America's Children and the Environment

Third Edition

January 25, 2013

I am pleased to present the U.S. Environmental Protection Agency's *America's Children and the Environment, Third Edition*. This report marks the important progress we have made as a nation to reduce environmental risks to children's health.

The report contains good news for children and families including significant improvements in the quality of the air we breathe, substantial decreases in childhood blood lead levels, and a steady reduction in children's exposure to secondhand smoke. We are encouraged by these findings. We also know that there is still much work to be done, including further research on the causes of increases in asthma rates, the potential impacts of early life exposures to chemicals, and disease disparities in minority children and children in low-income families. *America's Children and the Environment* will help focus our efforts in addressing these challenges and others.

Protecting children's health is central to the EPA's mission, and the agency has taken great strides to improve the environment for children where they live, learn, and play, including:

- **Finalizing the Mercury and Air-Toxics Standards Rule** to limit mercury and other air toxics emissions from electric generating utilities. These new standards address the largest remaining domestic source of mercury emissions to the environment—a well known neurotoxin in children. The controls put in place by these standards will also avoid 130,000 asthma attacks every year—which disproportionately impact children especially in underserved communities.
- **Updating the National Ambient Air Quality Standard for fine-particle pollution ($PM_{2.5}$)** to improve public health protection. Exposure to $PM_{2.5}$ can aggravate asthma and lead to other respiratory symptoms in children.
- **Establishment of new National Ambient Air-Quality Standards for nitrogen dioxide (NO_2) and sulfur dioxide (SO_2)**, and a network of monitors to limit near roadway exposures to NO_2. These new standards will limit respiratory-related emergency room visits and hospital admissions and will improve public health protection, especially for children, the elderly, and people with asthma.
- **Supporting cutting-edge research through the Centers for Children's Environmental Health and Disease Prevention Research**, in partnership with the National Institute of Environmental Health Sciences, to enhance scientific understanding of the relationships between environmental contaminants and children's health.
- **Launching new voluntary guidelines** that promote environmentally safe siting of schools and the establishment of school environmental health programs by states.
- **Working with other federal agencies to develop and implement the Coordinated Federal Action Plan to Reduce Racial and Ethnic Asthma**

Disparities to reduce the disproportionate impact of asthma on minority and low-income children.

As we move forward, the EPA is committed to continuing the success of our children's health efforts. The national indicators presented in this comprehensive report are important for informing future research related to children's health. We will continue to partner with other federal agencies to develop increasingly reliable information that will help us to further improve children's health.

I want to thank the many individuals who contributed to this report for their hard work and efforts. By monitoring trends, identifying successes, and shedding light on areas of concern, we can continue to improve the health of our children and all Americans.



Lisa P. Jackson

Administrator

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About this Report

What is *America's Children and the Environment*?

America's Children and the Environment (ACE) is EPA's report presenting data on children's environmental health. ACE brings together information from a variety of sources to provide national indicators in the following areas: Environments and Contaminants, Biomonitoring, and Health. Environments and Contaminants indicators describe conditions in the environment, such as levels of air pollution. Biomonitoring indicators include contaminants measured in the bodies of children and women of child-bearing age, such as children's blood lead levels. Health indicators report the rates at which selected health outcomes occur among U.S. children, such as the annual percentage of children who currently have asthma. Accompanying each indicator is text discussing the relevance of the issue to children's environmental health and describing the data used in preparing the indicator. Wherever possible, the indicators are based on data sources that are updated in a consistent manner, so that indicator values may be compared over time.

This report is the third edition of ACE (referred to as ACE3); previous editions of ACE were published in 2000 and 2003. EPA has provided updated indicator values on its website on a regular basis beginning in 2006, and will provide online updates for the indicators published in this edition (see www.epa.gov/ace).

What are the purposes of *America's Children and the Environment*?

America's Children and the Environment has three principal objectives:

- First, it compiles data from a variety of sources to present concrete, quantifiable indicators for key factors relevant to the environment and children's health in the United States.
- Second, it can inform discussions among policymakers and the public about how to improve data on children's health and the environment.
- Third, it includes indicators that can be used by policymakers and the public to track trends in children's environmental health, and ultimately to help identify and evaluate ways to minimize environmental impacts on children.

This report is motivated by EPA's belief that it is valuable to be aware of, and to share with the public, information on trends in children's environmental health. The purpose of ACE is to compile information, and make it available to a broad audience, that can help identify areas that warrant additional attention, potential issues of concern, and persistent problems. Some of the indicators can also support efforts to evaluate whether past environmental policies and actions have been effective. EPA hopes that the development and presentation of these indicators will motivate continuing research, additional data collection, and, when appropriate, necessary interventions.

The information in ACE3 is not intended to serve as a definitive basis for planning specific policies or projects. EPA and other federal agencies rely on a wide range of technical information to inform their activities on children's environmental health. Emerging and ongoing research will help shape these efforts for years to come. The presentation of findings from the scientific literature in ACE3 is not intended to constitute an authoritative summary or conclusion on the weight of scientific evidence.

What are children's environmental health indicators?

For ACE3, an indicator is defined as a quantitative depiction of an aspect of children's environmental health that summarizes the underlying data in a relevant, understandable, and technically appropriate manner. The data may represent measurements of environmental conditions, of chemicals measured in the bodies of children and women of child-bearing age, or of the frequency of certain childhood diseases and health outcomes. Federal data on children's environmental health issues come from a variety of agencies and are often very detailed and complex; ACE brings this information together into one report and summarizes the data in graphics that convey the key information. The ACE indicators generally focus on presenting data at the national scale in order to meet the three principal objectives described above.

Many indicators in this report provide a time series of data (i.e., a "trend" graph), to evaluate whether conditions have changed over time. Other indicators provide a "snapshot" that focuses on data from a single time period. These indicators may depict differences in conditions for different population groups (defined by race/ethnicity or income), or they may provide data for different children's health hazards for a single time period.

The World Health Organization defines environmental health as "all the physical, chemical, and biological factors external to a person, and all the related factors impacting behaviors. It encompasses the assessment and control of those environmental factors that can potentially affect health."[1] In concordance with this definition, use of the term "children's environmental health" in ACE3 refers to external physical, chemical, and biological factors that are known to affect children's health or may potentially affect children's health. The evidence of a relationship between environmental exposure and children's health continues to evolve for many of the indicators presented in this report. Inclusion of an indicator in this report does not necessarily imply a known relationship between environmental exposure and children's health effect. EPA aims to develop increasingly informative indicators of children's environmental health as more data become available to reduce these uncertainties.

The ACE3 indicators are intended to be easy to understand and to cover a broad range of topics. More extensive analyses are available for most of the datasets featured by ACE3 indicators; links to the associated studies and reports will be provided on the ACE website.

Although the ACE3 indicators focus on national statistics, environmental exposures and health may vary significantly across communities. Patterns of environmental exposure may vary due to the nature and extent of pollutants found in each community. Patterns of health may vary

across communities due to demographic and socioeconomic characteristics. Links to online resources with community-level information will be provided on the ACE website.

Why did EPA focus on indicators for children?

