D. B., … Whyatt, R. W. (2006, December). Impact of Prenatal Chlorpyrifos Exposure on Neurodevelopment in the First 3 Years of Life Among Inner-City Children. *Pediatrics, 118*(6), 1845-1859. PMID 17116700. doi:10.1542/peds.2006-0338

Rauh, V. A., Garfinkel, R., Perera, F. P., Andrews, H. F., Hoepner, L., & Barr, D. B. (2007, July). In Reply. (Peer Commentary on the article "Prenatal Chlorpyrifos and Early Neurodevelopment: How Good is the Science?" by Cicchetti, 2007, July.) *Pediatrics, 120*(1), 343-344. PMID 17116700

Ray, S. (2007). *Family transition experiences as perceived by caregivers of young children with spina bifida* (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 3281254)

Redding, L. E., Sohn, M. D., McKone, T. E., Chen, J.-W., Wang, S.-L., Hsieh, D. P.-H., & Yang, R. S.-H. (2008, December). Population Physiologically-Based Pharmacokinetic Modeling for the Human Lactational Transfer of PCB-153 with Consideration of Worldwide Human Biomonitoring Results. *Environmental Health Perspectives, 116*(12), 1629-1634. PMID 19079712. doi:10.1289/ehp.11519

Reed, J. (1992, July). Secondary data in nursing research. *Journal of Advanced Nursing, 17*(7), 877-883. PMID 1644985

Rees, D. C., Francis, E. Z., & Kimmel, C. A. (1990, May-June). Scientific and regulatory issues relevant to assessing risk for developmental neurotoxicity: an overview. *NeuroToxicology and Teratology, 12*(3), 175-181. PMID 2196418

Reif, A., Jacob, C. P., Rujescu, D., Herterich, S., Lang, S. Gutknecht, L., … Lesch, K. P. (2009, January). Influence of Functional Variant of Neuronal Nitric Oxide Synthase on Impulsive Behaviors in Humans. *Archives of General Psychiatry, 66*(1), 41-50. PMID 19124687. doi:10.1001/archgenpsychiatry.2008.510

Reif, J. S., Tsongas, T. A., Anger, W. K., Mitchell, J., Metzger, L., Keefe, T. J., … Amler, R. (1993, October-November). Two-Stage Evaluation of Exposure to Mercury and Biomarkers of Neurotoxicity at a Hazardous Waste Site. *Journal of Toxicology and Environmental Health, 40*(2-3), 413-422. PMID 8230312

Reilly, W. K. (1992, March-April). Environmental equity: EPA's position. *EPA Journal, 18*(1), 18-22. AN 9609101486

Reindl-Benjamins, M., Hummer, R., Eberstein, I. & Nam, C. (2004). Self-reported health and adult mortality risk: an analysis of cause-specific mortality. *Social Sciences & Medicine, 59*, 1297-1306. PMID 15210100. doi:10.1016/j.socscimed.2003.01.001

Reingold, A. (1998, January-March). Outbreak Investigations: A Perspective. *Emerging Infectious Diseases, 4*(1), 21-28. PMID 9452395

Reintjes, R., & Krumkamp, R. (2005, December). Methods of infectious disease epidemiology for public health services: case control studies as bridge between practical work, surveillance and epidemiology. *Gesundheitswesen, 67*(12), 840-844. PMID 16379045

René, A. A., Daniels, D. E., & Martin, S.A. Jr. (2000, June). Impact of environmental inequity on health outcome: where is the epidemiological evidence? *Journal of the National Medical Association, 92*(6), 275-280. PMID 10918762

Renner, R. (2010, February). Exposure on Tap: Drinking Water as an Overlooked Source of Lead. *Environmental Health Perspectives, 118*(2), A69-A74

Repetti, R., Matthews, K., & Waldron, I. (1989, November). Employment and Women's Health: Effects of Paid Employment on Women's Mental and Physical Health. *The American Psychologist, 44*(11), 1394-1401

Restricting Access to Select Agents and Toxins, 7 C. F. R. §331.10 (2009, December 2)

Restricting Access to Select Agents and Toxins, 9 C. F. R. §121.3-5 (2008, October 16)

Reynolds, R. M., Godfrey, K. M., Barker, M., Osmond, C., & Phillips, D. I. (2007, June). Stress Responsiveness in Adult Life: Influence of Mother's Diet in Late Pregnancy. *The Journal of Clinical Endocrinology & Metabolism, 92*(6), 2208-2210. PMID 17341553. doi:10.1210/jc.2007-0071

Rhode Island Department of Health, (2005, January). *Rules and Regulations for Lead Poisoning Prevention R23-24.6-PB*. Providence, R.I.: Department of Health

Rhode Island Department of Health, (2006, May). *A Healthier Rhode Island by 2010: Mid-Course Review*. Providence, RI: Author

Ricci, P. F., Rice, D., Ziagos, J., & Cox, L. A. Jr. (2003). Precaution, uncertainty and causation in environmental decisions. *Environment International, 29,* 1-19

Rice, D. (1995, December). Neurotoxicity of lead, methylmercury and PCBs in relation to the Great Lakes. *Environmental Health Perspectives, 103*(Supplement 9), 71-87. PMID 8635443

Rice, D. (2005, August). Assessing the Effects of Environmental Toxicant Exposure in Developmental Epidemiological Studies: Issues for Risk Assessment. *NeuroToxicology, 26*(4), 483-489. PMID 16112316. doi:10.1016/j.neuro.2004.12.009

Rice, D., & Barone, S. (2000, June). Critical Periods of Vulnerability for the Developing Nervous System: Evidence from Humans and Animal Models. *Environmental Health Perspectives, 108*(Supplement 3), S511-S533. PMID 10852851

Richardson, R. J. (1995). Assessment of the Neurotoxic Potential of Chlorpyrifos Relative to Other Organophosphorus Compounds: A Critical Review of the

Literature. *Journal of Toxicology and Environmental Health, 44*, 135-165. PMID 7531775

Richardson, R. J., & Miller, G. W. (2005). Toxicology. In H. Frumkin (Ed.), *Environmental Health from global to local* (pp. 24-45). San Francisco, CA: Jossey-Bass

Richmond, K. (2006). Being whole: aligning personhoods to achieve successful childbirth with a history of childhood sexual abuse during prenatal services (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 3218210)

Riederer, A. M., Pearson, M. A., & Lu, C. (2010, November). Comparison of food consumption frequencies among NHANES and CPES children: Implications for dietary pesticide exposure and risk assessment. *Journal of Exposure Science and Environmental Epidemiology, 20*(7), 602-614 . PMID 19738638. doi:10.1038/jes.2009.48

Rietjens, I. M., & Alink, G. M. (2006, August). Future of Toxicology: Low-Dose Toxicology and Risk-Benefit Analysis. *Chemical Research in Toxicology, 19*(8), 977-981. PMID 16918235. doi:10.1021/tx0601051

Rigas, M. L., Okino, M. S., & Quackenboss, J. J. (2001, June). Use of a Pharmacokinetic Model to Assess Chlorpyrifos Exposure and Dose in Children, Based on Urinary Biomarker Measurements. *Toxicological Sciences, 61*(2), 374-381. PMID 11353146

Rignell-Hydbom, A., Axmon, A., Lundh, T., Jonsson, B. A., Tiido, T., & Spano, M. (2007, May 8). Dietary exposure to methylmercury and PCBs and the association with semen parameters among Swedish fishermen. *Environmental Health, 6*, 1-14. PMID 17488503. doi:10.1186/1476-069X-6-14

Rignell-Hydbom, A., Rylander, L., & Hagmar, L. (2007, May). Exposure to persistent organochlorine pollutants and type 2 diabetes mellitus. *Human & Experimental Toxicology, 26*(5), 447-452. PMID 17623770. doi:10.1177/0960327107076886

Riley, D. M., Newby, C. A., Leal-Almeraz, T. O., & Thomas, V. M. (2001, August). Assessing Elemental Mercury Vapor Exposure from Cultural and Religious

Practices. *Environmental Health Perspectives, 109*(8), 779-784. PMID 11564612

Risher, J. F. (2003). *Elemental Mercury and Inorganic Mercury Compounds: Human Health Aspects*. Geneva, Switzerland: World Health Organization

Risher, J. F., Murray, H. E., & Prince, G. R. (2002, April). Organic mercury compounds: human exposure and its relevance to public health. *Toxicology and Industrial Health, 18*(3), 109-160. PMID 12974562

Ritchie, J. M., Vial, S. L., Fuortes, L. J., Guo, H., Reedy, V. E., & Smith, E. M. (2003, July). Organochlorines and Risk of Prostate Cancer. *Journal of Occupational and Environmental Medicine, 45*(7), 692-702. PMID 12855910. doi: 10.1097/01.jom.0000071510.96740.0b

Ritchie, K. A., Gilmour, W. H., Macdonald, E. B., Burke, F. L., McGowan, D. A., Dale, I. M., … Collington, D. (2002, May). Health and neuropsychological functioning of dentists exposed to mercury. *Occupational and Environmental Medicine, 59*(5), 287-293. PMID 11983843

Ritter, R., Scheringer, M., MacLeod, M., Schenker, U., & Hungerbuhler, K. (2009, August). A Multi-Individual Pharmacokinetic Model Framework for Interpreting Time Trends of Persistent Chemicals in Human Populations Application to a Postban Situation. *Environmental Health Perspectives, 117*(8), 1280-1286. PMID 19672409. doi:10.1289/ehp.0900648

Ritter, L., Solomon, K., Sibley, P., Hall, K., Keen, P., Mattu, G., & Linton, B. (2002, January 11). Sources, pathways and relative risks of contaminants in surface water and groundwater: a perspective prepared for the Walkerton inquiry. *Journal of Toxicology and Environmental Health Section A, 65*(1), 1-142. PMID 11809004

Robertson, G. L., Lebowitz, M. D., O'Rourke, M. K., Gordon, S., & Moschandreas, D. (1999, September-October). The National Human Exposure Assessment Survey (NHEXAS) study in Arizona – introduction and preliminary results. *Journal of Exposure Analysis and Environmental Epidemiology, 9*(5), 427-434. PMID 10554145

Robertson, L. W., & Hansen, L. G. (Eds.). (2001) *PCBs: Recent Advances in Environmental Toxicology and Health Effects*. Lexington, KY: The University Press of Kentucky

Robertson, W. G., & Marshall, R. W. (1979, December). Calcium measurements in serum and plasma – total and ionized. *CRC Critical Reviews in Clinical Laboratory Science, 11*(3), 271–305. PMID 116800

Robine, J.-M., Jagger, C., & Egidi, V. (2000, June). *Selection of a Coherent Set of Health Indicators*: *A First Step Towards A User's Guide to Health Expectancies for the European Union*. Montpellier, France: Euro-REVES

Robinson, C., Robinson, K., Tatgenhorst, C., Campbell, D., & Webb, C. (2006, July). Assessment of wastewater treatment plant workers exposed to biosolids: pilot test of a newly developed health survey. *Association of American Occupational Health Nurses Journal, 54*(7), 301-306. PMID 16862877

Rodier, P. M. (1995, September). Developing Brain as a Target of Toxicity. *Environmental Health Perspectives, 103*(Supplement 6), S73-S76. PMID 8549496

Rodriguez, C., Cook, A., Devine, B., Van Buynder, P., Lugg, R., Linge, K., & Weinstein, P. (2008, December). Dioxins, Furans and PCBs in Recycled Water for Indirect Potable Reuse. *International Journal of Environmental Research and Public Health, 5*(5), 356-367. PMID 19151430

Rodríguez, T., Younglove, L., Lu, C., Funez, A., Weppner, S., Barr, D. B., & Fenske, R. A. (2006, October-December). Biological Monitoring of Pesticide Exposures among Applicators and Their Children in Nicaragua. *International Journal of Occupational and Environmental Health, 12*(4), 312-320. PMID 17168218

Roegge, C. S., Wang, V. C., Powers, B. E., Klintsova, A. Y., Villareal, S., Greenough, W. T., & Schantz, S. L. (2004, February). Motor Impairment in Rats Exposed to PCBs and Methylmercury during Early Development. *Toxicological Sciences, 77*(2), 315-324. PMID 14600290. doi:10.1093/toxsci/kfg252

Roelofs, C. R., Barbeau, E., Ellenbecker, M., & Moure-Eraso, R. (2003, January-February). Prevention Strategies in Industrial Hygiene: A Critical Literature Review. *American Industrial Hygiene Association Journal, 64*, 62-67. PMID 12570397

Roels, H., Abdeladim, S., Ceulemans, E., & Lauwerys, R. (1987). Relationships between the Concentrations of Mercury in Air and in Blood or Urine in Workers Exposed to Mercury Vapor. *Annals of Occupational Hygiene, 31*(2), 135-145. PMID 3688710

Roels, H., Hubermont, G., Buchet, J., & Lauwerys, R. (1978). Placental Transfer of Lead, Mercury, Cadmium and Carbon Monoxide in Women. III. Factors Influencing the Accumulation of Heavy Metals in the Placenta and the Relationship Between Metal Concentration in the Placenta and in Maternal and Cord Blood. *Environmental Research, 16*, 236-247. PMID 679913

Rogan, W. J., Gladen, B. C., McKinney, J. D., Carreras, N., Hardy, P., Thullen, J., … Tully, M. (1986, February). Polychlorinated Biphenyls (PCBs) and Dichlorodiphenyl Dichloroethene (DDE) in Human Milk: Effects of Maternal Factors and Previous Lactation. *American Journal of Public Health, 76*(2), 172-177. PMID 3080910

Rogers, A., Huxley, P., Evans, S., & Gately, C. (2008, May). More than jobs and houses: mental health, quality of life and the perceptions of locality in an area undergoing urban regeneration. *Social Psychiatry and Psychiatric Epidemiology, 43*(5), 364-372. PMID 18274693. doi:10.1007/s00127-008-0316-2

Rogers, B. (1994a). *Occupational Health Nursing Concepts and Practice*. Philadelphia, PA: W. B. Saunders Company

Rogers, B. (1994b, July). Linkages in Environmental and Occupational Health. *Association of American Occupational Health Nurses Journal, 42*(7), 336-343. PMID 8060398

Rogers, B. (2005, April). Research with Protected Populations – Vulnerable Participants. *Association of American Occupational Health Nurses Journal*, 53(4), 156-157. PMID 15853290

Rogers, B., & Cox, A. (1998, January). Expanding horizons. Integrating environmental health in occupational health nursing. *Association of American Occupational Health Nurses Journal, 46*(1), 9-13. PMID 9481214

Rogers, B., McCurdy, L. E., Slavin, K., Grubb, K., & Roberts, J. R. (2009, May). Children's Environmental Health Faculty Champions Initiative: A Successful Model for Integrating Environmental Health into Pediatric Healthcare. *Environmental Health Perspectives, 117*(5), 850-855. PMID 19478972. doi:10.1289/ehp.0800203

Romero, D., Mounho, B., Lauer, F., Born, J., & Burchiel, S. (1997). Depletion of Glutathione by Benzo(a)pyrene Metabolites, Ionomycin, Thapsigargin and Phorbol Myristate in Human Peripheral Blood Mononuclear Cells. *Toxicology and Applied Pharmacology, 144*, 2-69. PMID 9169070. doi:10.1006/taap.1997.8113

Ronchetti, R., Van Den Hazel, P., Schoeters, G., Hanke, W., Rennezova, Z., Barreto, M., & Villa, M. P. (2006, October). Lead neurotoxicity in children: Is prenatal exposure more important than postnatal exposure? *Acta Paediatrica, 95*(Supplement 453), 45-49. PMID 17000569. doi:10.1080/08035320600886224

Ros, C., & Mwanri, L. (2003). Lead exposure, interactions and toxicity: food for thought. *Asian Pacific Journal of Clinical Nutrition, 12*(4), 388-395. PMID 14672861

Rosas, L., & Eskenazi, B. (2008, April). Pesticides and child neurodevelopment. *Current Opinion in Pediatrics, 20*(2), 191-197. PMID 18332717. doi:10.1097/MOP.0b013e3282f60a7d

Rosen, G. (1936). Social Aspects of Jacob Henle's Medical Thought (English translation of Henle, 1840). *Bulletin of the History of Medicine*, *6*, 911-983

Rosenstock, L., & Cullen, M. R. (Eds.). (1994). *Textbook of Clinical Occupational and Environmental Medicine*. Philadelphia, PA: W. B. Saunders Company

Rosenstock, L. & Lee, L. J. (2000, Spring). Women at Work. *Journal of the American Medical Women's Association, 55*(2), 67-68. PMID 10808653

Ross, E. M. (2002, September-October). Evaluation and Treatment of Iron Deficiency in Adults. *Nutrition in Clinical Care, 5*(5), 220-224. PMID 12455223

Rossi, L. C., Clemente, G. F., & Santaroni, G. (1976, May-June). Mercury and Selenium Distribution in a Defined Area and in Its Population. *Archives of Environmental Health, 31*(3), 160-165. PMID 1275561

Rothenberg, S. J., & Rothenberg, J. C. (2005, September). Testing the Dose-Response Specification in Epidemiology: Public Health and Policy Consequences for Lead. *Environmental Health Perspectives, 113*(9), 1190-1195. PMID 16140626

Rothman, K. J. (1993, December). Methodologic Frontiers in Environmental Epidemiology. *Environmental Health Perspectives, 101*(Supplement 4), 19-21. PMID 8206029

Rothman, K. J., Adami, H.-O., & Trichopoulos, D. (1998, September 5). Should the mission of epidemiology include the eradication of poverty? *Lancet, 352*, 810-813. PMID 9737304

Rothman, N., Stewart, W. F., & Shulte, P. A. (1995, June). Incorporating Biomarkers into Cancer Epidemiology: A Matrix of Biomarker and Study Design Categories. *Cancer Epidemiology, Biomarkers & Prevention, 4*(4), 301-311. PMID 7655323

Rotini, C., & Jorde, L. (2010, October 14). Ancestry and Disease in the Age of Genomic Medicine. *New England Journal of Medicine, 363*(16), 1551-1558. PMID 20942671

Rowland, A. S., & McKinstry, R. C. (2006). Lead toxicity, white matter lesions and aging. *Neurology, 66*, 1464-1465. PMID 16717201. doi:10.1212/01.wnl.0000219598.83289.84

Roy, A., Georgopoulos, P. G., Ouyang, M., Freeman, N., & Lioy, P. J. (2003). Environmental, dietary, demographic, and activity variables associated with biomarkers of exposure for benzene and lead. *Journal of Exposure Analysis*

and Environmental Epidemiology, 13(6), 417-426. PMID 14603342. doi:10.1038/sj.jea.7500296

Rozman, K. K., & Doull, J. (2000, April 3). Dose and time as variables of toxicity. *Toxicology, 144*(1-3), 169-178. PMID 10781885

Rozman, K. K., & Doull, J. (1999, July). Hormesis, Regulation, Toxicity and Risk Assessment. *Newsletter for Biological Effects of Low Level Exposures (BELLE)*, 1(8), 1-8

Rozman, K. K., Doull, J., & Hayes, W. J. (2001). Dose, Time and Other Factors Influencing Toxicity. In R. I. Kreiger (Ed.), *Handbook of Pesticide Toxicology* (2nd ed., Vol. I, pp. 1-93). San Diego, CA: Academic Press

Rozman, K. K., & Klaassen, C. D. (2003). Absorption, Distribution and Excretion of Toxicants. In C. D. Klaassen & J. B. Watkins (Eds.), *Casarett & Doull's Essentials of Toxicology* (pp. 59-70). New York, NY: McGraw-Hill

Rubin, R., & Murray, L. (2005). Environmental Justice. In L. Rosenstock, M. Cullen, C. A. Brodkin & C. A. Redlich (Eds.), *Textbook of Clinical Occupational and Environmental Medicine* (2nd ed., pp. 1255-1261). Philadelphia, PA: Elsevier Saunders

Rudel, R. A., Seryak, L. M., & Brody, J. G. (2008, January 17). PCB-containing wood floor finish is a likely source of elevated PCBs in residents' blood, household air and dust: a case study of exposure. *Environmental Health, 7*(2), 1-25. PMID 18201376. doi:10.1186/1476-069X-7-2

Rudmin, F. W. (2006). Debate in science: The case of acculturation. *AnthroGlobe Journal.* Retrieved April 1, 2011 from http://malinowski.kent.ac.uk/docs/rudminf_acculturation_061204.pdf

Russi, M. B., Borak, J. B., & Cullen, M. R. (2008, June 26). An Examination of Cancer Epidemiology Studies Among Populations Living Close to Toxic Waste Sites. *Environmental Health, 7*(32), 1-32. PMID 18578889. doi:10.1186/1476-069X-7-32

Ryan, M. A. (2006, October). The Politics of Risk: A Human Rights Paradigm for Children's Environmental Health Research. *Environmental Health Perspectives, 114*(10), 1613-1616. PMID 17035152

Ryan, P. B. (2005). Exposure Assessment, Industrial Hygiene and Environmental Management. In H. Frumkin (Ed.), *Environmental Health: from global to local* (pp. 72-95). San Francisco, CA: Jossey-Bass

Ryan, P. B., Burke, T. A., Cohen Hubal, E. A., Cura, J. J., & McKone, T. E. (2007, May). Using Biomarkers to Inform Cumulative Risk Assessment. *Environmental Health Perspectives, 115*(5), 833-840. PMID 17520075. doi:10.1289/ehp.9334

Rylander, L., Stromberg, U., Dyremark, E., Ostman, C., Nllsson-Ehle, P., & Hagmar, L. (1998, March 1). Polychlorinated Biphenyls in Blood Plasma among Swedish Female Fish consumers in Relation to Low Birth Weight. *American Journal of Epidemiology, 147*(5), 493-502. PMID 9525537

Sachs, H. K. (1996, June 12). Bone Lead Levels and Delinquent Behavior. (Peer Commentary on article "Bone lead levels in adjudicated delinquents: A case control study" by Needleman, McFarland, Ness, Fienberg & Tobin, 2002b, November-December). *Journal of the American Medical Association, 275*(22), 1725-1726. PMID 8637163

Sacker, A., Wiggins, R., Bartley, M., & McDonough, P. (2007, May). Self-Rated Health Trajectories in the United States and the United Kingdom: A Comparative Study. *American Journal of Public Health, 97*(5), 812-818. PMID 17395850. doi:10.2105/AJPH.2006.092320

Sadana, R., Mathers, C., Lopez, A., Murray, C., & Iburg, K. (2001). Comparative Analyses of More Than 50 Household Surveys on Health Status. *WHO/GPE Discussion Paper Series, 15*, 1-78. Retrieved April 1, 2011 from http://www.who.int/healthinfo/paper15.pdf

Safe Drinking Water Act Amendments, Pub. L. No. 99, §339 (1986, June 19)

Safe, S. (1987). PCBs and Human Health. In Safe, S. (Ed.), *Polychlorinated Biphenyls (PCBs): Mammalian and Environmental Toxicology* (pp. 133-146). New York, NY: Springer-Verlag

Safe, S., Safe, L., & Mullin, M. (1987). PCBs: Environmental Occurrence and Analysis. In Safe, S. (Ed.), *Polychlorinated Biphenyls (PCBs): Mammalian and Environmental Toxicology*, (pp. 1-14). New York, NY: Springer-Verlag

Safe, S. H. (2002). Exposure to POPs and other congeners. In S. H. Wilson & W. A. Suk (Eds.), *Biomarkers of environmentally associated disease* (pp. 455-470). Boca Raton, FL: Lewis Publishers

Saieva, C., Aprea, C., Tumino, R., Masala, G., Salvini, S., Frasca, G., … Palli, D. (2004, October). Twenty-four-hour urinary excretion of ten pesticide metabolites in healthy adults in two different areas of Italy (Florence and Ragusa). *Science of the Total Environment, 332*(1-3), 71-80. PMID 15336892. doi:10.1016/j.scitotenv.2004.02.026

Saint Phard, D., & Van Dorsten, B. (2004, April). Mercury Toxicity: clinical presentations in musculoskeletal medicine. *Orthopedics, 27*(4), 394-399. PMID 15101483

Sakamoto, M., Kubota, M., Liu, X. J., Murata, K., Nakai, K., & Satoh, H. (2004, July 15). Maternal and Fetal Mercury and n-3 Polyunsaturated Fatty Acids as a Risk and Benefit of Fish Consumption to Fetus. *Environmental Science & Technology, 38*(14), 3860-3863. PMID 15298193

Sala, M., Sunyer, J., Herrero, C., To-Figueras, J., & Grimalt, J. (2001, March). Association between serum concentrations of hexachlorobenzene and polychlorobiphenyls with thyroid hormone and liver enzymes in a sample of the general population. *Occupational and Environmental Medicine, 58*(3), 172-177. PMID 11171930

Salazar, M. K. (2000, March-April). Environmental Health: Responding to the Call. *Public Health Nursing, 17*(2), 73-74. PMID 10809635

Salazar, M. K., & Primomo, J. (1994, July). Taking the lead in environmental health: defining a model for practice. *Association of American Occupational Health Nurses Journal, 42*(7), 317-324. PMID 8060395

Salinas, J., & Peek, M. K. (2008, August). Work Experience and Gender Differences in Chronic Disease Risk in Older Mexicans. *Annals of Epidemiology, 18*(8), 628-630. PMID 18652980. doi:10.1016/j.annepidem.2008.04.005

Sällsten, G., Barregård, L., & Schütz, A. (1993, September). Decrease in mercury concentration in blood after long term exposure: a kinetic study of chloralkali workers. *British Journal of Industrial Medicine, 50*(9), 814-821. PMID 8398875

Sällsten, G., Kreku, S., & Unosson, H. (2000, May 26). A Small Dose of Ethanol Increases the Exhalation of Mercury in Low-Level-Exposed Humans. *Journal of Toxicology and Environmental Health, 60*(2), 89-100. PMID 10872631

Sällsten, G., Thorén, J., Barregård, L., Schütz, A., & Skarping, G. (1996, January). Long-term Use of Nicotine Chewing Gum and Mercury Exposure from Dental Amalgam Fillings. *Journal of Dental Research, 75*(1), 594-598. PMID 8655765

Salonen, J. T., Seppänen, K., Nyyssönen, K., Korpela, H., Kauhanen, J., Kantola, M., … Salonen, R. (1995, February 1). Intake of Mercury from Fish, Lipid Peroxidation, and the Risk of Myocardial Infarction and Coronary, Cardiovascular, and Any Death in Eastern Finnish Men. *Circulation, 91*(3), 645-655. PMID 7828289

Salvino, R., Ghanta, R., Seidner, D., Mascha, E., Xu, Y., & Steiger, E. (2006, May-June). Liver Failure is Uncommon in Adults Receiving Long-Term Parenteral Nutrition. *Journal of Parenteral and Enteral Nutrition, 30*(3), 202-208. PMID 16639066

Samarasinghe, K. (2007). *Facilitating a healthy transition for involuntary migrant families within primary health care* (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT C828223)

Samarawickrema, N., Pathmeswaran, A., Wickremasinghe, R., Peiris-John, R. Karunaratna, M., Buckley, N., … de Silva, J. (2008). Fetal effects of environmental exposure of pregnant women to organophosphorus compounds in a rural farming community in Sri Lanka. *Clinical Toxicology, 46*(6), 489-495. PMID 18584359. doi:10.1080/15563650701837030

Sampselle, C. M. (2007, January-March). Nickel-and-Dimed in America: underserved, understudied and underestimated. *Family Community Health, 30*(Supplement1), S4-S14. PMID 17159631

Sandberg, D. E., Vena, J. E., Weiner, J., Beehler, G. P., Swanson, M., & Meyer-Bahlburg, H. F. (2003, March). Hormonally active agents in the environment and children's behavior: assessing effects on children's gender-dimorphic outcomes. *Epidemiology, 14*(2), 148-154. PMID 12606879

Sandborgh-Englund, G., Elinder, C., Johanson, G., Lind, B., Skare, I., & Ekstrand, J. (1998, May). The Absorption, Blood Levels and Excretion of Mercury after a Single Dose of Mercury Vapor in Humans. *Toxicology and Applied Pharmacology, 150*(1), 146-153. PMID 9630463. doi:10.1006/taap.1998.8400

Sandel, M., Baeder, A., Bradman, A., Hughes, J., Mitchell, C., Shaughnessy, R., … Jacobs, D. (2010, September-October). Housing interventions and control of health-related chemical agents: a review of the evidence. *Journal of Public Health Management and Practice, 16*(5 Supplement), S24-S33. PMID 20689371

Sanders, T., Liu, Y., Buchner, V., & Tchounwou, P. B. (2009, January-March). Neurotoxic effects and biomarkers of lead exposure: a review. *Review in Environmental Health, 24*(1), 15-45. PMID 19476290

Sanzo, J. M., Dorronsoro, M., Amiano, P., Amurrio, A., Aguinagalde, F. X., Azpiri, M. A., & European Prospective Investigation into Cancer and Nutrition Group of Spain. (2001, October). Estimation and validation of mercury intake associated with fish consumption in an EPIC cohort of Spain. *Public Health Nutrition, 4*(5), 981-988. PMID 11784411. doi: 10.1079/PHN2001170

Saper, R. B., Kales, S. N., Paquin, J., Burns, M. J., Eisenberg, D. M., & Davis, R. B. (2004, December 15). Heavy Metal Content of Ayurvedic Herbal Medicine

Products. *Journal of the American Medical Association, 292*(23), 2868-2873. PMID 15598918. doi:10.1001/jama.292.23.2868

Saracci, R., & Vineis, P. (2007, November 28). Disease proportions attributable to environment. *Environmental Health, 6*, 1-38. PMID 18045465. doi:10.1186/1476-069X-6-38

Saraiva, M., Taichman, R., Nriagu, B., Eklund, S., & Burt, B. (2007, February). Lead exposure and periodontitis in U.S. adults. *Journal of Periodontal Research, 42*(1), 45-52. PMID 17214639. doi:10.1111/j.1600-0765.2006.00913.x

Sarnquist, C., Moix Grieb, E., Maldonado, Y. (2010, July). How Racial and Ethnic Groupings May Mask Disparities: The Importance of Separating Pacific Islanders From Asians in Prenatal Care Data. *Maternal and Child Health Journal, 14*(4), 635-641. PMID 19582560. doi:10.1007/s10995-009-0494-x

Sartwell, P. E. (1955, February). Uses of Epidemiology in Chronic Disease. *Public Health Reports, 70*(2), 170-171

SAS (version 9.2). (Computer Software). Cary, NC: SAS Institute, Inc.

Satcher, D. (2002, August 30). Women and Smoking: A Report of the Surgeon General. *Morbidity and Mortality Weekly Report, 51*(12), 1-30. Retrieved April 1, 2011 from http://www.cdc.gov/mmwr/PDF/rr/rr5112.pdf

Sato, A., & Nakajima, T. (1985, January). Enhanced metabolism of volatile hydrocarbons in rat liver following food deprivation, restricted carbohydrate intake and administration of ethanol, phenobarbital, polychlorinated biphenyl and 3-methylcholanthrene: a comparative study. (Abstract). *Xenobiotica, 15*(1), 67-75. PMID 3920836

Satoh, H. (2003, September). Behavioral Teratology of Mercury and Its Compounds. *Tohoku Journal of Experimental Medicine, 201*(1), 1-9. PMID 14609255

Sattler, B. (2002, August). Environmental Health Education and Nursing. In Nastoff, T., Drew, D., Wigington, P., Wakefield, J., Phillips, J. & O'Fallon, L. (Eds.),

Final Report of Nursing and Environmental Health Roundtable. Research Triangle Park, NC: Agency for Toxic Substances and Disease Registry

Sattler, B., Afzal, B., McPhaul, K. M., & Mood, L. H. (2006). Environmental Health. In M. Stanhope & J. Lancaster (Eds.), *Foundations of Nursing in the Community: Community-Oriented Practice* (2nd ed., pp. 93-113). St. Louis, MO: Mosby Elsevier

Sattler, B., & Del Bene Davis, A. (2008, July-August). Nurses' Role in Children's Environmental Health Protection. *Pediatric Nursing, 34*(4), 329-339. PMID 18814568

Sattler, B., & Lipscomb, J. (2003). *Environmental Health and Nursing Practice*. New York, NY: Springer Publishing Company

Sattler, B., McPhaul, K. M., Afzal, B., & Mood, L. H. (2004). Environmental Health. In M. Stanhope & J. Lancaster (Eds.), *Community & Public Health Nursing* (6th ed., pp. 220-247). St. Louis, MO: Mosby Elsevier

Saunders, N. R., & Dziegielewska, K. M. (2007, March 10). Developmental neurotoxicity of industrial chemicals. *Lancet, 369*(9564), 821-822. PMID 17350440. doi:10.1016/S0140-6736(07)60397-3

Sayre, J. W. (1996, June 12). Bone Lead Levels and Delinquent Behavior. (Peer Commentary on article "Bone lead levels in adjudicated delinquents: A case control study" by Needleman, McFarland, Ness, Fienberg & Tobin, 2002b, November-December). *Journal of the American Medical Association, 275*(22), 1726. PMID 8637163

Schamlaye, C., Marsh, D., Myers, G., Cox, C., Davidson, P., Choisy, O., … Clarkson, T. W. (1995, Winter). The Seychelles Child Development Study on Neurodevelopmental Outcomes in Children Following *in utero* Exposure to Methylmercury from a Maternal Fish Diet: Background and Demographics. *NeuroToxicology, 16*(4), 597-612. PMID 8714866

Schantz, S. L., Gardiner, J. E., Gasior, D. M., Sweeney, A. M., Humphrey, H. E., & McCaffrey, R. J. (1999, February). Motor Function in Aging Great Lakes Fish

Eaters. *Environmental Research A, 80*(2 Part 2), S46-S56. PMID 10092419. doi:10.1006/enrs.1998.3904

Schantz, S. L., Widholm, J. J., & Rice, D. C. (2003). Effects of PCB Exposure on Neuropsychological Function in Children. *Environmental Health Perspectives, 111,* 357-376. PMID 12611666

Schecter, A., Cramer, P., Boggess, K., Stanley, J., Päpke, O., Olson, J., … Schmitz, M. (2001). Intake of Dioxins and Related Compounds from Food in the U.S. Population. *Journal of Toxicology and Environmental Health, A, 63*, 1-18. PMID 11346131

Schecter, A., & Piskac, A. (2001). PCBs, Dioxins and Dibenzofurans: Measured Levels and Toxic Equivalents in Blood, Milk and Food from Various Countries. In L. W. Robertson & L. G. Hansen (Eds.) *PCBs: Recent Advances in Environmental Toxicology and Health Effects* (pp. 161-168). Lexington, KY: The University Press of Kentucky

Schecter, A., Stanley, J., Boggess, K., Masuda, Y., Mes, J., Wolff, M., … Chisholm, B. (1994, January). Polychlorinated Biphenyl Levels in the Tissues of Exposed and Nonexposed Humans. *Environmental Health Perspectives, 102*(Supplement 1), 149-158. PMID 8187704

Scheepers, P. T., & Heussen, G. A. (2005, January-February). New and improved biomarkers ready to be used in health-risk oriented exposure and susceptibility assessments: report of the 6th International Symposium on Biological Monitoring in Occupational and Environmental Health. *Biomarkers, 10*(1), 80-94. PMID 16097395. doi:10.1080/13547500500050085

Scheepers, P. T., & Heussen, G. A. (2008, March). New applications of biological monitoring for environmental exposure and susceptibility monitoring. Report of the 7th International Symposium on Biological Monitoring in Occupational and Environmental Health. *Biomarkers, 13*(2), 133-144. PMID 18270867. doi:10.1080/13547500701843510

Schell, L. M., Hubicki, L. A., DeCaprio, A. P., Gallo, M. V., Ravenscroft, J., Tarbell, A., … the Akwesasne Task Force on the Environment. (2003, June). Organochlorines, Lead and Mercury in Akwesasne Mohawk Youth. *Environmental Health Perspectives, 111*(7), 954-961. PMID 12782498

Scher, D. P. (2007). *Integrating biomonitoring and biological plausibility into pesticide risk assessment and epidemiology: an evaluation of exposure assessment techniques* (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 3280729)

Schettler, T. (2001, December). Toxic Threats to Neurologic Development of Children. *Environmental Health Perspectives, 109*(Supplement 6), 813-816. PMID 11744499

Scheuhammer, A., Meyer, M., Sandheinrich, M., & Murray, M. (2007, February). Effects of Environmental Methylmercury on the Health of Wild Birds, Mammals and Fish. *Ambio, 36*(1), 12-18. PMID 17408187

Scheuplein, R., Charnley, G., & Dourson, M. (2002). Differential Sensitivity of Children and Adults to Chemical Toxicity I: Biological Basis. *Regulatory Toxicology and Pharmacology, 35*, 429-447. PMID 12202057

Schisterman, E. F., Whitcomb, B. W., Buck Louis, G. M., & Louis, T. A. (2005, July). Lipid Adjustment in the Analysis of Environmental Contaminants and Human Health Risks. *Environmental Health Perspectives, 113*(7), 853-857. PMID 16002372

Schmidt, C. W. (1999, June). Poisoning young minds. *Environmental Health Perspectives, 107*(6), A302-A307. PMID 10339457

Schmidt, C. W. (2006a, September). Monitoring Environmental Exposures: Now It's Personal. *Environmental Health Perspectives, 114*(9), A528-A534. PMID 16966076