Under Executive Order 13045, EPA and other federal agencies are directed to "make it a high priority to identify and assess environmental health risks and safety risks that may disproportionately affect children."[2] Environmental contaminants can affect children quite differently than adults, both because children may be more highly exposed to contaminants, and because they are often more vulnerable to the toxic effects of contaminants.

Children generally eat more food, drink more water, and breathe more air relative to their size than adults do, and consequently may be exposed to relatively higher amounts of environmental chemicals. Children's normal activities, such as putting their hands in their mouths or playing on the ground, can result in exposures to chemicals that adults do not face. In addition, some environmental contaminants may affect children disproportionately because their bodies are not fully developed and their growing organs can be more easily harmed.

How is *America's Children and the Environment* organized?

After this introduction, ACE3 features four main sections: Environments and Contaminants, Biomonitoring, Health, and Supplementary Topics. Each section presents information on a series of children's environmental health topics, and at least one indicator is provided for each topic.

The Environments and Contaminants section presents information on chemicals and pollutants in environmental media to which children are commonly exposed (through air, drinking water, and food), along with other important aspects of children's environments. Topics addressed in the Environments and Contaminants section include criteria air pollutants, hazardous air pollutants, indoor environments, drinking water contaminants, chemicals in food, contaminated lands, and climate change.

The Biomonitoring section presents information on selected chemicals measured in the blood and urine of children and women of child-bearing age. Biomonitoring indicators for women ages 16 to 49 years are included based on concern for potential adverse health effects in children born to women who have been exposed to certain chemicals. Topics addressed in the Biomonitoring section include lead, mercury, cotinine (a marker for exposure to environmental tobacco smoke), perfluorochemicals (PFCs), polychlorinated biphenyls (PCBs), polybrominated diphenyl ethers (PBDEs), phthalates, bisphenol A (BPA), and perchlorate.

The Health section presents information on diseases, conditions, and outcomes that may be influenced by environmental exposures. Many factors contribute to children's health, including genetic inheritance, nutrition, and exercise, among others. The adverse health consequences of some environmental exposures may occur through interactions with other risk factors, and it is

often difficult to determine the extent to which the environment (or any other factor) contributes to children's health outcomes of concern. Topics addressed in the Health section include respiratory diseases, childhood cancer, neurodevelopmental disorders, obesity, and adverse birth outcomes.

The Supplementary Topics section presents topics for which adequate national data are not available, but for which more targeted data collection efforts could be used to provide measures illustrating additional children's environmental health issues of interest. Data sets used for these measures are representative of particular locations (such as a single state) and/or were surveys conducted a single time rather than on a continuing or periodic basis. Since these data sets are lacking in certain key elements desirable for ACE3 indicators, data presentations for the Supplementary Topics are referred to as "measures" rather than "indicators." Topics addressed in this section include birth defects in Texas and contaminants in schools and child care facilities.

How were the topics and indicators in the third edition of *America's Children and the Environment* selected?

In choosing indicators for ACE3, EPA considered a variety of factors, including public interest, magnitude of prevalence and/or trend in prevalence, extent of exposure, severity of health outcome, past EPA actions to address the issue, and research findings indicating or suggesting that an environmental exposure may contribute to a children's health outcome. ACE3 includes topics for which scientific evidence is sufficient to conclude there is a causal relationship between exposure and health effects, as well as topics for which there is less extensive scientific evidence. Inclusion of a topic in ACE3, therefore, does not imply that a cause-effect determination has been made.[i]

ACE3 includes updates and revisions to topics and indicators included in the 2003 ACE report, as well as new topics and indicators developed for this edition. Although ACE3 addresses a substantially expanded set of children's environmental health topics compared with the 2003 edition of this report, it is not intended to be inclusive of all children's environmental health issues.

The selection of topics involved generating a list of children's environmental health issues of potential interest, evaluating availability of suitable databases relevant to those topics, and

[i] See "What is known about the role of the environment in contributing to adverse children's health outcomes?" below for more information.

considering indicators that might be derived from those databases. EPA obtained input from members of EPA's Children's Health Protection Advisory Committee (CHPAC) on each stage of this process, including input on the ultimate selection of topics and indicators presented in ACE3.[3,4] Independent external peer reviewers provided their opinions to EPA regarding the suitability of the indicators and other information provided for each topic. EPA revised the report based on the peer review comments, and comments received from the public.

Selection of a topic for inclusion in ACE depended, in part, on whether data appropriate for indicator development were available. Available databases were considered in the context of the following:

- Relevance to the topic of interest.
- Degree to which scientifically sound data collection methodologies and quality assurance procedures were used.
- Availability of data documentation.
- Availability of raw data (individual measurements or survey responses).
- Degree to which the database can be used to characterize national patterns.
- Ongoing (continuous or periodic) data collection, with relatively recent data available.
- Comparability of target population, sample selection, and data collection methods across time.
- Ability to stratify data by race/ethnicity, income, and location (region, state, county, or other geographic unit).

The suitability of each database was determined through an overall weighing of these considerations. Some databases ranked comparatively better than others with respect to each of these considerations. For example, some databases contain the results of nationally representative surveys that cannot be stratified geographically but are excellent in other respects; inability to extract statistics for regions, states, or other geographic divisions does not preclude the use of these databases in ACE3. Similarly, some monitoring data sets are not explicitly designed to be nationally representative; however, they may still be informative as long as their limitations are understood.

ACE3 presents one or more indicators to illustrate status and/or trends for each selected topic with a suitable database. In some cases, a topic is represented with multiple indicators that portray different aspects of the underlying data or make use of different types of data.

Considerations that EPA used in developing specific indicators from each selected database include some of the same factors used in selecting the database, as well as others, including:

- Utility of the indicator for portraying some aspect of children's environmental health.
- Sensitivity to changes in the condition of interest.
- Robustness (unaffected by changes in factors not relevant to the condition of interest).

- Degree to which the indicator offers an appropriate summary of the underlying data.
- Ability to be presented as population-based statistic (for example, the indicator takes the form of "percentage of children affected," or as defined points in the population. distribution of values, such as medians), particularly a national population-based statistic.
- Clarity: Indicators that do not satisfy all considerations may still be considered suitable; for example, some indicators may lack data for presenting a trend over a number of years, but present useful information for some relatively recent time period (a single year or set of years). To help guide reader evaluations, text boxes are provided that summarize the characteristics of the data used for each indicator.

What are the sources of data for the indicators in *America's Children and the Environment*?

Federal agencies provided the data for most of the indicators. Data for the Environments and Contaminants indicators are generally from data systems maintained by EPA and by state environmental agencies. Data on indoor lead hazards are from surveys conducted by the U.S. Department of Housing and Urban Development. Pesticide residue data are from the Pesticide Data Program of the U.S. Department of Agriculture. Health and biomonitoring data are from the National Center for Health Statistics in the Centers for Disease Control and Prevention. Cancer data are from the National Cancer Institute. Child population data from the Census Bureau were used for calculations in several of the Environments and Contaminants indicators.

Data for the Supplementary Topics measures are from more targeted data collection efforts that illustrate some aspect of a children's environmental health issue of interest in the absence of a more comprehensive data source. Childcare facility measures are derived from a national study, and a study performed in North Carolina and Ohio. For schools, a measure on indoor pesticide application is derived from data reported by California schools and collected by the California Department of Pesticide Regulation. The data on birth defects are from the Texas birth defects monitoring program. Data from individual states are not intended to describe national conditions or conditions in other states.

What years are included in the *America's Children and the Environment* indicators?

ACE3 aims to include indicators that present trends over at least the past 10 years; however, for some indicators, data are not available for this length of time. When sufficient data are not available to show changes over time, indicators present the most current data available, frequently focusing the presentation on demographic comparisons of race/ethnicity and income. Some topics include both a trend indicator and a separate indicator with demographic comparisons using current data.

All ACE3 indicators incorporate the most current data that were available at the time of analysis. For some indicators, additional data were released prior to the publication of ACE3. Newer data will be incorporated for the indicator updates provided online at www.epa.gov/ace.

What groups of children are included in the *America's Children and the Environment* indicators?

Census Bureau data indicate that there were 74.2 million children ages 17 years and younger in the United States in 2010. The age range used for each indicator depends on data availability and the nature of the topic being addressed. Each indicator clearly identifies the age range in the title of the figure.

ACE3 presents (where possible) indicators for groups of children of different races and ethnicities and for children living in households with various levels of income. In some cases, these breakouts by race/ethnicity and family income are shown in the graphs, while in other cases they are included in the data tables.

The specific race/ethnicity categories used for each indicator depend on the underlying data source, and are further discussed in the introduction to each section of the report.

Many of the indicators also provide separate indicator values for children living in homes with family income below the poverty level (shown in graphs and tables as < Poverty) and those in homes at or above the poverty level (≥ Poverty). "Poverty level" is defined by the federal government and is based on income thresholds that vary by family size and composition. In 2010, for example, the poverty threshold was $22,113 for a household with two adults and two related children.[5] Based on this federal definition, 22% of children were living below poverty level in 2010, an increase from 18% of children in 2007.[6]

Why does *America's Children and the Environment* compare indicator values by race/ethnicity and income?

Under Executive Order 12898,[7] EPA (along with other federal agencies) is directed to "make achieving environmental justice part of its mission by identifying and addressing, as appropriate, disproportionately high and adverse human health or environmental effects of its programs, policies, and activities on minority populations and low-income populations." Comparing indicator values across these demographic groups helps identify differences in the distributions of exposures and health outcomes, which are factors in investigating the potential for disproportionate impacts.

Comparing indicator values by demographic groups is also in keeping with the goals of Healthy People 2020, the federal government's program of objectives for improving the health of all Americans. Among the overarching goals of Healthy People 2020 are to "achieve health equity, eliminate disparities, and improve the health of all groups" and to "create social and physical environments that promote good health for all."[8] Healthy People 2020 defines a health disparity

as "a particular type of health difference that is closely linked with social, economic, and/or environmental disadvantage. Health disparities adversely affect groups of people who have systematically experienced greater obstacles to health based on their racial or ethnic group; religion; socioeconomic status; gender; age; mental health; cognitive, sensory, or physical disability; sexual orientation or gender identity; geographic location; or other characteristics historically linked to discrimination or exclusion."[9] Presentation of ACE indicator values by race/ethnicity and income groups provides information useful for investigating possible health disparities and possible environmental contributors to health disparities. Additionally, EPA's regulations implementing Title VI of the Civil Rights Act state, in part, "No person shall be excluded from participation in, be denied the benefits of, or be subjected to discrimination under any program or activity receiving EPA assistance on the basis of race, color, [or] national origin" (40 C.F.R. 7.30). Where comparison data are available on a state-specific basis, it may help EPA and its assistance recipients (for example, state environmental agencies) assess whether discriminatory impacts are occurring.

What information is presented for each topic and indicator?

Presentation of each topic includes a discussion of the scope of the issue and a brief snapshot of the relevant scientific literature regarding associations between exposures and health effects. If an authoritative source has published conclusions regarding the strength of evidence relevant to the topic, such as a determination of a cause-effect relationship between exposure and outcome, these findings are summarized. In the absence of such a source, the discussion describes selected literature and highlights significant sources of uncertainty, but this review should not be considered either an evaluation of the available literature or a statement regarding the strength of the evidence.

This is followed by an explanation of the indicator chosen to represent the topic, including a discussion of the data source, a description of the data provided in the indicator, and information to aid in interpreting the indicator, including data limitations.

Following this background text, one or more indicators are provided. Each indicator is presented in a figure. A text box is provided to help readers understand the characteristics of the data displayed. Bullet points that highlight key data points from the indicator are included.

Appendices to the report provide data tables for each indicator. Detailed explanations of the methods for calculating each indicator are provided in the online materials available at www.epa.gov/ace.

What is known about the role of environmental exposures in contributing to adverse children's health outcomes?

Some environmental exposures have a well-established cause-effect relationship with children's health, such as effects of lead exposure on childhood IQ and effects of certain air pollutants on respiratory outcomes. For some other environmental exposures, there is

evidence suggestive of a relationship to children's health outcomes but not enough evidence to conclude the existence of a cause-effect relationship; and for many other environmental exposures there is very little information on potential health consequences of the exposure levels typically experienced by children in the United States. Furthermore, for many of the children's health effects discussed in this report, our scientific knowledge regarding causes is somewhat limited.

A major focus of environmental health research is to expand our understanding of the possible role of environmental contaminant exposures, as well as other environmental risk factors, in childhood diseases and disorders. Research is increasingly pointing to interactions of genetic factors and environmental factors as critical to the process for most diseases.

Even when a clear relationship between exposure to a particular hazardous environmental contaminant or factor has been documented, some children will have worse outcomes and others will be unaffected or have outcomes that are less severe. Exposure characteristics—such as the length of exposure, the magnitude of the exposure, the route of exposure and the developmental stage when a child is exposed—explain much of the variation in outcome. However, genetic variability in the population can mean that individuals vary greatly in how their body metabolizes a chemical and in their susceptibility to diseases that may result from an environmental exposure. In addition, variability in concurrent or prior exposures to other environmental contaminants and to non-chemical stressors can also lead to substantial variability of outcomes within an exposed population.[10]

A child's genetic inheritance can often play an important role in disease. However, as scientific methods for examining the role of genetics in disease have advanced, it has become clear that much of human chronic disease cannot be explained by genetic factors alone, and that environmental factors (broadly defined to include nutrition, exercise, exposures to environmental contaminants, and other factors) and their interactions with genetic factors also play an important role in chronic disease.[11-13]

The effects of an environmental exposure on children's health often depend on the developmental stage at which the exposure occurs. Different organ systems in a child's body go through critical developmental stages at different times, from conception through the entire period of fetal development, in infancy and early childhood, and continuing through adolescence. Some chemical exposures can result in adverse effects if they occur during a particular critical window, and may have different effects or no effect at all when occurring at a different stage of development.[14] For this reason, even some environmental exposures to adults can be important for children's health, as research has found that the prenatal period is the most sensitive developmental stage for adverse effects of some chemicals.[15] In some cases, the effects of a harmful exposure may not become evident until many years later; exposure during early developmental stages may even contribute to the onset of chronic diseases in adulthood.[15]

What types of scientific studies provide evidence about the potential relationships between environmental exposures and children's health outcomes?