Schmidt, C. W. (2006b, December). Signs of the Times: Biomarkers in Perspective. *Environmental Health Perspectives, 114*(12), A701-A705. PMID 17185263

Schmidt, C. W. (2010, July). Mito-Conundrum: Unraveling Environmental Effects on Mitochondria. *Environmental Health Perspectives, 118*(7), A293-A297

Schnaas, L., Rothenberg, S. J., Flores, M.-F., Martínez, S., Hernández, C., Osorio, E., … Perroni. E. (2006, May). Reduced Intellectual Development in Children with Prenatal Lead Exposure. *Environmental Health Perspectives, 114*(5), 791-797. PMID 16675439

Schnaas, L., Rothenberg, S. J., Perroni, E., Martínez, S., Hernández, C., & Hernández, R. M. (2000, November-December). Temporal pattern in the effect of postnatal blood lead level on intellectual development of young children. *NeuroToxicology and Teratology, 22*(6), 805-810. PMID 11120385

Schober, S. E., Sinks, T. H., Jones, R. L., Bolger, P. M., McDowell, M., Osterloh, J., … Mahaffey, K. R. (2003, April 2). Blood Mercury Levels in U.S. Children and Women of Childbearing Age, 1999-2000. *Journal of the American Medical Association, 289*(13), 1667-1674. PMID 12672735. doi:10.1001/jama.289.13.1667

Schoen, E. J. (1996, June 12). Bone Lead Levels and Delinquent Behavior. (Peer Commentary on article "Bone lead levels in adjudicated delinquents: A case control study" by Needleman, McFarland, Ness, Fienberg & Tobin, 2002b, November-December). *Journal of the American Medical Association, 275*(22), 1728. PMID 8637163

Schoenborn, C. (2004, December 15). Marital Status and Health: United States, 1999-2002. *Advance Data, 351*. Retrieved April 1, 2011 from http://www.cdc.gov/nchs/data/ad/ad351.pdf

Scholl, T. O. (2005, May). Iron status during pregnancy: setting the stage for mother and infant. *The American Journal of Clinical Nutrition, 81*(Supplement 5), 1218S-1222S. PMID 15883455

Schwartz-Barcott, D. (2003). Response to "Concept Advancement: Enhancing Inductive Validity". *Research and Theory for Nursing Practice: An International Journal, 17*(2), 169-175. PMID 12880220

Schwartz-Barcott, D., & Kim, H. S. (1986). A hybrid model for concept development. In P. L. Chinn (Ed.), *Nursing research methodology: issues and implementations* (pp. 91-101). Rockville, MD: Aspen Systems

Schwartz-Barcott, D., & Kim, H. S. (1993). An expansion and elaboration of the hybrid model of concept development. In B. L. Rogers & K. A. Knafl (Eds.), *Concept development in nursing* (pp. 107-133). Philadelphia, PA: W. B. Saunders

Schwela, D. (2000, January-June). Air pollution and health in urban areas. *Reviews in Environmental Health, 15*(1-2), 13-42. PMID 10939084

Scott, P. K., Haws, L. C., Staskal, D. F., Birnbaum, L. S., Walker, N. J., DeVito, M. J., … Unice, K. M. (2006). An Alternative Method for Establishing TEFs for Dioxin-Like Compounds. Part 1. Evaluation of Decision Analysis Methods for Use in Weighting Relative Potency Data. *Organohalogen Compounds, 68*, 2519-2522

Scott, R. (1997). *Basic Concepts of Industrial Hygiene*. Boca Raton, FL: Lewis Publishers

Searle, B., Smith, S., & Cook, N. (2009, January). From Housing Wealth to Well-Being? *Sociological Health and Illness, 31*(1), 112-127. PMID 19144086. doi:10.1111/j.1467-9566.2008.01113.x

Searles Nielsen, S., Mueller, B. A., De Roos, A.-C., Viernes, H.-M., Farin, F. M., & Checkoway, H. (2005, July). Risk of Brain Tumors in Children and Susceptibility to Organophosphorus Insecticides: The Potential Role of Paraoxonase (PON1). *Environmental Health Perspectives, 113*(7), 909-913. PMID 16002382

Seed, J., Brown, R. P., Olin, S. S., & Foran, J. A. (1995, August). Chemical Mixtures: Current Risk Assessment Methodologies and Future Directions. *Regulatory Toxicology and Pharmacology, 22*(1), 75-94. PMID 7494906. doi:10.1006/rtph.1995.1071

Seelbach, M., Chen., L., Powell, A., Choi, Y.-J., Zhang, B., Hennig., B., & Toborek, M. (2010, April). Polychlorinated biphenyls disrupt blood-brain barrier integrity and promote brain metastasis formation. *Environmental Health Perspectives, 118*(4), 479-484. PMID 20064788. doi:10.1289/ehp.0901334

Selected Agents and Toxins, 42 C. F. R. §73 (2008, October 16)

Selevan, S., Rice, D., Hogan, K., Euling, S., Pfahles-Hutchens, A., & Bethel, J. (2003, April 17). Blood lead concentration and delayed puberty in girls. *New England Journal of Medicine, 348*(16), 1527-1537. PMID 12700372. doi:10.1056/NEJMoa020880

Senn, K. M., McGuinness, B. M., Buck, G. M., Vena, J. E., Anderson, S., & Rogers, B. T. (2005). Longitudinal study of babies born to mothers enrolled in a preconception prospective pregnancy study: study design and methodology, New York State Angler Cohort Study. *Environmental Research, 97,* 163-169. PMID 15533332. doi:10.1016/j.envres.2004.08.001

Sesline, D. H., & Jackson, R. J. (1994). The Effects of Prenatal Exposure to Pesticides. In H. L. Needleman & D. Bellinger (Eds.), *Prenatal Exposures to Toxicants: Developmental Consequences* (pp. 233-248). Baltimore, MD: The Johns Hopkins University Press

Severtson, D. J. (2004). Applying the common sense model to understand responses to arsenic risk: a theory-based evaluation of an arsenic well test program (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 3155159)

Sexton, K. (1992a). The role of scientific research in risk assessment and risk management decisions. *Otolaryngology, Head and Neck Surgery, 106*, 635-641. PMID 1608626

Sexton, K. (1992b, March-April). Cause for immediate concern; minorities and the poor clearly are more exposed. *EPA Journal, 18*,(1), 38-39. AN 9609101499

Sexton, K. (1997). Sociodemographic aspects of human susceptibility to toxic chemicals: Do class and race matter for realistic risk assessment? *Environmental Toxicology and Pharmacology, 4*, 261-269

Sexton, K. (2000). Socioeconomic and Racial Disparities in Environmental Health: Is Risk Assessment Part of the Problem or Part of the Solution? *Human and Ecological Risk Assessment, 6*(4), 561-574

Sexton, K. (2009). *Curriculum Vitae*. Retrieved April 1, 2011 from http://www.sph.uth.tmc.edu/cv/sexton.pdf

Sexton, K., Adgate, J. L., Fredrickson, A. L., Ryan, A. D., Needham, L. L., & Ashley, D. L. (2006, March). Using Biologic Markers in Blood to Assess Exposure to Multiple Environmental Chemicals for Inner-City Children 3-6 Years of Age. *Environmental Health Perspectives, 114*(3), 453-459. PMID 16507471

Sexton, K., Beck, B. D., Bingham, E., Brain, J. D., DeMarini, D. M., Hertzberg, R. C., … Pounds, J. G. (1995c, December 28). Chemical mixtures from a public health perspective: the importance of research for informed decision making. *Toxicology, 105*(2-3), 429-441. PMID 8571378

Sexton, K., Callahan, M., & Bryan, E. (1995a, April). Estimating exposure and dose to characterize health risks: the role of human tissue monitoring in exposure assessment. *Environmental Health Perspectives 103*(Supplement 3), S13-S29. PMID 7635107

Sexton, K., Gong, H. Jr., Bailar, J. C., Ford, J. G., Gold, D. R., Lambert, W. E., & Utell, M. J. (1993b). Air Pollution Health Risks: Do Class and Race Matter? *Toxicology and Industrial Health, 9*(5), 843-878. PMID 8184446

Sexton, K., & Hattis, D. (2007, May). Assessing Cumulative Health Risks from Exposure to Environmental Mixtures – Three Fundamental Questions. *Environmental Health Perspectives, 115*(5), 825-832. PMID 17520074. doi:10.1289/ehp.9333

Sexton, K., Kleffman, D. E., & Callahan, M. A. (1995b, July-September). An Introduction to the National Human Exposure Assessment Survey (NHEXAS) and Related Phase I Field Studies. *Journal of Exposure Analysis and Environmental Epidemiology, 5*(3), 229-232. PMID 8814770

Sexton, K., Olden, K., & Johnson, B. (1993a). "Environmental Justice": The Central Role of Research in Establishing a Credible Scientific Foundation for Informed Decision Making. *Toxicology and Industrial Health, 9*(5), 685-727. PMID 8184441

Sexton, K., & Reiter, L. W. (1989). Health Research at the U. S. Environmental Protection Agency. *Environmental Science & Technology, 23*(8), 917-930

Sexton, K., Selevan, S. G., Wagener, D. K., & Lybarger, J. A., (1992, November-December). Estimating human exposures to environmental pollutants: availability and utility of existing databases. *Archives of Environmental Health, 47*(6), 398-407. PMID 1485803

Sexton, K., Waller, L. A., McMaster, R. B., Maldonado, G., & Adgate, J. L. (2002). The Importance of Spatial Effects for Environmental Health Policy and Research. *Human and Ecological Risk Assessment, 8*(1), 109-125. doi:10.1080/20028091056764

Shamlaye, C. F., Marsh, D. O., Myers, G. J., Cox, C., Davidson, P. W., Choisy, O., … Clarkson, T. W. (1995, Winter). The Seychelles Child Development Study on Neurodevelopmental Outcomes in Children Following *in utero* Exposure to Methylmercury from a Maternal Fish Diet: Background and Demographics. *NeuroToxicology, 16*(4), 597-612. PMID 8714866

Shankar, S. (2010, October-December). Biology of aging brain. *Indian Journal of Pathology and Microbiology, 53*(4), 595-604. PMID 21045377. doi:10.4103/0377-4929.71995

Shannon, M., Woolf, A., & Goldman, R. (2003, January-February). Children's Environmental Health: One Year in a Pediatric Environmental Health Specialty Unit. *Ambulatory Pediatrics, 3*(1), 53-56. PMID 12540255

Sharara, F. I., Seifer, D. B., & Flaws, J. A. (1998, October). Environmental toxicants and female reproduction. *Fertility and Sterility, 70*(4), 613-622. PMID 9797086

Sharp, R. R. (2003, November). Ethical issues in environmental health research. *Environmental Health Perspectives, 111*(14), 1786-1788. PMID 14594633

Shavers, V. (2007, September). Measurement of Socioeconomic Status in Health Disparities Research. *Journal of the National Medical Association, 99*(9), 1013-1023. PMID 17913111

Shea, K. M. (2004, December). Mercury Levels in Mothers. (Peer Commentary on the article: "Blood organic mercury and dietary mercury intake: National Health and Nutrition Examination Survey, 1999 and 2000" by Mahaffey, Clickner, & Bodurow, 2004, December). *Environmental Health Perspectives, 112*(17), A978. PMID 15579400

Sheesley, R. J., Schauer, J. J., Garshick, E., Laden, F., Smith, T. J., Blicharz, A. P., & Deminter, J. T. (2009, February). Tracking personal exposure to particulate diesel exhaust in a diesel freight terminal using organic tracer analysis. *Journal of Exposure Science & Environmental Epidemiology, 19*(2), 172-186. PMID 18322451. doi:10.1038/jes.2008.11

Sheldon, L., & Hubal, E. (2009, August). Exposure as Part of a Systems Approach for Assessing Risk. *Environmental Health Perspectives, 117*(8), 1181-1184. PMID 19672394. doi:10.1289/ehp.0800407

Sheffield, M. M. (2004). *Occupational beryllium exposure: reconciling federal policies, regulations and contractor implementation guides to protect worker health* (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 3143617)

Shen, X.-M., Yan, C.-H., Guo, D., Wu, S.-M., Li, R.-Q., Huang, H., … Tang, J.-M. (1998, October). Low-Level Prenatal Lead Exposure and Neurobehavioral Development of Children in the First Year of Life: A Prospective Study in Shanghai. *Environmental Research, 79*(Section A1), 1-8. PMID 9756675. doi:10.1006/enrs.1998.3851

Shenker, B. J., Guo, T. L., & Shapiro, I. M. (2000, October). Mercury-Induced Apoptosis in Human Lymphoid Cells: Evidence that the Apoptotic Pathway is Mercurial Species Dependent. *Environmental Research, 84*(Section A2), 89-99. PMID 11068922. doi:10.1006/enrs.2000.4078

Shepard, M. P., Carroll, R. M., Mahon, M. M., Moriarty, H. J., Feetham, S. L., Deatrick, J. A., & Orsi, A. J. (1999, April). Conceptual and Pragmatic Considerations in Conducting a Secondary Analysis. *Western Journal of Nursing Research, 21*(2), 154-167. PMID 11512174

Shi, L., & Stevens, G. (2005). Vulnerability and Unmet Health Care Needs: The Influence of Multiple Risk Factors. *Journal of General Internal Medicine, 20*, 148-154. PMID 15836548. doi:10.1111/j.1525-1497.2005.40136.x

Shi, L., Stevens, G., Lebrun, L., Faed, P., & Tsai, J. (2008, November). Enhancing the Measurement of Health Disparities for Vulnerable Populations. *Journal of Public Health Management and Practice, 14*(Supplement), S45-S52. PMID 18843237

Shimizu, K., Tsukazaki, N., Watanabe, M., Ogawa, F., Kondo, T., & Katayama, I. (2002, February). Serum concentration of nitric oxide in Yusho patients over 30 years after the accidental poisoning of polychlorinated biphenyls in Japan. (Peer commentary on article "Effects of polychlorinated biphenyls on the nervous system" by Faroon, Jones, & de Rosa, 2001). *Toxicology and Industrial Health, 18*(1), 45-47. PMID 12703682

Shimokura, G., Savitz, D., & Symanski, E. (1998, February). Assessment of water use for estimating exposure to tap water contaminants. *Environmental Health Perspectives, 106*(2), 55-59. PMID 9432970

Shotyk, W., & LeRoux, G. (2005). Biogeochemistry and cycling of lead. *Metal Ions and Biological Systems, 43,* 239-275. PMID 16370121

Shukla, R., Bornschein, R. L., Dietrich, K. N., Buncher, C. R., Berger, O. G., Hammond, P. B., & Succop, P. A. (1989, October). Fetal and Infant Lead Exposure: Effects on Growth in Stature. *Pediatrics, 84*(4), 604-612. AN 4744383

Silbergeld, E. K. (1995). The hazards of synthetic (anthropogenic) chemicals. *Toxicology Letters, 82-83,* 835-841. PMID 8597151

Silbergeld, E. K., & Patrick, T. E. (2005). Environmental exposures, toxicologic mechanisms and adverse pregnancy outcomes. *American Journal of Obstetrics and Gynecology, 192*, S11-S21. PMID 15891707. doi:10.1016/j.ajog.2004.06.117

Simmons, J. E. (1995, December 28). Chemical Mixtures: challenge for toxicology and risk assessment. *Toxicology, 105*(2-3), 111-119. PMID 8571350

Sinclair, K. D., Lea, R. G., Rees, W. D., & Young, L. E. (2007). The developmental origins of health and disease; current theories and epigenetic mechanisms. *Society of Reproduction and Fertility Supplement, 64,* 425-443. PMID 17491163

Sinclair, U. Jr. (1906). *The Jungle*. Scotts Valley, CA: CreateSpace, Inc.

Singh, B. K., Walker, A., Morgan, J. A., & Wright, D. J. (2003, September). Effects of soil pH on the biodegradation of chlorpyrifos and isolation of a chlorpyrifos-degrading bacterium. *Applied Environmental Microbiology, 69*(9), 5198-5206. PMID 12957902

Singhai, R. L., & Thomas, J. A. (1980). *Lead Toxicity*. Baltimore, MD: Urban & Schwarzenberg

Sipes, I., & Schnellmann, R. (1987). Biotransformation of PCBs: Metabolic Pathways and Mechanisms. In Safe, S. (Ed.), *Polychlorinated Biphenyls (PCBs): Mammalian and Environmental Toxicology* (pp. 97-110). New York, NY: Springer-Verlag

Sircar, K. D. (2004). *Occupational chemical causes of coronary artery disease in aluminum workers* (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 3152992)

Sitarek, K., & Gralewicz, S. (2009). Early developmental effects of separate or combined perinatal exposure to methylmercury (MeHg) and 2,2',4,4',5,5'-hexachlorobiphenyl (PCB 153) in the rat. *International Journal of Occupational Medicine and Environmental Health, 22*(2), 89-105. PMID 19617190. doi:10.2478/v10001-009-0015-6

Skalická, V., & Kunst, A. (2008, May). Effects of spouses' socioeconomic characteristics on mortality among men and women in a Norwegian longitudinal study. *Social Science & Medicine, 66*(9), 2035-2047. PMID 18313188. doi:10.1016/j.socscimed.2008.01.020

Skerfving, S. (1978, August). Interaction between selenium and methylmercury. *Environmental Health Perspectives, 25*, 57-65. PMID 363410

Slagle, M. W., Sun, S. M., & Mathis, M. G. (1998, March). A conceptual model of occupational health nursing: the resource model. *Association of American Occupational Health Nurses Journal, 46*(3), 121-126. PMID 9582728

Slikker, W., Levin, E., & Slotkin, T. (2005). Mode of Action: Disruption of Brain Cell Replication, Second Messenger and Neurotransmitter Systems During Development Leading to Cognitive Dysfunction – Developmental Neurotoxicity of Nicotine. *Critical Reviews in Toxicology, 35*, 703-711. PMID 16417037

Slotkin, T. A. (2004, June). Guidelines for Developmental Neurotoxicity and Their Impact on Organophosphate Pesticides: A Personal View from an Academic Perspective. *NeuroToxicology, 25*(4), 631-640. PMID 15183016. doi:10.1016/S0161-813X(03)00050-0

Smeeding, T. M. (2009, Fall). New Comparative Measures of Income, Material Deprivation and Well-Being. *Journal of Policy Analysis and Management, 28*(4), 745-752

Smegal, D. C. (2000, June 8). *Human Health Risk Assessment: Chlorpyrifos*. Washington, DC: U.S. Environmental Protection Agency

Smith, A. F. (1991). Cognitive Processes in Long-Term Dietary Recall. *Vital and Health Statistics, 6*(4), 92-1079

Smith, J. (2007). *Prenatal maternal stress and coping among vulnerable rural young women*. (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 3296690)

Smith, K. M., Barraj, L. M., Kantor, M., & Sahyoun, N. R. (2009). Relationship between fish intake, *n-3* fatty acids, mercury and risk markers of CHD (National Health and Nutrition Examination Survey 1999-2002). *Public Health Nutrition, 12*(8), 1261-1269. PMID 18986590. doi:10.1017/S1368980008003844

Smith, K. R., Corvalan, C. F., & Kjellstrom, T. (1999, September). How Much Global Ill Health Is Attributable to Environmental Factors? *Epidemiology, 10*(5), 573-584. PMID 10468437

Smith, K. R., & Desai, M. A. (2002). The contribution of global environmental factors to ill-health. In P. Martens & A. J. McMichael (Eds.), *Environmental Change, Climate and Health* (pp. 52-95). Cambridge, U.K.: Cambridge University Press

Smith, M. N., & Whitney, G. M. (1991). Caring for the Environment: The Ecology of Health. In P. L. Chinn. *Anthology on Caring*. New York, NY: National League for Nursing Press

Smith, N. (2007, February 26). Preconception Care the "New Prenatal Care." *Nursing Spectrum (New England), 11*(5), 16. AN 2009528074.