Developing conclusive evidence that environmental factors cause or contribute to the incidence of childhood health effects is difficult. Many health outcomes are hypothesized to be multi-factorial, with contributions from genetics, underlying health conditions, and lifestyle, as well as the social and physical environment. Scientific evidence linking the environment to health outcomes consists primarily of laboratory assays, experimental studies in animals, and epidemiological studies in humans. Each of these methods has limitations, but together they can provide complementary evidence in assessing how exposures can influence the development of health outcomes.

A major advantage of animal studies is that they are controlled experiments in which exposures are imposed upon the study subjects and all other variables are held constant. In many cases, animals can provide good models of human physiological systems and thus indicate how humans might respond to exposures. However, it is not always straightforward to interpret findings of animal studies and their meaning or importance for human health. Furthermore, animal studies are often conducted using exposure levels much greater than those typically experienced by humans, and some uncertainty exists as to whether the same effects would be seen at lower exposure levels.

In contrast, observational epidemiological studies are advantageous because they evaluate the relationship between environmental conditions and health outcomes in exposed human populations. Since this type of study is not a controlled experiment, there may be factors related to both the exposure and the health effect in the study population that can create false associations, or mask true associations, between the exposure and the health effect. Observational epidemiological studies provide the strongest evidence when they have been replicated in multiple populations to minimize the likelihood that an association between exposure and health outcomes occurred due to something other than a true causal relationship.

Some epidemiological studies are conducted in samples of the U.S. general population, or in other countries with similar exposure levels, and thus reflect exposures that occur on a routine basis. Sometimes studies in the United States or in other countries may be focused on communities that experience higher exposure levels than the rest of the country; these studies would be considered to have greater-than-average exposures but are still within the range of exposures occurring in the United States. In other cases, exposures in epidemiological studies are conducted in populations with substantially higher exposure, such as workers exposed to chemicals on the job, populations in other countries that have higher levels of pollution, residents of communities where disasters or accidental poisoning incidents have occurred, or populations in the United States or other industrialized countries before environmental protection efforts to reduce exposure occurred. In such cases, some uncertainty exists as to whether the same effects would be seen at lower exposure levels observed today.

An important additional consideration is the extent to which toxicological or epidemiological studies are available for environmental contaminants and chemicals in commerce. For many environmental exposures of interest, the epidemiological research is very limited and there are significant gaps in the available animal testing data.

How are the findings of scientific studies regarding children's health and the environment represented in *America's Children and the Environment*?

The level of knowledge regarding the relationship between environmental exposures and health outcomes varies widely among the topics presented in this report. Some associations between contaminants and health outcomes are supported by a large body of consistent evidence from rigorously designed and conducted studies. In other cases, research findings may suggest reason for potential concern but may not be sufficient to draw conclusions regarding the nature or strength of the relationship between an environmental contaminant exposure and a children's health outcome.

Where available, ACE3 relies on authoritative reviews of the scientific literature and reports their conclusions regarding the strength of the evidence for a causal role of specific environmental factors in the development of childhood diseases and disorders. Examples of authoritative sources are the National Research Council, the Institute of Medicine, and the National Toxicology Program.[ii] When such reviews are unavailable, selected findings from the epidemiological literature that address the potential role of environmental factors in contributing to an effect are summarized. Literature on animal studies is discussed in certain cases when epidemiological data are lacking. These reviews of scientific information are intended to summarize the concerns that have led to inclusion of the topic in this report. The literature summaries are not intended as reviews of the literature determining the strength of the evidence, which is an undertaking beyond the scope of this report.

How are children's environmental health indicators different from epidemiological research?

The presentation of children's environmental health indicators in ACE3 is intended to highlight issues of interest, describe indicator values over time, and describe indicator values for different demographic groups. However, the indicators themselves are not intended as a basis for reaching conclusions that an environmental factor is or is not related to a particular

[ii] The National Research Council and the Institute of Medicine are private, nonprofit institutions that provide expert advice on science and health matters. The National Toxicology Program, part of the U.S. Department of Health and Human Services, provides evaluations of substances of public health concern.

children's health outcome. Comparison of trends in Health indicators to trends in Environments and Contaminants or Biomonitoring indicators may suggest hypotheses for further research, but their presentation here cannot be used to conclude that a causal relationship may or may not exist. Indicators cannot account for the multiple factors that should inform these judgments.

Epidemiological studies can be designed that consider both individual-level or community-level exposures (or surrogates for exposures) and outcomes within the same population, along with other factors such as the timing of exposure relative to the timing of outcome, related variables that could influence the health outcome, and appropriate statistical models of a hypothesized relationship.

ACE3 indicators do not incorporate these factors, and thus are not intended as a basis for conclusions about associations between exposures and outcomes. Rather, the value of indicators is in their ability to reveal trends (or absence of trends) and variations (or lack of variation) within the population, which can then be used to identify areas for closer review. Since they are based on ongoing data collection programs, the indicators can be updated regularly and can be used to alert policy makers and the public when unexpected patterns emerge from new data, or to provide an indication of whether recently adopted exposure reduction interventions and actions are having an impact.

What is the difference between this report and an EPA risk assessment?

Human health risk assessment is the process used to estimate the nature and probability of adverse health effects in populations who may be exposed to chemicals. A risk assessment typically focuses in depth on a particular environmental contaminant to identify potential adverse health outcomes, likely exposure pathways, the estimated magnitude of exposure, and the likelihood of health outcomes occurring at different levels of exposure experienced by a population.

The indicators in this report do not constitute a risk assessment. The indicators present observed data on status and trends in environmental conditions, biomonitoring, and health outcomes; they do not attempt to provide the information relating exposures and outcomes provided in a risk assessment. The scope of a risk assessment involves a much more detailed examination of the health effects literature and of exposure data, including estimation of the relationship between particular levels of exposure and potential outcomes. The indicators in this report should not be construed to indicate the level of risk to children's health from exposures to environmental contaminants.

More information about risk assessment may be found at EPA's Risk Assessment Portal at http://www.epa.gov/risk_assessment/.

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Key Findings

These Key Findings summarize the observations obtained from each of the indicators presented in this report. Statistically significant trends or differences are identified by the terms "increase," "decrease," "higher," or "lower." Please see the body of the report for background helpful in understanding and interpreting each of these findings, including definitions, descriptions of data sets, and summaries of relevant scientific findings. The years for which data are available varies across the indicators.

The evidence of a relationship between environmental exposure and children's health continues to evolve for many of the indicators presented in this report. Inclusion of an indicator in this report does not necessarily imply a known relationship between environmental exposure and children's health effect.

Environments and Contaminants

Criteria Air Pollutants

- From 1999 to 2009, the proportion of children living in counties with measured pollutant concentrations above the levels of one or more national ambient air quality standards decreased from 75% to 59%. This includes both concentrations above the level of any current short-term standard at least once during the year as well as average concentrations above the level of any current long-term standards.
- In 2009, 6% of children lived in counties with measured ozone concentrations above the level of the 8-hour ozone standard on more than 25 days. An additional 3% of children lived in counties with measured concentrations above the level of the ozone standard between 11 and 25 days, and 12% of children lived in counties where concentrations were above the level of the standard between 4 and 10 days.
- In 2009, 1% of children lived in counties with measured fine particle ($PM_{2.5}$) concentrations above the level of the 24-hour $PM_{2.5}$ standard on more than 25 days. An additional 2% of children lived in counties with measured concentrations above the level of this standard between 11 and 25 days, and 1% of children lived in counties with measured concentrations above the level of the 24-hour $PM_{2.5}$ standard between 8 and 10 days.
- Based on categories from EPA's Air Quality Index, the percentage of children's days that were designated as having "unhealthy" air quality decreased from 9% in 1999 to 3% in 2009. The percentage of children's days with "good" air quality increased from 41% in 1999 to 57% in 2009. The percentage of children's days with "moderate" air quality was approximately constant at 21–23% from 1999 to 2007, and then decreased to 16% in 2009.

Hazardous Air Pollutants

- In 2005, nearly all children (99.9%) lived in census tracts in which hazardous air pollutant (HAP) concentrations combined to exceed the 1-in-100,000 cancer risk benchmark. Seven percent of children lived in census tracts in which HAPs combined to exceed the 1-in-10,000 cancer risk benchmark. Fifty-six percent of children lived in census tracts in which at least one HAP exceeded the benchmark for health effects other than cancer.

Indoor Environments

- In 2010, 6% of children ages 0 to 6 years lived in homes where someone smoked regularly, compared with 27% in 1994.
- In 2005–2006, 15% of children ages 0 to 5 years lived in homes with either an interior lead dust hazard or an interior deteriorated lead-based paint hazard, compared with 22% in 1998–1999.

Drinking Water Contaminants

- The estimated percentage of children served by community drinking water systems that did not meet all applicable health-based standards was 19% in 1993 and about 5% in 2001. Since 2002, this percentage has fluctuated between 7% and 13%, with the most recent estimate being 7% in 2009.
- Between 1993 and 2009, the estimated percentage of children served by community water systems that had at least one monitoring and reporting violation fluctuated between about 11% and 23%, and was 13% in 2009.

Chemicals in Food

- In 1999, 81% of sampled apples had detectable organophosphate pesticide residues, and in 2009, 35% had detectable residues. In 2000, 10% of sampled carrots had detectable organophosphate pesticide residues, and in 2007, 5% had detectable residues. In 2000, 21% of sampled grapes had detectable organophosphate pesticide residues, and in 2009, 8% had detectable residues. In 1998, 37% of sampled tomatoes had detectable organophosphate pesticide residues, and in 2008, 9% had detectable residues.

Contaminated Lands

- As of 2009, approximately 6% of all children in the United States lived within one mile of a Corrective Action or Superfund site that may not have had all human health protective measures in place.
- Approximately 21% of all children living within one mile of a Corrective Action or Superfund site that may not have had all human health protective measures in place were Black, while 15% of children in the United States as a whole are Black.

Biomonitoring

Lead

- The median concentration of lead in the blood of children between the ages of 1 and 5 years dropped from 15 micrograms per deciliter (µg/dL) in 1976–1980 to 1.2 µg/dL in 2009–2010, a decrease of 92%. At the 95th percentile, blood lead levels dropped from 29 µg/dL in 1976–1980 to 3.4 µg/dL in 2009–2010, a decrease of 88%.
- The median blood lead level in Black non-Hispanic children ages 1 to 5 years in 2007–2010 was 1.6 µg/dL, higher than the level of 1.2 µg/dL in White non-Hispanic children, Mexican-American children, and children of "All Other Races/Ethnicities."

Mercury

- The median concentration of total mercury in the blood of women ages 16 to 49 years has shown little change between 1999–2000 and 2009–2010, and was 0.8 micrograms per liter (µg/L) in 2009–2010.
- Among women in the 95th percentile of exposure, the concentration of total mercury in blood decreased from 7.4 µg/L in 1999–2000 to 3.7 µg/L in 2001–2002. From 2001–2002 to 2009–2010, the 95th percentile of total blood mercury remained between 3.7 and 4.5 µg/L.

Cotinine

- The median level of cotinine (a marker of exposure to environmental tobacco smoke) measured in blood serum of nonsmoking children ages 3 to 17 years dropped from 0.25 nanograms per milliliter (ng/mL) in 1988–1991 to 0.03 ng/mL in 2009–2010, a decrease of 88%. Cotinine values at the 95th percentile decreased by 34% from 1988–1991 to 2009–2010.
- The median level of cotinine measured in blood serum of nonsmoking women ages 16 to 49 years dropped from 3.2 ng/mL in 1988–1991 to 2.1 ng/mL in 2009–2010, a decrease of 86%. Cotinine values at the 95th percentile decreased by 35% from 1988–1991 to 2009–2010.

Perfluorochemicals (PFCs)

- Between 1999–2000 and 2007–2008, median blood serum levels of perfluorooctane sulfonic acid (PFOS) and perfluorooctanoic acid (PFOA) in women ages 16 to 49 years showed a decreasing trend; median levels of perfluorononanoic acid (PFNA) showed an increasing trend; and median levels of perfluorohexane sulfonic acid (PFHxS) remained relatively constant over time.

Polychlorinated Biphenyls (PCBs)

- In 2001–2004, the median level of polychlorinated biphenyls (PCBs), summing together four selected PCBs, in blood serum of women ages 16 to 49 years was 30 nanograms per gram (ng/g) lipid. Data are not yet available for comparing these PCB levels over time.

Polybrominated Diphenyl Ethers (PBDEs)

- The median concentration of polybrominated diphenyl ethers (PBDEs) in blood serum of women ages 16 to 49 years was 44 ng/g lipid in 2003–2004. Data are not yet available for comparing these PBDE levels over time.

Phthalates

- From 2001–2002 to 2007–2008, the median level of di-2-ethylhexyl phthalate (DEHP) metabolites in urine of women ages 16 to 49 years varied between 41 µg/L and 51 µg/L, and was 51 µg/L in 2007–2008. From 1999–2000 to 2007–2008, the median level of dibutyl phthalate (DBP) metabolites in women ages 16 to 49 years varied between 27 µg/L and 36 µg/L, and was 36 µg/L in 2007–2008. From 1999–2000 to 2007–2008, the median level of

butyl benzyl phthalate (BBzP) metabolites in women ages 16 to 49 years varied between 10 µg/L and 14 µg/L, and was 12 µg/L in 2007–2008.