Smith, S. K., Trevena, L., Nutbeam, D., Dixon, A., & McCaffery, K. J. (2009, December). Exploring patient involvement in healthcare decision making across different education and functional health literacy groups. *Social Science & Medicine, 69*(12), 1805-1812. PMID 19846245. doi:10.1016/j.socscimed.2009.09.056

Smith, W. E., & Smith, A. M. (1975). *Minamata*. New York, NY: Holt, Rinehart and Winston

Smyth, J. C. (2006). *A novel air sampling and analytical method for determination of airborne bronopol* (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 3248310)

Snyder, M., Ruth, V., Sattler, B., & Strasser, J. (1994, July). Environmental and Occupational Health Education. *Association of American Occupational Health Nurses Journal, 42*(7), 325-328. PMID 8060396

Soeters, P., Reijven, P., van Bokhorst-de van der Schueren, M. A., Schols, J., Halfens, R., Meijers, J. M., & van Gemert, W. G. (2008). A rational approach to nutritional assessment. *Clinical Nutrition, 27*, 706-716. PMID 18783855. doi:10.1016/j.clnu.2008.07.009

Solis, M. T., Yuen, E., Cortez, P. S., & Goebel, P. J. (2000, September). Family Poisoned by Mercury Vapor Inhalation. *American Journal of Emergency Medicine, 18*(5), 599-602. PMID 10999577. doi:10.1053/ajem.2000.4006

Solomon, G. M., & Moodley, J. (2007, May). Acute chlorpyrifos poisoning in pregnancy: A case report. *Clinical Toxicology (PHL), 45*(4), 416-419. PMID 17486485. doi:10.1080/15563650601117988

Son, J.-Y., Lee, J., Paek, D., & Lee, J.-T. (2009, August). Blood levels of lead, cadmium and mercury in the Korean population: results from the Second Korean National Human Exposure and Biomonitoring Examination. *Environmental Research, 109*(8), 738-744. PMID 19555934. doi:10.1016/j.envres.2009.03.012

Sorensen, N., Murata, K., Budtz-Jørgensen, E., Weihe, P., & Grandjean, P. (1999, July). Prenatal Methylmercury Exposure as a Cardiovascular Risk Factor at Seven Years of Age. *Epidemiology, 10*(4), 370-375. PMID 10401870

Spalt, E. W., Kissel, J. C., Shiral, J. H., & Bunge, A. L. (2009, March). Dermal absorption of environmental contaminants from soil and sediment: a critical review. *Journal of Exposure Science & Environmental Epidemiology, 19*(2), 119-148. PMID 18830234. doi:10.1038/jes.2008.57

SPSS (version 19.0). (Computer Software). Chicago, IL: SPSS, Inc.

Spurgeon, A. (2006, February). Prenatal Methylmercury Exposure and Developmental Outcomes: Review of the Evidence and Discussion of Future Directions. *Environmental Health Perspectives, 114*(2), 307-312. PMID 16451873

Stajich, G. V., Lopez, G. P., Harry, S. W., & Sexson, W. R. (2000, May). Iatrogenic exposure to mercury after hepatitis B vaccination in preterm infants. *The Journal of Pediatrics, 136*(5), 679-681. PMID 10802503. doi:10.1067/mpd.2000.105133

Stanton, M. E., & Spear, L. P. (1990, May-June). Workshop on the qualitative and quantitative comparability of human and animal developmental neurotoxicity. Work Group I Report: comparability of measures of developmental

neurotoxicity in humans and laboratory animals. *NeuroToxicology and Teratology, 12*(3), 261-267. PMID 2115099

StatTransfer (version 10.0). (Computer Software). Seattle, WA: Circle Systems, Inc.

Stavenes Andersen, I., Voie, Ø., Fonnum, F., & Mariussen, E. (2009). Effects of Methylmercury in Combination with Polychlorinated Biphenyls and Brominated Flame Retardants on the Uptake of Glutamate in Rat Brain Synaptosomes: A Mathematical Approach for the Study of Mixtures. *Toxicological Sciences, 112*(1), 175-184. doi: 10.1093/toxsci/kfp178

Stedman-Smith, M. M. (2008). *Documenting perceptions about pesticides and other environmental exposures with photovoice: Mothers' concerns for their children* (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 3336429)

Stellman, J. M. (1994, August). Where Women Work and the Hazards They May Face on the Job. *Journal of Occupational Medicine, 36*(8), 814-825. PMID 7807260

Stellman, J. M. (2000, Spring). Perspectives of Women's Occupational Health. *Journal of the American Medical Women's Association, 55*(2), 69-71, 95. PMID 10808654

Stern, A. H. (2005, May). A review of the studies of the cardiovascular health effects of methylmercury with consideration of their suitability for risk assessment. *Environmental Research, 98*(1), 133-142. PMID 15721894. doi:10.1016/j.envres.2004.07.016

Stern, A. H. (2007, October 23). Public health guidance on cardiovascular benefits and risks related to fish consumption. *Environmental Health, 6*, 1-31. PMID 17956606. doi:10.1186/1476-069X-6-31

Stern, A. H., & Gochfeld, M. (1999, March 10). Effects of Methylmercury Exposure on Neurodevelopment. (Peer Commentary on paper "Effects of Prenatal and Postnatal Methylmercury Exposure from Fish Consumption on Neurodevelopment: outcomes at 66 months of age in the Seychelles child development study" by Davidson, Myers, Cox, Axtell, Shamlaye, Sloan-

Reeves, … Clarkson, 1998, August 26). *Journal of the American Medical Association, 281*(10), 896-897. PMID 10078480

Steuerwald, U., Weihe, P., Jørgensen, P. J., Bjerve, K., Brock, J., Heinzow, B., … Grandjean, P. (2000, May). Maternal seafood diet, methylmercury exposure, and neonatal neurologic function. *The Journal of Pediatrics, 136*(5), 599-605. PMID 10802490. doi:10.1067/mpd.2000.102774

Stewart, P. W., Darvill, T., Lonky, E., Reihman, J., Pagano, J., & Bush, B. (1999, February). Assessment of Prenatal Exposure to PCBs from Maternal Consumption of Great Lakes Fish: An Analysis of PCB Pattern and Concentration. *Environmental Research Section A, 80*(2 Part 2), 87-96. PMID 10092422. doi:10.1006/enrs.1998.3905

Stewart, P. W., Lonky, E., Reihman, J., Pagano, J., Gump, B. B., & Darvill, T. (2008, October). The relationship between prenatal PCB exposure and intelligence (IQ) in 9-year-old children. *Environmental Health Perspectives, 116*(10), 1416-1422. PMID 18941588. doi:10.1289/ehp.11058

Stewart, P. W., Reihman, J., Lonky, E. I., Darvill, T. J., & Pagano, J. (2003, January-February). Cognitive development in preschool children prenatally exposed to PCBs and MeHg. *NeuroToxicology and Teratology, 25*(1), 11-22. PMID 12633733

Stewart, P. W., Sargent, D. M., Reihman, J., Gump, B. B., Lonky, E., Darvill, T., … Pagano, J. (2006, December). Response Inhibition During Differential Reinforcement of Low Rates (DRL) Schedules May Be Sensitive to Low-Level Polychlorinated Biphenyl, Methylmercury, and Lead Exposure in Children. *Environmental Health Perspectives, 114*(12), 1923-1929. PMID 17185286

Stewart, W. F., & Schwartz, B. S. (2007, October). Effects of Lead on the Adult Brain: A 15-Year Exploration. *American Journal of Industrial Medicine, 50*(10), 729-739. PMID 17311281. doi:10.1002/ajim.20434

Stewart, W. F., Schwartz, B. S., Davatizikos, C., Shen, D., Liu, D., Wu, X., … Youssem, D. (2006a, May). Past adult lead exposure is linked to neurodegeneration measured by brain MRI. *Neurology, 66*, 1476-1484. PMID 16717205. doi:10.1212/01.wnl.0000216138.69777.15

Strickland, O. L., DiIorio, C., Coverson, D., & Nelson, M. (2007). Advancing nursing science in vulnerable populations: measurement issues. *Annual Review of Nursing Research, 25*, 27-48. PMID 17958288

Stockard, J. J., Pope-Stockard, J. E., & Sharbrough, F. W. (1992). Brainstem Auditory Evoked Potentials in Neurology: Methodology, Interpretation, and Clinical Application. In M. J. Aminoff (Ed.), *Electrodiagnosis in Clinical Neurology* (3rd ed., pp. 503-536). New York, NY: Churchill Livingstone

Stone, D., Sudakin, D., & Jenkins, J. (2009). Longitudinal trends in organophosphate incidents reported to the National Pesticide Information Center, 1995-2007. *Environmental Health, 8*(18), 1-8. PMID 19379510. doi:10.1186/1476-069X-8-18

Storck, J. (1966, June). History of the United States National Committee on Vital and Health Statistics 1949-1964. *Vital and Health Statistics Series, 4*(5), 1-49. PMID 5296349

Storelli, M. M. (2008, August). Potential human health risks from metals (Hg, Cd, and Pb) and polychlorinated biphenyls (PCBs) via seafood consumption: estimation of target hazard quotients (THQs) and toxic equivalents (TEQs). *Food and Chemical Toxicology, 46*(8), 2782-2788. PMID 18584931. doi:10.1016/j.fct.2008.05.011

Storms, G., Saerens, J., & Deyn, P. (2004, December). Normative data for the Boston Naming Test in native Dutch-speaking Belgian children and the relation with intelligence. *Brain and Language, 91*(3), 274-281. PMID 15533553. doi:10.1016/j.bandl.2004.03.005

Strom, B. L. (2001, August-September). Data validity issues in using claims data. *Pharmacoepidemiology and Drug Safety, 10*(5), 389-392. PMID 11802582. doi:10.1002/pds.610

Stuart, A., Mudhasakul, S., & Sriwatanapongse, W. (2009, May). The social distribution of neighborhood-scale air pollution and monitoring protection. *Journal of the Air & Waste Management Association, 59*(5), 591-602. PMID 19583159

Struck, S., Schmidt, U., Gruening, B., Jaeger, I. S., Hossbach, J., & Preissner, R. (2008). Toxicity vs. Potency: Elucidation of Toxicity Properties Discriminating between Toxins, Drugs and Natural Compounds. *Genome Informatics*, 20, 231-242. PMID 19425137

Strully, K. (2009, September). Racial-ethnic disparities in health and the labor market: Losing and leaving jobs. *Social Science & Medicine, 69*(5), 768-776. PMID 19615805. doi:10.1016/j.socscimed.2009.06.025

Suarez, L., Gilani, Z., Felkner, M., Brender, J., Henry, J., & Hendricks, K. (2005, July-September). Exposure to Polychlorinated Biphenyls and Risk of Neural-tube Defects in a Mexican American Population. *International Journal of Occupational and Environmental Health, 11*(3), 233-237. PMID 16130963

SUDAAN (version 10.0). (Computer Software). Research Triangle Park, NC: RTI International

Sue, Y. (2000). Mercury. In L. R. Goldfrank, N. E. Flomenbaum, N. A. Lewin, M. Howland, R. S. Hoffman & L. S. Nelson (Eds.), *Goldfrank's Toxicologic Emergencies* (7th ed., pp. 241-244). New York, NY: McGraw-Hill

Sugarman, S. D. (2007, September 27). Cases in Vaccine Court – Legal Battles over Vaccines and Autism (Perspective). *The New England Journal of Medicine, 357*(13), 1275-1277. PMID 17898095. doi:10.1056/NEJMp078168

Sugawara, N., Ohba, T., Nakai, K., Kakita, A., Nakamura, T., Suzuki, K., … Satoh, H. (2008, June). Effects of perinatal coexposure to methylmercury and polychlorinated biphenyls on neurobehavioral development in mice. *Archives of Toxicology, 82*(6), 387-397. PMID 17992516. doi: 10.1007/s00204-007-0254-x

Suhonen, R., Välimäki, M., & Leino-Kilpi, H. (2008, April). A review of outcomes of individualized nursing interventions on adult patients. *Journal of Clinical Nursing, 17*(7), 843-860. PMID 18321285. doi:10.1111/j.1365-2702.2007.01979.x

Sullivan, M. C. & McGrath, M. M. (2003). Perinatal morbidity, mild motor delay and later school outcomes. *Developmental Medicine & Child Neurology, 45*, 104-112. PMID 12578236

Sun, J. (2004). *Cutting fluid mist formation and behavior mechanisms* (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 3126288)

Sun, P., Basu, I., Blanchard, P., Brice, K., & Hites, R. (2007, February 15). Temporal and spatial trends of atmospheric polychlorinated biphenyl concentrations near the Great Lakes. *Environmental Science & Technology, 41*(4), 1131-1136. PMID 17593710

Sunderland, E. M. (2007). Mercury Exposure from Domestic and Imported Estuarine and Marine Fish in the U.S. Seafood Market. *Environmental Health Perspectives, 115,* 235-242. PMID 17384771

Suñol, C. (2010, January). [Peer Commentary on paper "Use of gene expression of neural markers in cultured neural cells to identify developmental neurotoxicants" by H. Hogberg]. *Toxicological Sciences, 113*(1), 1-3. PMID 19808862. doi:10.1093/toxsci/kfp240

Surkan, P. J., Schnaas, L., Wright, R. J., Téllez-Rojo, M. M., Lamadrid-Figueroa, H., Hu, H., … Wright, R. O. (2008, March). Maternal self-esteem, exposure to lead and child neurodevelopment. *Neurotoxicology, 29*(2), 278-285. PMID 18261800. doi:10.1016/j.neuro.2007.11.006

Susser, M. (1973). *Causal Thinking in the Health Sciences: Concepts and Strategies of Epidemiology*. New York, NY: Oxford University Press

Susser, M. (1998, October). Does risk factor epidemiology put epidemiology at risk? Peering into the future. *Journal of Epidemiology and Community Health, 52*(10), 612-613. PMID 10023453

Susser, M., & Susser, E. (1996a, May). Choosing a Future for Epidemiology I. Eras and Paradigms. *American Journal of Public Health, 86*(5), 668-673. PMID 8629717

Susser, M., & Susser, E. (1996b, May). Choosing a Future for Epidemiology II. From Black Box to Chinese Boxes and Eco-Epidemiology. *American Journal of Public Health, 86*(5), 674-677. PMID 8629718

Suzuki, T. (1988). Hair and Nails: Advantages and Pitfalls when used in biological monitoring. In T. W. Clarkson, L. Friberg, G. F. Nordberg & P. R. Sager (Eds.), *Biological Monitoring of Toxic Metals* (pp. 623-637). New York, NY: Plenum Press

Suzuki, T., Imura, N., & Clarkson, T. W. (1991). Overview. In T. Suzuki, N. Imura & T. W. Clarkson (Eds.), *Advances in Mercury Toxicology* (pp. 1-40). New York, NY: Plenum Press

Suzuki, T., Miyama, T., & Katsunuma, H. (1971). Comparison of Mercury Contents in Maternal Blood, Umbilical Cord Blood and Placental Tissues. *Bulletin of Environmental Contamination and Toxicology, 5*(6), 502-508. BF 01539978

Svedberg, U. R. (2004). Fourier transform infrared spectroscopy in industrial hygiene applications: assessment of emissions from and exposures in wood processing industries (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT C817862)

Svensson, B.-G., Schütz, A., Nilsson, A., Åkesson, I., Åkesson, B., & Skerfving, S. (1992, September 11). Fish as a source of exposure to mercury and selenium. *The Science of the Total Environment, 126*(1-2), 61-74. PMID 1439752. doi:10.1016/0048-9697(92)90484-A

Swain, E. B., Jakus, P. M., Rice, G., Lupi, F., Maxson, P. A., Pacyna, J. M., … Veiga, M. M. (2007, February). Socioeconomic Consequences of Mercury Use and Pollution. *Ambio, 36*(1), 45-61. PMID 17408190

Swierzcek, S., Abuknesha, R. A., Chivers, I., Baranovska, I., Cunningham, P., & Price, R. G. (2004, July-October). Enzyme-immunoassay for the determination of metallothionein in human urine: application to environmental monitoring. *Biomarkers, 9*(4 - 5), 331-340. AN 15963184

Sykes, B. (2001). *The Seven Daughters of Eve*. New York: W. W. Norton & Company

Sze, J., & Prakash, S. (2004, May). Human Genetics, Environment and Communities of Color: Ethical and Social Implications. *Environmental Health Perspectives, 112*(6), 740-745. PMID 15121518

Szpir, M. (2006, February). New Thinking on Neurodevelopment. *Environmental Health Perspectives, 114*(2), A101-A107. PMID 16451834

Takeuchi, T., Kambara, T., Morikawa, N., Matsumoto, H., Shiraishi, Y., & Ito, H. (1959, November). Pathologic Observations of the Minamata Disease. *Acta Pathologica Japonica, 9*(Supplement), 769-783. PMID 13919260

Takser, L., Mergler, D., Baldwin, M., De Grosbois, S., Smargiassi, A., & Lafond, J. (2005, August). Thyroid Hormones in Pregnancy in Relation to Environmental Exposure to Organochlorine Compounds and Mercury. *Environmental Health Perspectives, 113*(8), 1039-1045. PMID 16079076

Tang, D., Li, T.-Y., Liu, J. J., Zhou, Z.-J., Yuan, T., Chen, Y.-H., … Perera, F. (2008, May). Effects of prenatal exposure to coal-burning pollutants on children's development in China. *Environmental Health Perspectives, 116*(5), 674-679. PMID 18470301. doi:10.1289/ehp.10471