- From 2001–2002 to 2007–2008, the median level of DEHP metabolites in urine of children ages 6 to 17 years varied between 45 µg/L and 62 µg/L, and was 45 µg/L in 2007–2008. From 1999–2000 to 2007–2008, the median level of DBP metabolites in children ages 6 to 17 years varied between 36 µg/L and 42 µg/L, and was 41 µg/L in 2007–2008. The median level of BBzP metabolite in children ages 6 to 17 years decreased from 25 µg/L in 1999–2000 to 16 µg/L in 2007–2008.

Bisphenol A (BPA)

- From 2003–2004 to 2009–2010, the median concentration of bisphenol A (BPA) in urine among women ages 16 to 49 years varied between 2 µg/L and 3 µg/L. From 2003–2004 to 2009–2010, the concentration of BPA in urine at the 95th percentile varied between 10 µg/L and 16 µg/L, and was 10 µg/L in 2009–2010.
- Among children ages 6 to 17 years the median concentration of BPA in urine decreased from 4 µg/L in 2003–2004 to 2 µg/L in 2009–2010. The concentrations of BPA in urine at the 95th percentile decreased from 16 µg/L in 2003–2004 to 10 µg/L 2009–2010.

Perchlorate

- From 2001–2002 to 2007–2008, the median level of perchlorate in urine among women ages 16 to 49 years was 3 µg/L with little variation over time. Over the same period, the 95th percentile varied between 13 and 17 µg/L.

Health

Respiratory Diseases

- The proportion of children reported to currently have asthma has increased from 8.7% in 2001 to 9.4% in 2010.
- In 2007–2010, the percentages of Black non-Hispanic children and children of "All Other Races" reported to currently have asthma, 16.0% and 12.4% respectively, were greater than for White non-Hispanic children (8.2%), Hispanic children (7.9%), and Asian non-Hispanic children (6.8%).
- The rate of emergency room visits for asthma decreased from 114 visits per 10,000 children in 1996 to 103 visits per 10,000 children in 2008. Between 1996 and 2008, hospitalizations for asthma and for all other respiratory causes decreased from 90 hospitalizations per 10,000 children to 56 hospitalizations per 10,000 children.

Childhood Cancer

- The age-adjusted annual incidence of cancer increased from 1992–2009. The incidence ranged from 153 to 161 cases per million children between 1992 and 1994 and from 172 to 175 cases per million children between 2007 and 2009.

- Childhood cancer mortality has decreased from 33 deaths per million children in 1992 to 24 deaths per million children in 2009.
- Leukemia was the most common cancer diagnosis for children from 2004–2006, representing 28% of total cancer cases. Incidence of acute lymphoblastic (lymphocytic) leukemia increased from 30 cases per million in 1992–1994 to 35 cases per million in 2004–2006. The rate of acute myeloid (myelogenous) leukemia was 7 cases per million in 1992–1994 and 9 cases per million in 2004–2006.

Neurodevelopmental Disorders

- From 1997 to 2010, the proportion of children ages 5 to 17 years reported to have ever been diagnosed with attention-deficit/hyperactivity disorder (ADHD) increased from 6.3% to 9.5%.
- In 2010, 8.6% of children ages 5 to 17 years had ever been diagnosed with a learning disability. There was little change in this percentage between 1997 and 2010.
- The percentage of children ages 5 to 17 years reported to have ever been diagnosed with autism increased from 0.1% in 1997 to 1.0% in 2010.
- In 2010, 0.7% of children ages 5 to 17 years were reported to have ever been diagnosed with intellectual disability (mental retardation). There was little change in this percentage between 1997 and 2010.

Obesity

- Between 1976–1980 and 2007–2008, the percentage of children identified as obese showed an increasing trend. In 1976–1980, 5% of children ages 2 to 17 years were obese. This percentage reached a high of 17% in 2007–2008. Between 1999–2000 and 2007–2008, the percentage of children identified as obese remained between 15% and 17%.
- In 2005–2008, a higher percentage of Mexican-American and Black non-Hispanic children were obese at 22% and 20%, respectively, compared with 14% of White non-Hispanic children and 14% of children of "All Other Races/Ethnicities."

Adverse Birth Outcomes

- Between 1993 and 2008, the rate of preterm birth showed an increasing trend, ranging from 11.0% in 1993 to its highest value of 12.8% in 2006.
- Between 1993 and 2008, the rate of term low birth weight for all races/ethnicities stayed relatively constant, ranging between 2.5% and 2.8%.

Supplementary Topics

The Supplementary Topics section presents topics for which adequate national data are not available, but for which more targeted data collection efforts could be used to provide measures illustrating additional children's environmental health issues of interest. Data sets used for these measures are representative of particular locations (such as a single state) and/or were surveys conducted a single time rather than on a continuing or periodic basis. Since these data sets are

lacking in certain key elements desirable for ACE3 indicators, data presentations for the Supplementary Topics are referred to as "measures" rather than "indicators."

Birth Defects

- The rates for all categories of birth defects in Texas have increased or remained stable for the period of 1999–2007. Some of the biggest increases were seen for musculoskeletal defects, cardiac and circulatory defects, genitourinary defects, eye and ear defects, and central nervous system defects.

Contaminants in Schools and Child Care Facilities

- The pesticides chlorpyrifos, *cis*-permethrin, and diazinon were detected in all dust samples collected at Ohio and North Carolina child care centers in 2000–2001. Chlorpyrifos and diazinon were also detected in all indoor air samples collected at these child care centers.
- Dibutyl phthalate was detected in all indoor air and dust samples collected at Ohio and North Carolina child care centers.
- Pyrethrin and pyrethroid insecticides accounted for the greatest volume of pesticide use in California schools overall from 2002 to 2007.

Introduction

Why is EPA tracking levels of contaminants and other aspects of children's environments in *America's Children and the Environment*?

Pollutants or contaminants that can affect the health of children can be found in air, water, food, and soil. This section describes contaminants in the air children breathe, the water they drink, and the food they eat. This section also addresses the conditions of children's environments by considering indoor environments, contaminated lands, and climate change. Trends over time can indicate the successes and shortcomings of efforts to reduce potential exposures and also identify opportunities for future action. Differences in the environmental conditions between geographic areas or demographic groups can inform more targeted actions.

What Environments and Contaminants topics are included in America's Children and the Environment, Third Edition (ACE3)?

Environments and Contaminants topics were selected for ACE3 based on: (1) research findings identifying environmental contaminants or characteristics that may have adverse effects on children's health; and (2) the availability of data suitable for constructing a national indicator. EPA obtained input from its Children's Health Protection Advisory Committee to assist in selecting topics from among the many contaminants and other aspects of the environment that may affect children's health. The ACE3 Environments and Contaminants indicators address the following topics:

- Criteria air pollutants
- Hazardous air pollutants
- Indoor environments
- Drinking water contaminants
- Chemicals in food
- Contaminated lands
- Climate change[i]

Data for all of the Environments and Contaminants indicators were obtained from surveys and databases maintained by U.S. government agencies. These include the Air Quality System

[i] Although climate change is addressed in this section, a climate change indicator is not currently presented. EPA is currently developing a new children's environmental health indicator for climate change. The new indicator will focus on the frequency of extreme heat events over time. EPA intends to complete development of this new indicator in 2014, and it will be made available at www.epa.gov/ace when completed.