Tanimura, T., Ema, M., & Kihara, T. (1980). Effects of combined treatment with methylmercury and polychlorinated biphenyls (PCBs) on the development of mouse offspring. In Persuad, T. (Ed.), *Neural and Behavioural Teratology*, (pp. 163-198). Baltimore, MD: University Park

Tarcher, A. B. (1995). Principles and Scope of Environmental Medicine. In J. M. Stellman (Ed.), *Encyclopedia of Occupational Health & Safety* (4th ed., pp. 1-18). Geneva, CH: International Labour Organisation

Tart, J. (2008). What are Biomarkers? *Environmental Health Perspectives, Student Edition*. Retrieved April 1, 2011 from http://www.ehponline.org/science-ed/2007/Biomarkers.pdf

Tart, K. T. (2006, October). Meeting Report: Looking Hard At Early Exposures. *Environmental Health Perspectives, 114*(10), A577

Taylor, D. (1992). Can the Environmental Movement Attract and Maintain the Support of Minorities? In B. Bryant & P. Mohai (Eds.), *Race and the Incidence of Environmental Hazards* (pp. 28-54). Boulder, CO: Westview Press

Taylor, J. K. (1987). *Quality Assurance of Chemical Measurements*. Boca Raton, FL: Lewis Publishers

Taylor, K. C., Jackson, L. W., Lynch, C. D., Kostyniak, P. J., & Buck-Louis, G. M. (2007). Preconception maternal polychlorinated biphenyl concentrations and the secondary sex ratio. *Environmental Research, 103,* 99-105. PMID 16780830. doi:10.1016/j.envres.2006.04.009

Taylor, P. R., Lawrence, C. E., Hwang, H., & Paulson, A. (1984, October). Polychlorinated Biphenyls: Influence on Birthweight and Gestation. *American Journal of Public Health, 74*(10), 1153-1154. PMID 6433730

Téllez-Rojo, M. M., Bellinger, D. C., Arroyo-Quiroz, C., Lamadrid-Figueroa, H., Mercado-Garcia, A., Schnaas-Arrieta, L., … Hu, H. (2006, August). Longitudinal Associations Between Blood Lead Concentrations Lower Than 10 μg/dl and Neurobehavioral Development in Environmentally Exposed Children in Mexico City. *Pediatrics, 118*(2), 323-330. PMID 16882776

Tenenbaum, D. (2009, April). It All Adds Up Over Time: Cumulative Lead Exposure and Cognition in Older Women. *Environmental Health Perspectives, 117*(4), 574-580. PMID 19440482

Terrell, M., Berzen, A., Small, C., Cameron, L., Wirth, J., & Marcus, M. (2009, August 15). A cohort study of the association between secondary sex ratio and parental exposure to polybrominated biphenyl (PBB) and polychlorinated biphenyl (PCB). *Environmental Health, 8*, 1-35. PMID 19682390. doi:10.1186/1476-069X-8-35

Teuschler, L. K. (2007). Deciding which chemical mixtures risk assessment methods work best for what mixtures. *Toxicology and Applied Pharmacology, 223,* 139-147. PMID 16997340. doi:10.1016/j.taap.2006.07.010

Thacker, S. B., Stroup, D. F., Parrish, R. G., & Anderson, H. A. (1996, May). Surveillance in Environmental Public Health: Issues, Systems, and Sources. *American Journal of Public Health, 86*(5), 633-638. PMID 8629712

Thain, W. (1980). *Monitoring Toxic Gases in the Atmosphere for Hygiene and Pollution Control*. Oxford, UK: Pergamon Press

Thayer, K. A., Melnick, R., Burns, K., Davis, D., & Huff, J. (2005, October). Fundamental Flaws of Hormesis for Public Health Decisions. *Environmental Health Perspectives, 113*(10), 1271-1276. PMID 16203233

The Annie E. Casey Foundation. *Kids Count Data Book*. (2008). Retrieved April 1, 2011 from http://datacenter.kidscount.org/databook/2008/PdfFiles.aspx

The Fifth International Metropolis Conference. (2000, November 16). *Myths and Realities: Exploring the Influences on Immigrant Health (Proceedings)*. Retrieved April 1, 2011 from http://international.metropolis.net/events/vancouver/workshop40.html

Theppeang, K., Glass, T., Bandeen-Roche, K., Todd, A., Rohde, C., Links, J., & Schwartz, B. (2008, June). Associations of bone mineral density and lead levels in blood, tibia and patella in urban-dwelling women. *Environmental Health Perspectives, 116*(6), 784-790. PMID 18560535. doi:10.1289/ehp.10977

Thompson, M. R. (2006). *The Concept of Exposure*. Unpublished manuscript, College of Nursing, University of Rhode Island, Kingston, RI.

Thompson, W. W., Price, C., Goodson, B., Shay, D. K., Benson, P., Hinrichsen, V. L., … DeStefano, F. (2007, September 27). Early Thimerosal Exposure and Neuropsychological Outcomes at 7 to 10 Years. *The New England Journal of Medicine*, *357*(13), 1281-1292. PMID 17898097. doi:10.1056/NEJMoa071434

Thomson, M., & Hoffman-Goetz, L. (2009, June 12). Defining and measuring acculturation: A systematic review of public health studies with Hispanic populations in the United States. *Social Science & Medicine*, 1-9. PMID 19525050. doi:10.1016/j.socscimed.2009.05.011

Thorpe, L. E., Gwynn, C., Mandel-Ricci, J., Roberts, S., Tsoi, B., Berman, L., … Frieden, T. R. (2006, July). Study Design and Participation Rates of the New York City Health and Nutrition Examination Survey, 2004. *Preventing Chronic Disease: Public Health Research, Practice and Policy, 3*(3), A94. PMID 16776895

Thrasher, J. D., Heuser, G., & Broughton, A. (2002, May-June). Immunological Abnormalities in Humans Chronically Exposed to Chlorpyrifos. *Archives of Environmental Health, 57*(3), 181-187. PMID 12507170

Thulier, D. (2010, November). A call for clarity in infant breast and bottle-feeding definitions for research. *Journal of Obstetric, Gynecologic and Neonatal Nursing, 39*(6), 627-634. PMID 21044147. doi:10.1111/j.1552-6909.2010.01197.x

Thygerson, S. M. (2006). *Increasing use of respirators through a leadership-based intervention* (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 3246312)

Tickner, J. A. (2005). Prevention. In H. Frumkin (Ed.), *Environmental Health from global to local* (pp. 849-894). San Francisco, CA: Jossey-Bass

Tiedje, L. B., & Wood, J. (1995, December). Sensitizing Nurses for a Changing Environmental Health Role. *Public Health Nursing, 12*(6), 359-365. PMID 8545302

Tiezheng, G., & Baasner, J. (1993, December). On-line microwave sample pretreatment for the determination of mercury in blood by flow injection cold vapor atomic absorption spectrometry. *Talanta, 40*(12), 1927-1936. PMID 18965872

Tilson, H. A. (1998a, June). Developmental Neurotoxicology of Endocrine Disruptors and Pesticides: Identification of Information Gaps and Research Needs. *Environmental Health Perspectives, 106*(Supplement 3), 807-811. PMID 9646041

Tilson, H. A., Jacobson, J. L., & Rogan, W. J. (1990, May-June). Polychlorinated Bipenyls and the Developing Nervous System: Cross-Species Comparisons. *NeuroToxicology and Teratology, 12*(3), 239-248. PMID 2115098

Tilson, H. A., Kodavanti, P., Mundy, W., & Bushnell, P. (1998b). Neurotoxicity of environmental chemicals and their mechanism of action. *Toxicology Letters, 102-103*, 631-635. PMID 10022326

Toffoletto, F., Crippa, M., & Torri, D. (2007, November-December). Interactions between alcohol and work exposure to chemical substances. *di Medicina del Lavoro, 98*(6), 513-520. PMID 18041472

Toft, G., Thulstrup, A., Jonsson, B., Pedersen, H., Ludwicki, J., Zvezday, V., & Bonde, J. (2010, May 10). Fetal loss and maternal serum levels of 2,2',4,4',5,5'-hexachlorobiphenyl (CB-153) and 1,1-dichloro-2,2-bis(p-chlorophenyl)ethylene (p,p'-DDE) exposure: a cohort study in Greenland and two European populations. *Environmental Health, 9*(22). doi:10.1186/1476-069X-9-22

Torres-Sánchez, L., Rothenberg, S. J., Schnaas, L., Cebrián, M. E., Osorio, E., del Carmen Hernández, M., … López-Carrillo, L. (2007, March). *In Utero* p,p'-DDE Exposure and Infant Neurodevelopment: A Perinatal Cohort in Mexico. *Environmental Health Perspectives, 115*(3), 435-439. PMID 17431495. doi:10.1289/ehp.9566

Toscano, C. D., & Guilarte, T. R. (2005). Lead neurotoxicity: from exposure to molecular effects. *Brain Research Reviews, 49,* 529-554. PMID 16269318. doi:10.1016/j.brainresrev.2005.02.004

Toth, D., & DiBenedetto, D. V. (2003, July). A Standardized Language for Occupational Health Nursing – The Nursing Minimum Data Set. *Association of American Occupational Health Nurses Journal, 51*(7), 283-286. PMID 12880230

Townsend, S. M. (2005). *Organizational correlates of secondary traumatic stress and burnout among sexual assault nurse examiners* (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 3199893)

Trasande, L., Cortes, J. E., Landrigan, P. J., Abercrombie, M. I., Bopp, R. F., & Cifuentes, E. (2010, January 11). Methylmercury exposure in a subsistence fishing community in Lake Chapala, Mexico: An ecological approach. *Environmental Health, 9*(1), 1. PMID 20064246. doi:10.1186/1476-069X-9-1

Trasande, L., & Landrigan, P. J. (2004, October). The National Children's Study: A Critical National Investment. *Environmental Health Perspectives, 112*(14), A789-A790. PMID 15471708

Trasande, L., Schecher, C. B., Haynes, K. A., & Landrigan, P. J. (2006, March). Mental retardation and prenatal methylmercury toxicity. *American Journal of Industrial Medicine, 49*(3), 153-158. PMID 16470549. doi:10.1002/ajim.20268

Trosclair, A., Husten, C., Pederson, L., & Dhillon, I. (2002, July 26). Cigarette Smoking Among Adults – United States, 2000. *Morbidity and Mortality Weekly Report, 51*(29), 642-645. PMID 12186222

Tsai, J., & Floyd, R. L. (2004). Alcohol Consumption Among Women Who are Pregnant or Who Might Become Pregnant – United States, 2002. *Morbidity and Mortality Weekly Report, 53*(50), 1178-1181. PMID 15614234

Tsai, J., Floyd, R. L., Green, P. P., & Boyle, C. A. (2007, September). Patterns and average volume of alcohol use among women of childbearing age. *Maternal and Child Health Journal, 11*(5), 437-445. PMID 17333387. doi:10.1007/s10995-007-0185-4

Tsubaki, T., & Irukayama, K. (1977). *Minamata Disease: methylmercury poisoning in Minamata and Niigata, Japan*. Amsterdam, The Netherlands: Elsevier Scientific Publishing Company

Tsuji, L. J., Wainman, B. C., Martin, I. D., Sutherland, C., Weber, J. P., Dumas, P., & Nieboer, E. (2008, April 15). The identification of lead ammunition as a source of lead exposure in First Nations: the use of lead isotope ratios. *Science of the Total Environment, 393*(2-3), 291-298. PMID 18272204. doi:10.1016/j.scitotenv.2008.01.022

Tulve, N. S., Jones, P. A., Nishioka, M. G., Fortmann, R. C., Croghan, C. W., Zhou, J. Y., … Friedman, W. (2006, October 15). Pesticide Measurements from the

First National Environmental Health Survey of Child Care Centers Using a Multi-Residue GC/MS Analysis Method. *Environmental Science Technology, 40*(20), 6269-6274. PMID 17120552

Tuomisto, J. (2006). Protecting our unborn children: how to measure exposure to thousands of chemicals? *Archives of Disease in Childhood, 91*, 627-628. PMID 16861478. doi:10.1136/adc.2006.095059

Turner, B. L. II, Kasperson, R. E., Matson, P. A., McCarthy, J. J., Corell, R. W., Christensen, L., … Schiller, A. (2003a, July 8). A framework for vulnerability analysis in sustainability science. *Proceedings of the National Academy of Sciences of the United States of America, 100*(14), 8074-8079. PMID 12792023. doi:10.1073/pnas.1231335100

Turner, B. L. II, Matson, P. A., McCarthy, J. J., Corell, R. W., Christensen, L., Eckley, N., … Tyler, N. (2003b, July 8). Illustrating the coupled human-environment system for vulnerability analysis: three case studies. *Proceedings of the National Academy of Sciences of the United States of America, 100*(14), 8080-8085. PMID 12815106. doi:10.1073/pnas.1231334100

Turner, C. J., Bhatnagar, M. K., & Yamashiro, S. (1981, March). Ethanol Potentiation of Methylmercury Toxicity: A Preliminary Report. *Journal of Toxicology and Environmental Health, 7*(3-4), 665-668. PMID 7288907

Turner, J. (2002, January 18). *Number of Naturally-Occurring Elements.* Retrieved April 1, 2011 from http://hps.org/publicinformation/ate/q933.htm

Turner, W. E., DiPietro, E., Cash, T. P., McClure, P. C., Patterson, D. G. Jr., & Shirkhan, H. (1994). An improved SPE extraction and automated sample cleanup method for serum PCDDs, PCDFs and coplanar PCBs. *Organohalogen Compounds, 19*, 31-35

Turrell, G., & Kavanagh, A. M. (2006, May). Socioeconomic pathways to diet: modeling the association between socio-economic position and food purchasing behavior. *Public Health Nutrition, 9*(3), 375-383. PMID 16684390

Twaroski, T. P., O'Brien, M. L., & Robertson, L. W. (2001, August 1). Effects of selected polychlorinated biphenyl (PCB) congeners on hepatic glutathione,

glutathione-related enzymes and selenium status: implications for oxidative stress. *Biochemical Pharmacology, 62*(3), 273-281. PMID 11434900

Tyl, R. W., & Sette, W. F. (1990, May-June). Workshop on the Qualitative and Quantitative Comparability of Human and Animal Developmental Neurotoxicology. Work Group III Report: Weight of Evidence and Quantitative Evaluation of Developmental Neurotoxicity Data. *NeuroToxicology and Teratology, 12*(3), 275-280. PMID 2196425

Uehara, R., Nakamura, Y., Matsuura, N., Kondo, N., & Tada, H. (2007, June). Dioxins in human milk and smoking of mothers. *Chemosphere, 68*(5), 915-920. PMID 17346770. doi:10.1016/j.chemosphere.2007.01.050

United Church of Christ Commission for Racial Justice. (1987). *Toxic Waste and Race in the United States: A National Report on the Racial and Socioeconomic Characteristics of Communities with Hazardous Wastes Sites*. Retrieved April 1, 2011 from http://www.ucc.org/about-us/archives/pdfs/toxwrace87.pdf

University of Rhode Island. (2009, September 2). *University Manual 10.42.15 Thesis and Dissertation Copyright Ownership.* Retrieved April 1, 2011 from http://www.uri.edu/facsen/CHAPTER_1007.htm

U.S. Census Bureau. (n.d.). *American Community Survey 2000-2005*. Retrieved April 1, 2011 from http://factfinder.census.gov/servlet/DatasetMainPageServlet?_program=ACS&_submenuId=factsheet_1&_lang=en&_ts=

U.S. Census Bureau (2000). *American Fact Finder (Decennial Census)*. Retrieved April 1, 2011 from http://factfinder.census.gov/home/saff/main.html?_lang=en

U.S. Census Bureau. (2001a, October). *Detailed Industry Code List: 1997 NAICS and U.S. Census 2000.* Retrieved April 1, 2011 from http://factfinder.census.gov/metadoc/industry.pdf

U.S. Census Bureau. (2001b, October). *Occupation Detailed Code List: Decennial 2000 SOC and U.S. Census 2000.* Retrieved April 1, 2011 from http://factfinder.census.gov/metadoc/occupation.pdf

U.S. Census Bureau. (2001c). *Women Ages 15-44 Without Health Insurance by State, 1999-2000*. Retrieved April 1, 2011 from http://www.marchofdimes.com/aboutus/680_2212.asp

U.S. Census Bureau. (2003a, March). *North American Industry Classification System (NAICS) Index of Industry and Occupations*: *Alternate Aggregation Structure*. Retrieved April 1, 2011 from http://www.dlt.ri.gov/lmi/pdf/alternate.pdf

U.S. Census Bureau. (2003b, August). *Occupations: 2000*. Retrieved April 1, 2011 from http://www.census.gov/prod/2003pubs/c2kbr-25.pdf

U.S. Census Bureau. (2008, August 26). *Basic Facts About Poverty*. Retrieved April 1, 2011 from http://www.census.gov/hhes/www/poverty

U.S. Census Bureau. (2009, September). *Census Data on Uninsured Women and Children*. Retrieved April 1, 2011 from http://www.marchofdimes.com/downloads/Census_data_on_Uninsured_Highlights09.pdf

U.S. Commission on Civil Rights. (2003, October). *Not in My Backyard: Executive Order 12,898 and Title VI as Tools for Achieving Environmental Justice*. Retrieved April 1, 2011 from http://www.usccr.gov/pubs/envjust/ej0104.pdf

U.S. Department of Health and Human Services. (n.d.) *CD4 Count*. Retrieved April 1, 2011 from http://www.aids.gov/hiv-aids-basics/diagnosed-with-hiv-aids/understand-your-test-results/cd4-count/

U.S. Department of Health and Human Services, Health Resources and Services Administration. (2008). *Women's Health USA 2008*. Retrieved April 1, 2011 from http://mchb.hrsa.gov/whusa08

U.S. Department of Health, Education & Welfare, National Institutes of Health, National Cancer Institute. (2009, July 28). *Biomarkers*. Retrieved April 1, 2011 from http://riskfactor.cancer.gov/areas/biomarkers

U.S. Department of Health, Education & Welfare, National Institutes of Health, National Heart, Lung and Blood Institute. (1998, September). *Clinical*

Guidelines on the Identification, Evaluation and Treatment of Overweight and Obesity in Adults (98-4083). Washington, DC: Author

U.S. Department of Health, Education & Welfare, National Institutes of Health, National Institute of Nursing Research. (2003). *Optimizing Pregnancy Outcomes in Minority Populations*. Bethesda, MD: Author

U.S. Department of Health, Education & Welfare, National Institutes of Health, Office of Dietary Supplements. (2004, August 1). *Dietary Supplement Fact Sheet: Selenium.* Retrieved April 1, 2011 from http://ods.od.nih.gov/factsheets/selenium.asp