(Environmental Protection Agency, EPA); National Air Toxics Assessment (EPA); National Health Interview Survey (National Center for Health Statistics); American Healthy Homes Survey (Housing and Urban Development, HUD); National Survey of Lead and Allergens in Housing (HUD); Safe Drinking Water Information System (EPA); Pesticide Data Program (U.S. Department of Agriculture); Comprehensive Environmental Response, Compensation, and Liability Information System (EPA); and the Resource Conservation and Recovery Act Information dataset (EPA). Although all of the data sources feature data collected across the United States, some are not designed to produce estimates describing the nation overall. These and other data limitations are described for each indicator presented. However, targeted samples can provide important insight into environmental conditions and lead to improved measurement over time.

Other environmental hazards that may potentially be of concern for children's health are not addressed in this section. Examples of these additional environmental hazards include contaminants in surface waters, ionizing radiation, and chemicals that may be present in parks and playgrounds.

What can we learn from the Environments and Contaminants indicators?

For some of the selected Environments and Contaminants topics, health-based standards have been established. By comparing data on contaminant levels against these standards, which often include a margin of safety, it is possible to determine the percentage of children living in areas where standards or targeted levels have been exceeded. For topics where health-based standards do not exist, indicator values may still summarize conditions over time or the conditions of different groups of children, such as by race/ethnicity or income level.

It is important to realize that children may be exposed to the same contaminant through a variety of sources and pathways. For example, children can be exposed to lead by ingesting dust, consuming drinking water, and breathing air that contains lead. Each Environments and Contaminants indicator shown here only informs our understanding of potential exposure from a single pathway. A separate Biomonitoring section of ACE3 presents indicators that report levels of selected chemicals measured in blood and urine samples. The biomonitoring approach provides an integrated measure of exposure from all possible sources and pathways. However, biomonitoring data are not available for all chemicals and contaminants represented in the Environments and Contaminants indicators. The Environments and Contaminants indicators and the Biomonitoring indicators are complementary in that they represent different types of information about children's potential environmental exposures.

What information is provided for each Environments and Contaminants topic?

An introduction section defines the topic and describes its relevance to children's health, including a discussion of potential health concerns associated with exposures to the contaminants or environmental conditions. The introduction is followed by a description of the indicators, including a summary of the data available and a brief description of how each

indicator was calculated. One to three indicators, each a graphical presentation of the available data, are included for each topic. Most of the indicators present time series data. Where data over time are unavailable, the indicators present data for the most recent year available. Where possible, the indicators incorporate information on race/ethnicity and income level. Beneath each figure are explanatory bullet points highlighting key findings from the data presented in the figure, along with key data from any supplemental data tables. References are provided for each topic at the end of the report.[ii]

Data tables are provided in Appendix A. The tables include all indicator values depicted in the indicator figures, along with additional data of interest not shown in the figures. Metadata describing the data sources are provided in Appendix B. Documents providing details of how the indicators were calculated are available on the ACE website (www.epa.gov/ace).[iii]

Many of the topics presented in the Environments and Contaminants indicators are addressed in Healthy People 2020, which provides science-based, 10-year national objectives for improving the health of all Americans. Appendix C provides examples of the alignment of the Environments and Contaminants topics presented in ACE3 with objectives in Healthy People 2020.

How were the indicators calculated and presented?

Data files: The indicators were calculated using data files obtained from the government agency websites or from government agency staff.

Population age groups: Most of the indicators used data for children ages 17 years and younger. The indicators for indoor environments were restricted to younger ages because younger children have been specifically identified as more susceptible to the effects of tobacco smoke and lead exposure. The indicator for environmental tobacco smoke (E5) used data for children ages 0 to 6 years. The indicator for interior lead hazards (E6) used data for children ages 0 to 5 years.

Calculation of percentages: For most of the Environments and Contaminants topics, information on environmental contaminants/characteristics was used to identify counties where one or more environmental contaminants were above target levels established for the indicator. For example, the calculation of percentages in Indicator E1 involved identifying counties with at least one air pollutant measurement above the level of a National Ambient Air Quality Standard. The population of children in counties with an environmental contaminant above the target level was then calculated using census data, and divided by the total

[ii] In the current pre-publication copy, references are provided at the end of each topic section. In the final printed report, references will appear, separated by topic, at the end of the report.
[iii] The Methods documents will be posted to the ACE website when the final ACE3 report is posted in early 2013.

population of children to derive the indicator value as a percentage of all children in the United States.

For the indoor environments topics, survey data were obtained from representative samples of people (to estimate the percentage of children in homes with regular exposure to environmental tobacco smoke) and homes (to estimate the percentage of children in homes with lead hazards). Sample weights equal to the number of children in the U.S. population represented by each sampled child were applied to yield estimates representing the U.S. population of children. The indicator on chemicals in food reports the percentage of samples of selected foods with detectable levels of pesticides.

Statistical testing: Statistical analysis has been applied to the two indicators derived from probability-based sample data (the indoor environments indicators for environmental tobacco smoke and lead) to evaluate differences over time or between demographic groups. Statistical analysis has also been applied to the criteria pollutants data to evaluate trends over time. The remaining environment and contaminant indicators do not readily lend themselves to statistical analysis, due to the characteristics of the underlying databases.[iv]

[iv] Standard errors for the indoor environments indicator values, which are derived from survey data, are provided in a file available on the ACE website (www.epa.gov/ace/seedata.html). (This file will be posted to the ACE website when the final ACE3 report is posted in early 2013.) Standard errors could not be calculated for the remaining Environments and Contaminants indicators.

Criteria Air Pollutants

Air pollution contributes to a wide variety of adverse health effects. EPA has established national ambient air quality standards (NAAQS) for six of the most common air pollutants—carbon monoxide, lead, ground-level ozone, particulate matter, nitrogen dioxide, and sulfur dioxide—known as "criteria" air pollutants (or simply "criteria pollutants"). The presence of these pollutants in ambient air is generally due to numerous diverse and widespread sources of emissions. The primary NAAQS are set to protect public health. EPA also sets secondary NAAQS to protect public welfare from adverse effects of criteria pollutants, including protection against visibility impairment, or damage to animals, crops, vegetation, or buildings.