U.S. Department of Health, Education & Welfare, National Institutes of Health, Office of Human Subjects Research. (1979, April 18). *The Belmont Report: Ethical Principles and Guidelines for the protection of human subjects of research*. Retrieved April 1, 2011 from http://ohsr.od.nih.gov/guidelines/belmont.htm

U.S. Department of Health, Education & Welfare, National Institutes of Health, Office of Human Subjects Research. (2005, June 23). *Protection of Human Subjects (45 Fed. Reg. §46).* Retrieved April 1, 2011 from http://www.hhs.gov/ohrp/humansubjects/guidance/45cfr46.htm

U.S. Department of Housing and Urban Development. (2009, July). *Income and Allowances Calculator*. Retrieved April 1, 2011 from http://www.hud.gov/offices/cpd/affordablehousing/training/web/calculator/calculator.cfm

U.S. Department of Labor, Bureau of Labor Statistics. (2003, July 15). *Unemployment of parents with children under 18*. Retrieved April 1, 2011 from http://www.bls.gov/opub/ted/2003/jul/wk2/art02.htm

U.S. Department of Labor, Bureau of Labor Statistics. (2005). *Women in the Labor Force: A Databook*. Retrieved April 1, 2011 from http://www.bls.gov/cps/wlf-databook-2005.pdf

U.S. Department of Labor, Bureau of Labor Statistics. (2008a, February 28). *Glossary of Terms*. Retrieved April 1, 2011 from http://www.bls.gov/bls/glossary.htm

U.S. Department of Labor, Bureau of Labor Statistics. (2008b, June 27). *Economic News Release: number of jobs held, labor market activity and earnings growth among the youngest baby boomers: results from a longitudinal survey summary*. Retrieved April 1, 2011 from http://www.bls.gov/news.release/nlsoy.nr0.htm

U.S. Department of Labor, Bureau of Labor Statistics. (2009). *Consumer Price Indexes*. Retrieved April 1, 2011 from http://www.bls.gov/cpi/home.htm

U.S. Department of Labor, Bureau of Labor Statistics. (2010, August 6). *Economic News Release: selected employment indicators*. Retrieved April 1, 2011 from http://www.bls.gov/news.release/empsit.t09.htm

U.S. Environmental Protection Agency. (1992a). *Environmental Equity: Reducing Risk for All Communities*. Washington, DC: Office of Solid Waste and Emergency Response

U.S. Environmental Protection Agency. (1995a, May 1). *Mercuric Chloride.* Retrieved April 1, 2011 from http://www.epa.gov/iris/subst/0692.htm

U.S. Environmental Protection Agency. (1995b, June 1). *Mercury, Elemental.* Retrieved April 1, 2011 from http://www.epa.gov/iris/subst/0370.htm

U.S. Environmental Protection Agency. (1997, December). *Mercury Study Report to Congress.* Washington, DC: U.S. Author

U.S. Environmental Protection Agency. (1998a, April). *What Do We Really Know About the Safety of High Production Volume Chemicals*? Washington, DC: Author

U.S. Environmental Protection Agency. (1999). *Proceedings of the NHEXAS Data Analysis Workshop (EPA 600/R-99/077).* Washington, DC: Author

U.S. Environmental Protection Agency. (2000). *Supplementary Guidance for Conducting Health Risk Assessment of Chemical Mixtures (EPA/630/R-00/002)*. Washington, DC: Author

U.S. Environmental Protection Agency. (2001, July 27). *Methylmercury.* Retrieved April 1, 2011 from http://www.epa.gov/iris/subst/0073.htm

U.S. Environmental Protection Agency. (2002, January). *Drinking Water from Household Wells. (EPA 816-K-02-003)*. Washington, DC: Author

U.S. Environmental Protection Agency. (2003b, May). *Framework for Cumulative Risk Assessment (EPA/630/P-02/001F)*. Washington, DC: Author

U.S. Environmental Protection Agency. (2003c, September). *Human Health Research Strategy (EPA/600/R-02/050*. Washington, DC: Author

U.S. Environmental Protection Agency. (2006). *Proceedings of the NHEXAS Data Analysis Workshop.* Retrieved April 1, 2011 from http://www.epa.gov/nerl/research/nhexas/nhexas.htm

U.S. Environmental Protection Agency. (2008a, January 15). *Breaking the cycle: 2001-2002 PBT Program Accomplishments.* Retrieved April 1, 2011 from http://www.epa.gov/pbt/pubs/pbtreport2002.htm

U.S. Environmental Protection Agency. (2008b, June 12). *Air Emissions Summary Through 2005.* Retrieved April 1, 2011 from http://www.epa.gov/airtrends/2006/emissions_summary_2005.htm

U.S. Environmental Protection Agency. (2008c, November). *Integrated Modeling for Integrate Environmental Decision Making*. Washington, DC: Author

U.S. Environmental Protection Agency. (2008d, December). *2006 Inventory Update Reporting: Data Summary.* Retrieved April 1, 2011 from http://www.epa.gov/oppt/iur/pubs/2006_data_summary.pdf

U.S. Environmental Protection Agency. (2008e, February 21). *2006 TRI Public Data Release.* Retrieved April 1, 2011 from http://www.epa.gov/tri/tridata/tri06/index.htm

U.S. Environmental Protection Agency. (2009a, January 29). *Chemical Assessment and Management Program (ChAMP).* Retrieved April 1, 2011 from http://www.epa.gov/champ

U.S. Environmental Protection Agency. (2009b, August 19). *What is the TSCA Chemical Substance Inventory?* Retrieved April 1, 2011 from http://www.epa.gov/oppt/newchems/pubs/invntory.htm

U.S. Environmental Protection Agency. (2009c, October 29). *Basic Information about Drinking Water Contaminants*. Retrieved April 1, 2011 from http://water.epa.gov/drink/contaminants/basicinformation/index.cfm

U.S. Environmental Protection Agency. (2009d, August 12). *EPA Bans PCB Manufacture; Phases Out Uses April 19, 197*9. Retrieved April 1, 2011 from http://www.epa.gov/history/topics/pcbs/01.htm

U.S. Environmental Protection Agency. (2010a, April 21). *Lead in Paint, Dust and Soil*. Retrieved April 1, 2011 from http://www.epa.gov/lead/

U.S. Environmental Protection Agency. (2010b, August 19). *Report on the Environment: Blood Cotinine Level*. Retrieved April 1, 2011 from http://cfpub.epa.gov/eroe/index.cfm?fuseaction=detail.viewInd&lv=list.listByAlpha&r=223968&subtop=208

U.S. Environmental Protection Agency. (2010c, September 16). *The Risk Assessment Process*. Retrieved April 1, 2011 from http://www.epa.gov/nheerl/research/human_health_risk/risk_process.html

U.S. Environmental Protection Agency. (2010d, November 2). *Environmental Justice*. Retrieved April 1, 2011 from http://www.epa.gov/compliance/environmentaljustice/

U.S. Environmental Protection Agency. (2010e, September 23). *Public Information: Mercury in Fish and Shellfish*. Retrieved April 1, 2011 from http://water.epa.gov/scitech/swguidance/fishshellfish/fishadvisories/publicinfo.cfm

U.S. Environmental Protection Agency. (2010f, April 28). *Essential Principles for Reform of Chemical Management Legislation*. Retrieved April 1, 2011 from http://www.epa.gov/oppt/existingchemicals/pubs/principles.html

U.S. Environmental Protection Agency. (2010g, August). *Enhancing EPA's Chemical Management Program*. Retrieved April 1, 2011 from http://www.epa.gov/oppt/existingchemicals/pubs/enhanchems.html

U.S. Environmental Protection Agency. Great Lakes Region. (1998b). *Mercury in Your Community and the Environment.* Retrieved April 1, 2011 from http://www.epa.gov/greatlakes/bnsdocs/merccomm/merccomm.pdf

U.S. Environmental Protection Agency, National Center for Environmental Assessment. (1996, September). *PCBs: Cancer Dose-Response Assessment and Application to Environmental Mixtures.* Washington, DC: Author

U.S. Environmental Protection Agency, National Center for Environmental Assessment. (2003, March). *EPA Handbook for Use of Data from the National Health and Nutrition Examination Surveys (NHANES): A Goldmine of Data for Environmental Health Analyses.* Washington, DC: Author

U.S. Environmental Protection Agency, National Environmental Justice Advisory Council. (2004, December). *Ensuring Risk Reduction in Communities with Multiple Stressors: Environmental Justice and Cumulative Risks/Impacts*. Washington, DC: Author

U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards. (1998c, May). *Locating and Estimating Air Emissions from Sources of Lead and Lead Compounds*. Research Triangle Park, NC: Author

U.S. Environmental Protection Agency, Office of Emergency and Remedial Response. (2002, December). *Task Force on Ritualistic Uses of Mercury (540-R-01-005).* Washington, DC: Author

U.S. Environmental Protection Agency, Office of Pollution Prevention and Toxic Substances. (2009, October 14). *Potential Export of Mercury Compounds from the United States for Conversion to Elemental Mercury*. Washington, DC: Author

U.S. Environmental Protection Agency, Region 5. (2001, May 22). *Mercury Response Guidebook.* Cincinnati, OH: Author

U.S. Environmental Protection Agency, Risk Assessment Forum. (1986, September). *Guidelines for the Health Risk Assessment of Chemical Mixtures*. Retrieved April 1, 2011 from http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=22567

U.S. Environmental Protection Agency, Risk Assessment Forum. (1992b, February). *Framework for Ecological Assessment* (EPA/630/R-92/001). Washington, DC: Author

U.S. Food and Drug Administration. (2004, March). *Mercury Levels in Commercial Fish and Shellfish.* Retrieved April 1, 2011 from http://www.fda.gov/Food/FoodSafety/Product-SpecificInformation/Seafood/FoodbornePathogensContaminants/Methylmercury/ucm115644.htm

U.S. Government Accounting Office. (1983, June 1). *Siting of Hazardous Waste Landfills and Their Correspondece with Racial and Economic Status of Surrounding Communities*. (GAO-RCED-83-168.) Retrieved April 1, 2011 from http://archive.gao.gov/d48t13/121648.pdf

U.S. Government Accounting Office. (2008, March). *EPA Science: New Assessment Process Further Limits the Credibility and Timeliness of EPA's Assessments of Toxic Chemicals.* Retrieved April 1, 2011 from http://www.gao.gov/new.items/d081168t.pdf

U.S. National Committee on Vital and Health Statistics. (1953, October). Recommendations for the Collection of Data on the Distribution and Effects of Illness, Injuries and Impairments in the United States. *Vital and Health Statistics Series*, 3(33), 19-38

U.S. National Committee on Vital and Health Statistics. (1968, March). Use of Vital and Health Records in Epidemiologic Research. *Vital and Health Statistics Series, 4*(7), 1-22. PMID 303833

U.S. National Committee on Vital and Health Statistics. (1977, July). Statistics Needed for Determining the Effects of the Environment on Health. *Vital and Health Statistics Series, 4*(20), 1-60. PMID 303833

U.S. National Committee on Vital and Health Statistics. (2007, December 19). *Enhanced Protections for Uses of Health Data*: *A Stewardship Framework for 'Secondary Uses' of Electronically Collected and Transmitted Health Data*. Retrieved April 1, 2011 from http://www.ncvhs.hhs.gov/071221lt.pdf

U.S. National Committee on Vital and Health Statistics, Public Health Services Division of Public Health Methods. (1957, October 21). *Proposal for Collection of Data on Illness and Impairments: United States*. Washington, DC: Author

U.S. Office of Management and Budget. (1978, May). *Statistical Policy Directive No. 14: Definition of Poverty for Statistical Purposes*. Retrieved April 1, 2011 from http://www.census.gov/hhes/www/povmeas/ombdir14.htm

U.S. Public Health Service. (1957a, January). The National Health Survey Act. *Public Health Reports, 72*(1), 1-4. PMID 13389694

U.S. Public Health Service. (1957b, January). Organization of National Health Survey. *Public Health Reports, 72*(1), 5-8. PMID 13389695

Ushio, F., & Doguchi, M. (1977, June). Dietary intakes of some chlorinated hydrocarbons and heavy metals estimated on the experimentally prepared diets. *Bulletin of Environmental Contamination Toxicology, 17*(6), 707-711. PMID 406952

Usydus, Z., Szlinder-Richert, J., Polak-Juszczak, L., Komar, K., Adamczyk, M., Malesa-Ciecwierz, M., & Ruczynska, W. (2009, March). Fish products available in Polish market – assessment of the nutritive value and human exposure to dioxins and other contaminants. *Chemosphere, 74*(11), 1420-1428. PMID 19147175. doi:10.1016/j.chemosphere.2008.12.023

Vahter, M., Akesson, A., Lind, B., Bjors, U., Schutz, A., & Berglund, M. (2000, October). Longitudinal Study of Methylmercury and Inorganic Mercury in Blood and Urine of Pregnant and Lactating Women, as well as in Umbilical

Cord Blood. *Environmental Research, 84*(Section A 2), 186-194. PMID 11068932. doi:10.1006/enrs.2000.4098

Valbonesi, P., Sartor, G., & Fabbri, E. (2003, August 1). Characterization of cholinesterase activity in three bivalves inhabiting the North Adriatic Sea and their possible use as sentinel organisms for biosurveillance programmes. *Science and the Total Environment, 312*(1-3), 79-88. PMID 12873401. doi:10.1016/S0048-9697(03)00227-4

Valcke, M., & Bouchard, M. (2009). Determination of no-observed effect level (NOEL)-biomarker equivalents to interpret biomonitoring data for organophosphorus pesticides in children. *Environmental Health, 8*, (5), 1-29. PMID 19228383. doi:10.1186/1476-069X-8-5

Valerius, A. J. (2005, May). *Population Prevalence and Risk Factors for Hepatitis C Infections in U. S. Male Military Veterans and Non-Veterans* (Doctoral dissertation). Retrieved from ProQuest Dissertations and Theses database. (AAT 3168220)

Van den Berg, M., Birnbaum, L., Bosveld, A. T., Brunström, B., Cook, P., Feeley, M., … Zacharewski, T. (1998, December). Toxic Equivalency Factors (TEFs) for PCBs, PCDDs and PCDFs for Humans and Wildlife. *Environmental Health Perspectives, 106*(12), 775-792. PMID 9831538

Van den Berg, M., Sinnige, T. L., Tysklind, M., Bosveld, A. T., Huisman, M., Koopmans-Essenboom, C., & Koppe, J. G. (1995, September). Individual PCBs as Predictors for Concentrations of Non- and Mono-Ortho PCBs in Human Milk. *Environmental Science & Pollution Research, 2*(20), 73-82

Van den Heuvel, J. P., & Davis, J. W. II (1999, March). Molecular approaches to identify exposure and risk to specific environmental pollutants. *Biomarkers, 4*(2), 93-105. AN 3859433

van den Hooven, E. H., Jaddoe, V. W., de Kluizenaar, Y., Hofman, A., Mackenbah, J. P., Steegers, E. A., ... Pierik, F. H. (2009, December 22). Residential traffic exposure and pregnancy-related outcomes: a prospective birth cohort study. *Environmental Health, 8*(1), 1-59. PMID 20028508. doi: 10.1186/1476-069X-8-59

Van Hemmen, J. J., Groeneveld, C. N., Van Drooge, H., Van Haelst, A. G., Schipper, A. H., & Van Der Jagt, K. E. (2001). Risk Assessment of Worker and Residential Exposure to Pesticides: Conclusions and Recommendations. *Annals of Occupational Hygiene, 45*(1001), S171-S174. PMID 11290365

Van Larebeke, N., Koppen, G., Nelen, V., Schoeters, G., Van Loon, H., Albering, H., … the Flemish Enviroment and Health Study Group (2004, January-February). Difference in HPRT mutant frequency among middle-aged Flemish women in association with area of residence and blood lead levels. *Biomarkers, 9*(1), 71-84. PMID 15204312. doi:10.1080/13547500310001652160

Van Ulirsch, G., Gleason, K., Gerstenberger, S., Moffett, D., Pulliam, G., Ahmed, T., & Fagliano, J. (2010, October). Evaluating and Regulating Lead in Synthetic Turf. *Environmental Health Perspectives, 118*(10), 1345-1349. PMID 20884393. doi:10.1289/ehp.1002239

Vandenbroucke, J. P. (2000, July 1). The testimony of Dr. Snow. *American Journal of Epidemiology, 152*(1), 1-10. PMID 10901324

Vause, T. D., Jones, L., Evans, M., Wilkie, V., & Leader, A. (2009, August). Pre-conception health awareness in infertility patients. *Journal of Obstetrics and Gynaecology Canada, 31*(8), 717-720. PMID 19772703

Vena, J. E., Buck, G. M., Kostyniak, P., Mendola, P., Fitzgerald, E., Sever, L., … Olson, J. (1996, May-August). The New York Angler Cohort Study: exposure characterization and reproductive and developmental health. *Toxicology and Industrial Health, 12*(3-4), 327-334. PMID 8843550

Venerosi, A., Ricceri, L., Scattoni, M., & Calamandrei, G. (2009, March). Prenatal chlorpyrifos exposure alters motor behavior and ultrasonic vocalization in cd-1 mouse pups. *Environmental Health, 8*(1-12), 1-30. PMID 19331648. doi:10.1186/1476-069X-8-12

Venturin, D. E. (2004). *The diffusion of innovative risk management strategies within the industrial manufacturing environment* (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 3141327)

Vettori, M., Goldoni, M., Caglieri, A., Poli, D., Folesani, G., Ceccatelli, S., & Mutti, A. (2006, September). Antagonistic effects of methyl-mercury and PCB153 on PC12 cells after a combined and simultaneous exposure. *Food and Chemical Toxicology, 44*(9), 1505-1512. PMID 16757078. doi:10.1016/j.fct.2006.04.009

Viel, J.-F., Fournier, E., & Danzon, A. (2010, August 8). Age-period-cohort modeling of non-Hodgkin's lymphoma incidence in a French region: a period effect compatible with an environmental exposure. *Environmental Health, 9*(1), 47. PMID 20691115. doi10.1186/1476-069X-9-47

Viltart, O., & Vanbesien-Mailliot, C. (2007, September). Impact of prenatal stress on neuroendocrine programming. *The Scientific World Journal, 1*(7), 1493-1537. PMID 17767365. doi:10.1100/tsw.2007.204

Vineis, P. (1998, October). Epidemiology between social and natural sciences. (Peer Commentary on the article "Does risk factor epidemiology put epidemiology at risk? Peering into the future" by Susser, 1998, October). *Journal of Epidemiology and Community Health, 52*(10), 616-617. PMID 10023453

Vineis, P., Khan, A., Vlaanderen, J., & Vermeulen, R. (2009, November 30). The impact of new research technologies on our understanding of environmental causes of disease: the concept of clinical vulnerability. *Environmental Health, 8*(1), 54. PMID 19948053. doi:10.1186/1476-069X-8-54

Virtanen, J. K., Rissanen, T. H., Voutilainen, S., & Tuomainen, T. (2007, February). Mercury as a risk factor for cardiovascular diseases. *The Journal of Nutritional Biochemistry, 18*(2), 75-85. PMID 16781863. doi:10.1016/j.jnutbio.2006.05.001

Vladeck, B. C. (2007, September-October). How Useful Is "Vulnerable" As A Concept? *Health Affairs, 26*(5), 1231-1234. PMID 17848430. doi:10.1377/hlthaff.26.5.1231

Voie, Ø., & Fonnum, F. (2000, January). Effect of polychlorinated biphenyls on production of reactive oxygen species (ROS) in rat synaptosomes. *Archives of Toxicology, 73*(10-11),588-93. PMID 10663391

Volz, S. A., Johnston, J. J., & Griffin, D. L. (2001). Solid Phase Extraction Gas Chromatography/Electron Capture Detector Method for the Determination of Organochlorine Pesticides in Wildlife Whole Blood. *Journal of Agriculture and Food Chemistry, 39*, 2741-2745

Vreugdenhil, H. J., Van Zanten, G. A., Brocaar, M. P., Mulder, P. G., & Weisglas-Kuperus, N. (2004). Prenatal exposure to polychlorinated biphenyls and breastfeeding: opposing effects on auditory P300 latencies in 9-year-old Dutch children. *Developmental Medicine & Child Neurology, 46,* 398-405. PMID 15174531

Vrijheid, M., Martinez, D., Aguilera, I., Ballester, F., Basterrechea, M., Esplugues, A., … Sunyer, J. (2010, October 25). Socioeconomic status and exposure to multiple environmental pollutants during pregnancy: evidence for environmental inequity? *Journal of Epidemiology and Community Health*, Advance online publication. PMID 20974841. doi:10.1136/jech.2010.117408

Vucic, V., Glibetic, M., Novakovic, R., Ngo, J., Ristic-Medic, D., Tepsic, J., … Gurinovic M. (2009, July). Dietary assessment methods used for low-income populations in food consumption surveys: a literature review. *British Journal of Nutrition, 101*(Supplement 2), S95-S101. PMID 19594969. doi:10.1017/S0007114509990626

Vupputri, S., Longnecker, M. P., Daniels, J. L., Guo, X., & Sandler, D. P. (2005, February). Blood mercury level and blood pressure among U.S. women: results from the National Health and Nutrition Examination Survey 1999-2000. *Environmental Research, 97*(2), 195-200. PMID 15533335. doi:10.1016/j.envres.2004.05.001

Wagener, D. K., Williams, D. R., & Wilson, P. (1993, September-October). Equity in environmental health: data collection and interpretation issues. *Toxicology and Industrial Health, 9*(5), 775-795. PMID 8184443

Walcott, R. M. & Milligan, R. (1992, March-April). Findings and recommendations of EPA's Environmental Equity Workgroup. *EPA Journal, 18*(1), 20-22

Waldron, I., Hughes, M., & Brooks, T. (1996, July). Marriage protection and marriage selection – prospective evidence for reciprocal effects of marital status and health. *Social Science & Medicine, 43*(1), 113-213. PMID 8816016

Waldron, I., Weiss, C., & Hughes, M. (1997, November). Marital status effects on health: Are there differences between never married women and divorced and separated women? *Social Science & Medicine, 45*(9), 1387-1397. PMID 9351156

Waldron, I., Weiss, C., & Hughes, M. (1998, September). Interacting effects of multiple roles on women's health. *Journal of Health and Social Behavior, 39*(3), 216-236. PMID 9785695

Walker, B. (2000, September). Neurotoxicity in Human Beings. *The Journal of Laboratory and Clinical Medicine, 136*(3), 168-180. PMID 10985495. doi:10.1067/mlc.2000.108940

Wallace, L. A. (2007). Biomarkers of Exposure. In W. R. Ott, A. C. Steinemann & L. A. Wallace (Eds.). *Exposure Analysis* (pp. 395-407). Boca Raton, FL: CRC Taylor & Francis Group

Wang, C. Y., Baldwin, L.-M., Saver, B. G., Dobie, S. A., Green, P. K., Cai, Y., & Klabunde, C. N. (2009, July). The Contribution of Longitudinal Comorbidity Measurements to Survival Analysis. *Medical Care, 47*(7), 813-821. PMID 19536031. doi:10.1097/MLR.0b013e318197929c

Wang, R., Jain, R., Wolkin, A., Rubin, C., & Needham, L. (2009, August). Serum concentrations of selected persistent organic pollutants in a sample of pregnant females and changes in their concentrations during gestation. *Environmental Health Perspectives, 117*(8), 1244-1299, PMID 19672404. doi:10.1289/ehp.0800105

Wang, S.-L., Chang, Y.-C., Chao, H.-R., Li, C.-M., Li, L.-A., Lin, L.-Y., & Päpke, O. (2006, May). Body Burdens of Polychlorinated Dibenzo-p-dioxins, Dibenzofurans, and Biphenyls and Their Relations to Estrogen Metabolism in Pregnant Women. *Environmental Health Perspectives, 114*(5), 740-745. PMID 16675430. doi:10.1289/ehp.8809

Wang, S.-L., Su, P.-H., Jong, S.B., Guo, Y. L., Chou, W.-L., & Päpke, O. (2005, November). *In Utero* Exposure to Dioxins and Polychlorinated Biphenyls and Its Relation to Thyroid Function and Growth Hormone in Newborns. *Environmental Health Perspectives, 113*(11), 1645-1650. PMID 16263525

Ware, J., Brook, R., Davies, A., & Lohr, K. (1981, June). Choosing Measures of Health Status for Individuals in General Populations. *American Journal of Public Health, 71*(6), 620-625. PMID 7235100

Wasserman, G. A., Liu, X., Popovac, D., Factor-Litvak, P., Kline, J., Waternaux, C., … Graziano, J. H. (2000, November-December). The Yugoslavia Prospective Lead Study: contributions of prenatal and postnatal lead exposure to early intelligence. *NeuroToxicology and Teratology, 22*(6), 811-818. PMID 11120386

Watanabe, C. (2001, January). Selenium deficiency and brain function: the significance for methylmercury toxicity (Abstract). *Nihon eiseigaku zasshi (Japanese Journal of Hygiene), 55*(4), 581-589. PMID 11265129

Watanabe, C. (2002, February). Modification of mercury toxicity by selenium: practical importance? *Tohoku Journal of Experimental Medicine, 196*(2), 71-77. PMID 12498318

Watanabe, C., & Satoh, H. (1996, April). Evolution of Our Understanding of Methylmercury as a Health Threat. *Environmental Health Perspectives, 104*(Supplement 2), 367-379. PMID 9182044

Watson, R. T. (2002). Foreword. In P. Martens & A. J. McMichael (Eds.). *Environmental Change, Climate and Health* (pp. xi-xiii). Cambridge, UK: Cambridge University Press

Watson, S. J. (1991). An analysis of the concept of experience. *Journal of Advanced Nursing, 16*, 1117-1121. PMID 1939925.

Watson, W. P., & Mutti, A. (2004, May-June). Role of biomarkers in monitoring exposures to chemicals: present position, future prospects. *Biomarkers, 9*(3), 211-242. PMID 15764289. doi:10.1080/13547500400015642

Watts, J. (2001, October 20). Mercury poisoning victims could increase by 20,000. *The Lancet (Br.), 358*(9290), 1349. PMID 11684229

Weaver, V. M., Buckley, T. J., & Groopman, J. D. (1998, June). Approaches to environmental exposure assessment in children. *Environmental Health Perspectives, 106*(Supplement 3), 827-832. PMID 9646045

Weaver, V. M., Davoli, C. T., Heller, P. J., Fitzwilliam, A., Peters, H. L., Sunyer, J., … Groopman, J. D. (1996, March). Benzene exposure assessed by urinary trans-muconic acid, in urban children with elevated blood lead levels. *Environmental Health Perspectives, 104*(3), 318-323. PMID 8919771

Webster, T. F. (2007, July 5). Bias magnification in ecologic studies: a methodological investigation. *Environmental Health, 6*, 1-17. PMID 17615079. doi:10.1186/1476-069X-6-17

Weihe, P., Hansen, J. C., Murata, K., Debes, F., Jørgensen, P. J., Steuerwald, U., … Grandjean, P. (2002). Neurobehavioral Performance of Inuit Children with Increased Prenatal Exposure to Methylmercury. *International Journal of Circumpolar Health, 61,* 41-49. PMID 12002946

Weinberg, D. H. (2004, May). *Evidence from Census 2000 About Earnings by Detailed Occupation for Men and Women*. Retrieved April 1, 2011 from http://www.census.gov/prod/2004pubs/censr-15.pdf

Weinstein, M., & Bernstein, S. (2003, January 21). Pink ladies: mercury poisoning in twin girls (clinical report). *Canadian Medical Association Journal, 168*(2), 201. PMID 12538551

Weintraub, M., & Birnbaum, L. S. (2008, July). Catfish consumption as a contributor to elevated PCB levels in a non-Hispanic black subpopulation. *Environmental Research, 107*(3), 412-417. PMID 18407261. doi:10.1016/j.envres.2008.03.001

Weis, B. K., Balshaw, D., Barr, J. R., Brown, D., Ellisman, M., Lioy, P. J., … Wilson, S. H. (2005, July). Personalized Exposure Assessment: Promising Approaches for Human Environmental Health Research. *Environmental Health Perspectives, 113*(7), 840-848. PMID 16002370

Weiss, B. (1994). The Developmental Neurotoxicity of Methylmercury. In H. L. Needleman & D. Bellinger (Eds.). *Prenatal Exposure to Toxicants* (pp. 112-129). Baltimore, MD: The Johns Hopkins University Press

Weiss, B. (1996, Spring). Long Ago and Far Away: A Retrospective on the Implications of Minamata. *NeuroToxicology, 17*(1), 257-263. PMID 8784837

Weiss, B. (1998, January-April). A Risk Assessment Perspective on the Neurobehavioral Toxicity of Endocrine Disruptors. *Toxicology and Industrial Health, 14*(1-2), 341-359. PMID 9460185

Weiss, B., & Bellinger, D. C. (2006, October). Social Ecology of Children's Vulnerability to Environmental Pollutants. *Environmental Health Perspectives, 114*(10), 1479-1485. PMID 17035129

Weiss, B., Clarkson, T. W., & Simon, W. (2002, October). Silent Latency Periods in Methylmercury Poisoning and in Neurodegenerative Disease. *Environmental Health Perspectives, 110*(Supplement 5), 851-854. PMID 12426145

Weiss, B., Cory-Slechta, D., Gilbert, S. G., Mergler, D., Miller, E., Newland, M. C., … Schettler, T. (2008, April). The new tapestry of risk assessment. *NeuroToxicology, 29,* 883-890. PMID 18501430. doi:10.1016/j.neuro.2008.04.004

Weiss, B., & Myers, J. P. (2001). Social, Economic and Cultural Context Influence the Expression of Exposure to Neurotoxicants. Session IV. Summary and Research Needs. *NeuroToxicology, 22,* 559-561. PMID 11770875

Weiss, J., Trip, L., & Mahaffey, K. R. (1999, September-October). Human Exposures to Inorganic Mercury. *Public Health Reports, 114*(5), 400-401. PMID 10590760

Weldon, M. M., Smolinski, M. S., Maroufi, A., Hasty, B. W., Gruss, D. L., Boulanger, … Dutton, R. J. (2000, July). Mercury poisoning associated with a Mexican beauty cream. *Western Journal of Medicine, 173*(1), 15-18. PMID 10903281

Weldon, R., Webster, M., Harley, K., Bradman, A., Fenster, L., Davis, M., … Eskenzai, B. (2010, June 30). Serum Persistent Organic Pollutants and Duration of Lactation among Mexican-American Women. *Journal of Environmental and Public Health, 2010.* Advance online publication. PMID 20671963. doi:10.1155/2010/861757

WELL Network. (2006, March). *Taking it to the States*. *A Call for Action on Comprehensive Chemicals Policy Development*. Retrieved April 1, 2011 from http://www.wellnetwork.org/pdfs/TITTSc906.pdf

Wendler, D., Kington, R., Madans, J., Van Wye, G., Christ-Schmidt, H., Pratt, L., … Emanuel, E. (2006, February). Are racial and ethnic minorities less willing to participate in health research? *PLoS Medicine, 3*(2), 201-210. PMID 16318411. doi:10.1371/journal.pmed.0030019

Weschler, C. (2009, January). Changes in indoor pollutants since the 1950s. *Atmospheric Environment, 43*(1), 153-169. doi:10.1016/j.atmosenv.2008.09.044

Weselak, M., Arbuckle, T. E., & Foster, W. (2007, January-March). Pesticide Exposures and Developmental Outcomes: The Epidemiological Evidence. *Journal of Toxicology and Environmental Health, Part B, 10*(1-2), 41-80. PMID 18074304. doi:10.1080/10937400601034571

West, P. C. (1992). Invitation to Poison? Detroit Minorities and Toxic Fish Consumption from the Detroit River. In B. Bryant & P. Mohai (Eds.). *Race and the Incidence of Environmental Hazards* (pp. 96-99). Boulder, CO: Westview Press

White, H. (1992). Hazardous Waste Incineration and Minority Communities. In B. Bryant & P. Mohai (Eds.). *Race and the Incidence of Environmental Hazards* (pp. 126-139). Boulder, CO: Westview Press

White, L. D., Cory-Slechta, D. A., Gilbert, M. E., Tiffany-Castiglioni, E., Zawia, N. H., Virgolini, M., … Riyaz Basha, M. (2007, November 15). New and evolving concepts in the neurotoxicology of lead. *Toxicology and Applied Pharmacology, 225*(1), 1-27. PMID 17904601. doi:10.1016/j.taap.2007.08.001

Whitmore, R. W., Pellizzari, E. D., Zelon, H. S., Michael, L. C., & Quackenboss, J. J. (2005, November). Cost/variance optimization for human exposure assessment studies. *Journal of Exposure Analysis and Environmental Epidemiology, 15*(6), 464-472. PMID 15886716. doi:10.1038/sj.jea.7500424

Whyatt, R. M., Barr, D. B., Camann, D. E., Kinney, P. L., Barr, J. R., Andrews, H. F., … Perera, F. P. (2003, May). Contemporary-Use Pesticides in Personal Air Samples during Pregnancy and Blood Samples at Delivery among Urban Minority Mothers and Newborns. *Environmental Health Perspectives, 111*(5), 749-756. PMID 12727605

Whyatt, R. M., Camann, D. E., Kinney, P. L., Reyes, A., Ramirez, J., Dietrich, J., … Perera, F. P. (2002, May). Residential Pesticide Use during Pregnancy among a Cohort of Urban Minority Women. *Environmental Health Perspectives, 110*(5), 507-514. PMID 12003754

Whyatt, R. M., Camann, D., Perera, F. P., Rauh, V. A., Tang, D., Kinney, P. L., … Barr, D. B. (2005, August 7). Biomarkers in assessing residential insecticide exposures during pregnancy and effects on fetal growth. *Toxicology and Applied Pharmacology, 206*(2), 246-254. PMID 15967215. doi:10.1016/j.taap.2004.11.027

Whyatt, R. M., Garfinkel, R., Hoepner, L. A., Holmes, D., Borjas, M., Williams, M. K., … Camann, D. E. (2007, March). Within- and Between-Home Variability in Indoor Air Insecticide Levels during Pregnancy among an Inner City Cohort from New York City. *Environmental Health Perspectives, 115*(3), 383-389. PMID 17431487. doi:10.1289/ehp.9546

Wibberly, D. G., Khere, A. K., Edwards, J. H., & Rushton, D. I. (1977, October). Lead levels in human placentae from normal and malformed births. *Journal of Medical Genetics, 14*(5), 339-345. PMID 592350

Wibowo, A., Del Castilho, P. Herber, R., & Zielhuis, R. (1977). Blood Lead and Serum Iron Levels in Non-Occupationally Exposed Males and Females. *International Archives of Occupational and Environmental Health, 39*, 113-120. PMID 885620

Wickrama, K., Lorenz, F., Conger, R., Elder, G., Abraham, W. T., & Fang, S.-A. (2006, July). Changes in family financial circumstances and the physical health

of married and recently divorced mothers. *Social Science & Medicine, 63*(1), 123-136. PMID 16414162. doi:10.1016/j.socscimed.2005.12.003

Widholm, J., Villareal, S., Seegal, R., & Schantz, S. (2004, December). Spatial alternation deficits following developmental exposure to Aroclor 1254 and/or methylmercury in rats. *Toxicological Science, 82*(2), 577-589. PMID 15456922. doi:10.1093/toxsci/kfh290

Wigle, D. T., Arbuckle, T. E., Turner, M. C., Bérube, A., Yang, Q., Liu, S., & Krewski, D. (2008, May). Epidemiologic Evidence of Relationships Between Reproductive and Child Health Outcomes and Environmental Chemical Contaminants. *Journal of Toxicology and Environmental Health, Part B, 11*(5-6), 373-517. PMID 18470797.doi:10.1080/10937400801921320

Wigle, D. T., Arbuckle, T. E., Walker, M., Wade, M. G., Liu, S., & Krewski, D. (2007, January-March). Environmental Hazards: Evidence for Effects on Child Health. *Journal of Toxicology and Environmental Health, Part B, 10*(1-2), 3-39. PMID 18074303. doi:10.1080/10937400601034563

Wilkes, C., Small, M., Davidson, I., & Andelman, J. (1996, October-December). Modeling the effects of water usage and co-behavior on inhalation exposures to contaminants volatilized from household water. *Journal of Exposure and Analytical Environmental Epidemiology, 6*(4), 393-412. PMID 9087861

Wilkinson, C. F., Christoph, G. R., Julien, E., Kelley, J. M., Kronenberg, J., McCarthy, J., & Reiss, R. (2000). Assessing the Risks of Exposures to Multiple Chemicals with a Common Mechanism of Toxicity: How to Cumulate? *Regulatory Toxicology and Pharmacology, 31,* 30-43. PMID 10715222. doi:10.1006/rtph.1999.1361

Willers, S., Gerhardsson, L., & Lundh, T. (2005, December). Environmental tobacco smoke (ETS) exposure in children with asthma-relation between lead and cadmium and cotinine concentrations in urine. *Respiratory Medicine, 99*(12), 1521-1527. PMID 16291074. doi:10.1016/j.rmed.2005.03.017

Williams, D. (1995, September). *William K. Reilly: Oral History Interview*. Retrieved April 1, 2011 from http://www.epa.gov/history/publications/print/reilly.htm

Williams, M. K., Barr, D. B., Camann, D. E., Cruz, L. A., Carlton, E. J., Borjas, M., … Whyatt, R. M. (2006, November). An Intervention to Reduce Residential Insecticide Exposure during Pregnancy among an Inner-City Cohort. *Environmental Health Perspectives, 114*(11), 1684-1689. PMID 17107853

Williams, P. L., Frumkin, H., Pierce, M. L., Manning, C. C., Elon, L., & Sanders, A. G. (2001, February). Reconstruction of occupational mercury exposures at a chloralkali plant. *Occupational and Environmental Medicine, 58*(2), 81-86. PMID 11160985

Wilson, J. G. (1959, August). Experimental studies on congenital malformations. *Journal of Chronic Diseases, 10*(2), 111-130. PMID 13673078

Wilson, J. G. (1973). *Environment and Birth Defects*. New York, NY: Academic Press

Wilson, J. G., & Fraser, F. C. (1977). *Handbook of Teratology*. New York, NY: Plenum Press

Wilson, S. H., & Suk, W. A. (2002). *Biomarkers of Environmentally Associated Disease*. Boca Raton, FL: Lewis Publishers

Wiltshire, J. C., Person, S. D., Kiefe, C. I., & Allison, J. J. (2009, September). Disentangling the influence of socioeconomic status on differences between African American and white women in unmet medical needs. *American Journal of Public Health, 99*(9), 1659-1665. PMID 19608942

Wingo, P., Kulkami, A., Borrud, L., McDonald, J., Villalobos, S., & Green, D. (2009, July). Health Disparities Among Mexican American Women Aged 15-44 Years: National Health and Nutrition Examination Survey, 1999-2004. *American Journal of Public Health, 99*(7), 1300-1307. PMID 19443827. doi:10.2105/AJPH.2008.145169

Winkleby, M. A., Jatulis, D. E., Frank, E., & Fortmann, S. P. (1992, June). Socioeconomic status and health: how education, income, and occupation contribute to risk factors for cardiovascular disease. *American Journal of Public Health, 82*(6), 816-820. PMID 1585961

Winkelstein, W., & Balfour, J. L. (1996, June 12). Bone Lead Levels and Delinquent Behavior. (Peer Commentary on article "Bone lead levels in adjudicated delinquents: A case control study" by Needleman, McFarland, Ness, Fienberg & Tobin, 2002b, November-December). *Journal of American Medical Association, 275*(22), 1728. PMID 8637163

Winneke, G. (2007, October). Appraisal of neurobehavioral methods in environmental health research: the developing brain as a target for neurotoxic chemicals. *International Journal of Hygiene and Environmental Health, 210*(5), 601-609. PMID 17869181. doi:10.1016/j.ijheh.2007.07.015

Winneke, G., Bucholski, A., Heinzow, B., Krämer, U., Schmidt, E., Walkowiak, J., … Steingrüber, H.-J. (1998, December 28). Developmental neurotoxicity of polychlorinated biphenyls (PCBs): cognitive and psychomotor functions in 7-month old children. *Toxicology Letters, 102-103,* 423-428. PMID 10022290

Winston, G. W., Narayan, S., & Bounds, P. L. (1990). Profiles of ethanol-induced microsomal alkoxyresorufin (alkoxyphenoxazone) o-dealkylation: comparison with phenobarbital- and Aroclor 1254-induced systems. *Alcohol and Alcoholism, 25*(6), 667-672. PMID 2085350

Winters, D. (2003, December). *Dioxin-like PCBs*. Retrieved April 1, 2011 from: http://epa.gov/bns/dioxin/pres/winters_dec2003.pdf

Wipfli, H., Avila-Tang, E., Navas-Acien, A., Kim, S., Onicescu, G., Yuan, J., … Famri Homes Study Investigators. (2008, April). Secondhand Smoke Exposure Among Women and Children: Evidence From 31 Countries. *American Journal of Public Health*, *98*(4), 672-679. PMID 18309121. doi:10.2105/AJPH.2007.126631

Wittassek, M., Wiesmüller, G. A., Koch, H. M., Eckard, R., Dobler, L., Müller, J., … Schlüter, C. (2007, May). Internal phthalate exposure over the last two decades – a retrospective human biomonitoring study. *International Journal of Hygiene and Environmental Health, 210*(3-4), 319-333. PMID 17400024. doi:10.1016/j.ijheh.2007.01.037

Wolcott, R. M., & Milligan, R. (1992, March-April). Findings and recommendations of EPA's Environmental Equity Workgroup. *EPA Journal, 18*(1), 20-21. AN 9609101488

Wolff, M., Britton, J., Boguski, L., Hochman, S., Maloney, N., Serra, N., … Forman, J. (2008, July). Environmental exposures and puberty in inner-city girls. *Environmental Research, 107*(3), 393-400. PMID 18479682. doi:10.1016/j.envres.2008.03.006

Wolff, M. S., Camann, D., Gammon, M., & Stellman, S. (1997, January). Proposed PCB Congener Groupings for Epidemiological Studies. *Environmental Health Perspectives, 105*(1), 13-14. PMID 9074863

Wolff, M. S., Deych, E., Ojo, F., & Berkowitz, G. S. (2005, February). Predictors of organochlorines in New York City pregnant women, 1998-2001. *Environmental Research, 97*(2), 170-177. PMID 15533333. doi:10.1016/j.envres.2004.07.014

Wolff, M. S., Engel, S., Berkowitz, G., Teitelbaum, S., Siskind, J., Barr, D. B., & Wetmur, J. (2007, February). Prenatal Pesticide and PCB Exposures and Birth Outcomes. *Pediatric Research, 61*(2), 243-250. PMID 17237730. doi:10.1203/pdr.0b013e31802d77f0

Wood, G. (2007). *Neurodevelopmental models of schizophrenia*. (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT NR47997)

Wood, S. L., Jarrell, J. J., Swaby, C., & Chan, S. (2007, November 15). Endocrine disruptors and spontaneous premature labor: a case control study. *Environmental Health, 6*(35), PMID 18005447. doi:10.1186/1476-069X-6-35

Woodruff, T., & Sutton, P. (2010, August). Pulling Back the Curtain: Improving Reviews in Environmental Health. *Environmental Health Perspectives, 118*(8), A326-A327. PMID 20675250. doi:10.1289/ehp.1002691

Woodruff, T., Zeise, L., Axelrad, D., Guyton, K., Janssen, S., Miller, M., … Zoeller, R. T. (2008, November). Meeting Report: Moving Upstream – Evaluating Adverse Upstream End Points for Improved Risk Assessment and Decision-Making. *Environmental Health Perspectives, 116*(11), 1568-1575. PMID 19057713. doi:10.1289/ehp.11516

Woodruff, T., Zota, A. & Schwartz, J. (2011, January 14). Environmental Chemicals in Pregnant Women in the U.S.: NHANES 2003-2004. *Environmental Health Perspectives*. Advance online publication. PMID 21233055. doi:10.1289/ehp.1002727

Woodward, A. (2002). Epidemiology, environmental health and global change. In P. Martens & A. J. McMichael (Eds.). *Environmental Change, Climate and Health* (pp. 290-310). Cambridge, UK: Cambridge University Press

Wootton, B. H. (1997, April). Gender differences in occupational employment. *Monthly Labor Review*, *130*(6), 15-24

World Health Organization. (1990). *First European Conference on Environment and Health. Frankfurt December 7-8, 1989*. Copenhagen, Denmark: Author. Regional Publication European Series 35C

World Health Organization. (1995). *Physical Status: The Use and Interpretation of Anthropometry*. Geneva, Switzerland: Author

World Health Organization. (2001). Comparative Analyses of More Than 50 Household Surveys on Health Status. *WHO/GPE Discussion Paper Series, 15*, 1-78. Retrieved April 1, 2011 from http://www.who.int/healthinfo/paper15.pdf

World Health Organization, Joint Expert Committee on Food Additives. (2003, June 3). *Important Developments in Scientific Evidence on Methylmercury Toxicity Exposure and Policies on Permissible Exposure Levels 2000-2003*. Geneva, Switzerland: Author

World Health Organization, European Centre for Environment and Health. (2006). *Report on the WHO technical meeting on quantifying disease from inadequate housing (November 28-30, 2005)*. Copenhagen, Denmark: WHO Regional Office for Europe

Woteki, C. E. (2003, February). Integrated NHANES: Uses in National Policy. *The Journal of Nutrition, 133*(Supplement 2), S582-S584. PMID 12566507

Wright, B. H. (1992). The Effects of Occupational Illness and Disease on the Health Status of Black Americans: A Review. In B. Bryant & P. Mohai (Eds.), *Race and the Incidence of Environmental Hazards* (pp. 114-125). Boulder, CO: Westview Press

Wright, J. P., Dietrich, K. N., Ris, M. D., Hornung, R. W., Wessel, S. D., Lanphear, B., … Rae, M. N. (2008, May 27). Association of Prenatal and Childhood Blood Lead Concentrations with Criminal Arrests in Early Adulthood. *PLoS Medicine, 5*(5), 732-740. PMID 18507497. doi:10.1371/journal.pmed.0050101

Wright, J. P., Shaw, M. C., & Keeler, L. C. (2002, January 2). Refinements in Acute Dietary Exposure Assessments for Chlorpyrifos. *Journal of Agricultural and Food Chemistry, 50*(1), 235-241. PMID 11754574

Wright, R. O., Amarasiriwardena, C., Woolf, A. D., Jim, R., & Bellinger, D. C. (2006, March). Neuropsychological correlates of hair arsenic, manganese and cadmium levels in school-age children residing near a hazardous waste site. *Neurotoxicology, 27*(2), 210-216. PMID 16310252. doi:10.1016/j.neuro.2005.10.001

Xu, F. (2005). *Occupational particle exposure assessment in a pharmaceutical facility using video exposure monitoring system* (Doctoral dissertation). Abstract retrieved from ProQuest Dissertations and Theses database. (AAT 3287407)

Xue, F., Holzman, C., Rahbar, M. H., Trosko, K., & Fischer, L. (2007, January). Maternal Fish Consumption, Mercury Levels and Risk of Preterm Delivery. *Environmental Health Perspectives, 115*(1), 42-47. PMID 17366817

Yang, C.-Y., Wang, Y.-J., Chen, P.-C., Tsai, S.-J., & Guo, Y. L. (2008, May). Exposure to a Mixture of Polychlorinated Biphenyls and Polychlorinated Dibenzofurans Resulted in a Prolonged Time to Pregnancy in Women. *Environmental Health Perspectives, 116*(5), 599-604. PMID 18470317. doi:10.1289/ehp.10715

Yang, Q.-H., Carter, H. K., Mulinare, J., Berry, R. J., Friedman, J. M., & Erickson, J. D. (2007, May). Race-ethnicity differences in folic acid intake in women of childbearing age in the United States after folic acid fortification: findings from the National Health and Nutrition Examination Survey, 2001-2002. *The American Journal of Clinical Nutrition, 85*(5), 1409-1416. PMID 17490980

Yasuda, Y., Matsuyama, A., Yasutake, A., Yamaguchi, M., Aramaki, R., Xiaojie, L., … Liya, Q. (2004, March). Mercury Distribution in Farmlands Downstream from an Acetaldehyde Producing Chemical Company in Qungzhen City, Guizhou, People's Republic of China. *Bulletin of Environmental Contamination Toxicology, 72*(3), 445-451. PMID 15114441

Yong, R. N. (2001). *Geoenvironmental Engineering*. Boca Raton, FL: CRC Press

Yoshida, M. (1985, February). Relation of mercury exposure to elemental mercury levels in the urine and blood. *Scandinavian Journal of Work, Environment and Health, 11*(1), 33-37. PMID 3992219

Young, G. (1983). Human Ecology. In M. Allaby (Ed.), *A Dictionary of the Environment*, (2nd ed., pp. 339-342). New York, NY: New York University Press

Young, J. G., Eskenazi, B., Gladstone, E. A., Bradman, A., Pedersen, L., Johnson, C., … Holland, N. T. (2005). Association Between *in utero* Organophosphate Pesticide Exposure and Abnormal Reflexes in Neonates. *NeuroToxicology, 26*, 199-209. PMID 15713341. doi:10.1016/j.neuro.2004.10.004

Youngstrom, E., LaKind, J., Kenworthy, L., Lipkin, P., Goodman, M., Squibb, K., … Gutermuth Anthony, L. (2010 January). Advancing the selection of neurodevelopmental measures in epidemiological studies of environmental chemical exposure and health effects. *International Journal of Environmental Research and Public Health, 7*(1), 229-268. PMID 20195443. doi:10.3390/ijerph7010229

Yu, C. H., Yiin, L. M., Tina Fan, Z. H., & Rhoads, G. G. (2009, January). Evaluation of HEPA vacuum cleaning and dry steam cleaning in reducing levels of polycyclic aromatic hydrocarbons and house dust mite allergens in carpets. *Journal of Environmental Monitoring, 11*(1), 205-211. PMID 19137159. doi:10.1039/b807821a

Zajac, L., Sprecher, E., Landrigan, P., & Trasande, L. (2009). A systematic review of U.S. state environmental legislation and regulation with regard to the

prevention of neurodevelopmental disabilities and asthma. *Environmental Health, 8*(9), 1-37. PMID 19323818. doi:10.1186/1476-069X-8-9

Zajacova, A., & Hummer, R. A. (2009, August). Gender differences in education effects on all-cause mortality for white and black adults in the United States. *Social Science & Medicine, 69*(4), 529-537. PMID 19589633. doi:10.1016/j.socscimed.2009.06.028

Zarnescu, O. (2009). Tracing the accumulation and effects of mercury uptake in the previtellogenic ovary of crucian carp, *Carassius auratus gibelio* by autometallography and caspase-3 immunohistochemistry. *Histology & Histopathology, 24*, 141-148. PMID 19085830

Zartarian, V. G., Bahadori, T., & McKone, T. (2005, January). Adoption of an official ISEA glossary. *Journal of Exposure Analysis and Environmental Epidemiology, 15*(1), 1-5. PMID 15562291. doi:10.1038/sj.jea.7500411

Zartarian, V. G., Ott, W. R., & Duan, N. (1997, October-December). A Quantitative Definition of Exposure and Related Concepts. *Journal of Exposure Analysis and Environmental Epidemiology, 7*(4), 411-437. PMID 9306230

Zartarian, V. G., Ott, W. R., & Duan, N. (2007). Basic Concepts and Definitions of Exposure and Dose. In W. R. Ott, A. C. Steinemann, & L. A. Wallace (Eds.). *Exposure Analysis* (pp. 33-63). Boca Raton, FL: CRC Taylor & Francis Group

Zartarian, V. G., Ozkaynak, H., Burke, J. M., Zufall, M. J., Rigas, M. L., & Furtaw, E. J. (2000, June). A Modeling Framework for Estimating Children's Residential Exposure and Dose to Chlorpyrifos via Dermal Residue Contact and Non-Dietary Ingestion. *Environmental Health Perspectives, 108*(6), 505-514. PMID 10856023

Zawia, N., & Riyaz Basha, M. (2005). Environmental Risk Factors and the Developmental Basis for Alzheimer's Disease. *Reviews in the Neurosciences, 16*(4), 325-337. PMID 16519009

Zeni, M., & Kogan, M. D. (2007, January-February). Existing population-based health databases: useful resources for nursing research. *Nursing Outlook, 55*(1), 20-30. PMID 17289464. doi:10.1016/j.outlook.2006.09.007

Zhang, A., Park, S. K., Wright, R., Weisskopf, M., Mujherjee, B., Nie, H., … Hu, H. (2010, September). HFE H63D Polymorphism as a Modifier of the Effect of Cumulative Lead Exposure on Pulse Pressure: The Normative Aging Study. *Environmental Health Perspectives, 118*(9), 1261-1266. PMID 20478760. doi:10.1289/ehp.1002251

Zhang, H., Feng, X., Larssen, T., Qiu, G., & Vogt, R. (2010, September). In inland China, rice, rather than fish, is the major pathway for methylmercury exposure. *Environmental Health Perspectives, 118*(9), 1183-1188. PMID 20378486. doi:10.1289/ehp.1001915

Zhang, Y., Wise, J. P., Holford, T. R., Xie, H., Boyle, P., Zahm, S. H., … Zheng, T. (2004, December 15). Serum Polychlorinated Biphenyls, Cytochrome P-450 1A1 Polymorphisms, and Risk of Breast Cancer in Connecticut Women. *American Journal of Epidemiology, 160*(12), 1177-1183. PMID 15583370

Zhang, Z.-W., Shimbo, S., Ochi, N., Eguchi, M., Watanabe, T., Moon, C.-S., & Ikeda, M. (1997, October). Determination of lead and cadmium in food and blood by inductively coupled plasma mass spectrometry: a comparison with graphite furnace atomic absorption spectrometry. *Science of the Total Environment, 205*(2-3), 179-187. PMID 9372629. doi:10.1016/S0048-9697(97)00197-6

Zhao, G., Xu, Y., Han, G., & Ling, B. (2006a, August). Biotransfer of persistent organic pollutants from a large site in China used for the disassembly of electronic and electrical waste. *Environmental Geochemistry and Health, 28*(4), 341-351. PMID 16724243. doi:10.1007/s10653-005-9003-3

Zhao, Q., Gadagbui, B., & Dourson, M. (2005). Lower birth weight as a critical effect of chlorpyrifos: A comparison of human and animal data. *Regulatory Toxicology and Pharmacology, 42*, 55-63. PMID 15896443. doi:10.1016/j.yrtph.2005.01.009

Zhao, Q., Dourson, M., & Gadagbui, B. (2006b, March). A review of the reference dose for chlorpyrifos. *Regulatory Toxicology and Pharmacology, 44*(2), 111-124. PMID 16360256. doi:10.1016/j.yrtph.2005.10.003

Zhao, Y., & Malyon, R. (2009, September). Life Years at Risk: A Population Health Measure from a Prevention Perspective. *International Journal of Environmental Research and Public Health, 6*(9), 2387-2396. PMID 19826550. doi:10.3390/ijerph6092387

Zhong, H., & Schwartz, J. (2010, August). Exploring gender-specific trends in underage drinking across adolescent age groups and measure of drinking: is girls' drinking catching up with boys'? *Journal of Youth and Adolescence, 39*(8), 911-926. PMID 20596818. doi:10.1007/s10964-009-9413-0

Zhu, M., Fitzgerald, E., Gelberg, K., Lin, S., & Druschel, C. (2010, October). Maternal Low-Level Lead Exposure and Fetal Growth. *Environmental Health Perspectives, 118*(10), 1471-1475. PMID 20562053. doi:10.1289/ehp.0901561

Zou, C., Zhao, Z., Tang, L., Chen, Z., & Du, L. (2003, April). The effect of lead on brainstem auditory evoked potentials in children. *Chinese Medical Journal, 116*(4), 565-568. PMID 12875723