As required by the Clean Air Act,[1] EPA periodically conducts comprehensive reviews of the scientific literature on health and welfare effects associated with exposure to the criteria air pollutants.[2-7] The resulting assessments serve as the basis for making regulatory decisions about whether to retain or revise the NAAQS that specify the allowable concentrations of each of these pollutants in the ambient air.[8]

The primary standards are set at a level intended to protect public health, including the health of at-risk populations, with an adequate margin of safety. In selecting a margin of safety, EPA considers such factors as the strengths and limitations of the evidence and related uncertainties, the nature and severity of the health effects, the size of the at-risk populations, and whether discernible thresholds have been identified below which health effects do not occur. In general, for the criteria air pollutants, there is no evidence of discernible thresholds.[2-7]

The Clean Air Act does not require EPA to establish primary NAAQS at a zero-risk level, but rather at a level that reduces risk sufficiently so as to protect public health with an adequate margin of safety. In all NAAQS reviews, EPA gives particular attention to exposures and associated health risks for at-risk populations. Standards include consideration of providing protection for a representative sample of persons comprising at-risk populations rather than to the most susceptible single person in such groups. Even in areas that meet the current standards, individual members of at-risk populations may at times experience health effects related to air pollution.[9-13]

Childhood is often identified as a susceptible lifestage in the NAAQS reviews, because children's lungs and other organ systems are still developing, because they may have a preexisting disease (e.g., asthma), and because they may experience higher exposures due to their activities, including outdoor play.[14-17] Evaluating the effects of criteria air pollutants in children has been a central focus in several recent NAAQS reviews, including revisions of the lead,[18] ozone,[19] and particulate matter[20] standards to strengthen public health protection.

Some of the air quality standards are designed to protect the public from adverse health effects that can occur after being exposed for a short time, such as hours to days. Other standards are designed to protect people from adverse health effects that are associated with long-term

exposures (months to years). For example, the standard for ozone is based on pollutant concentrations measured over a short-term period of eight hours. By contrast, the standard for lead considers average concentrations measured over a rolling three-month period. For fine particulate matter ($PM_{2.5}$), annual and 24-hour standards work together to provide protection against effects associated with long- and short-term exposures.

Health effects that have been associated with each of the criteria pollutants are summarized below. This information is drawn primarily from EPA's assessments of the scientific literature for the criteria pollutants.

Ozone

Ground-level ozone forms through the reaction of pollutants emitted by industrial facilities, electric utilities, and motor vehicles; chemicals that are precursors to ozone formation can also be emitted by natural sources, particularly trees and other plants.[2] Ground-level ozone can pose risks to human health, in contrast to the stratospheric ozone layer that protects the earth from harmful wavelengths of solar ultraviolet radiation. Short-term exposure to ground-level ozone can cause a variety of respiratory health effects, including inflammation of the lining of the lungs, reduced lung function, and respiratory symptoms such as cough, wheezing, chest pain, burning in the chest, and shortness of breath.[2,13,21] Ozone exposure can decrease the capacity to perform exercise.[2] Exposure to ozone can also increase susceptibility to respiratory infection. Exposure to ambient concentrations of ozone has been associated with the aggravation of respiratory illnesses such as asthma, emphysema, and bronchitis, leading to increased use of medication, absences from school, doctor and emergency department visits, and hospital admissions. Short-term exposure to ozone is associated with premature mortality.[2] Studies have also found that long-term ozone exposure may contribute to the development of asthma, especially among children with certain genetic susceptibilities and children who frequently exercise outdoors.[22-24] Long-term exposure to ozone can permanently damage lung tissue.

Particulate Matter

Particulate matter (PM) is a generic term for a broad class of chemically and physically diverse substances that exist as discrete particles (liquid droplets or solids) over a wide range of sizes. Particles originate from a variety of man-made stationary and mobile sources, as well as from natural sources such as forest fires. Particles may be emitted directly, or may be formed in the atmosphere by transformations of gaseous emissions such as oxides of sulfur (SO_x), oxides of nitrogen (NO_x), and volatile organic compounds (VOCs). The chemical and physical properties of PM vary greatly with time, region, meteorology, and the source of emissions. For regulatory purposes, EPA distinguishes between categories of particles based on size, and has established standards for fine and coarse particles. PM_{10}, in general terms, is an abbreviation for particles

with an aerodynamic diameter less than or equal to 10 micrometers (µm), and represents inhalable particles small enough to penetrate deeply into the lungs (i.e., thoracic particles).[v] PM_{10} is composed of a coarse fraction referred to as $PM_{10\text{-}2.5}$ or as thoracic coarse particles (i.e., particles with an aerodynamic diameter less than or equal to 10 µm and greater than 2.5 µm) and a fine fraction referred to as $PM_{2.5}$ or fine particles (i.e., particles with an aerodynamic diameter less than or equal to 2.5 µm). Thoracic coarse particles are emitted largely as a result of mechanical processes and uncontrolled burning. Important sources include resuspended dust (e.g., resuspended by cars, wind, etc.), industrial processes, construction and demolition operations, residential burning, and wildfires. Fine particles are formed chiefly by combustion processes (e.g., from power plants, gas and diesel engines, wood combustion, and many industrial processes) and by atmospheric reactions of gaseous pollutants.

Although scientific evidence links harmful human health effects with exposures to both fine particles and thoracic coarse particles, the evidence is much stronger for fine particles than for thoracic coarse particles. Effects associated with exposures to both $PM_{2.5}$ and $PM_{10\text{-}2.5}$ include premature mortality, aggravation of respiratory and cardiovascular disease (as indicated by increased hospital and emergency department visits), and changes in sub-clinical indicators of respiratory and cardiac function. Such health effects have been associated with short- and/or long-term exposure to PM.[vi] Exposures to $PM_{2.5}$ are also associated with decreased lung function growth, exacerbation of allergic symptoms, and increased respiratory symptoms.[6] Children, older adults, individuals with preexisting heart and lung disease (including asthma), and persons with lower socioeconomic status are considered to be among the groups most at risk for effects associated with PM exposures.[6] Information is accumulating and currently provides suggestive evidence for associations between long-term $PM_{2.5}$ exposure and developmental effects such as low birth weight and infant mortality due to respiratory causes.[6]

Sulfur Dioxide

Fossil fuel combustion by electrical utilities and industry is the primary source of sulfur dioxide in the United States.[5] People with asthma are especially susceptible to the effects of sulfur dioxide.[5] Short-term exposures of asthmatic individuals to elevated levels of sulfur dioxide while exercising at a moderate level may result in breathing difficulties, accompanied by symptoms such as wheezing, chest tightness, or shortness of breath. Studies also provide consistent evidence of an association between short-term sulfur dioxide exposures and increased respiratory symptoms in children, especially those with asthma or chronic respiratory symptoms. Short-term exposures to sulfur dioxide have also been associated with respiratory-related emergency department visits and hospital admissions, particularly for children and older adults.[5]

[v] For comparison, the diameter of PM_{10} particles is 1/7 the diameter of an average human hair or less.
[vi] For $PM_{10\text{-}2.5}$, the evidence linking health effects to short-term (e.g., 24-hour) exposures is stronger than the evidence for effects of long-term exposures.

Nitrogen Dioxide

Nitric oxide (NO) and nitrogen dioxide (NO_2) are emitted by cars, trucks, buses, power plants, and non-road engines and equipment. Emitted NO is rapidly oxidized into NO_2 in the atmosphere.[4] Exposure to nitrogen dioxide has been associated with a variety of health effects, including respiratory symptoms, especially among asthmatic children, and respiratory-related emergency department visits and hospital admissions, particularly for children and older adults.[4]

Lead

Historically, the major source of lead emissions to the air was combustion of leaded gasoline in motor vehicles (such as cars and trucks). Following the elimination of leaded gasoline in the United States by the mid-1990s, the remaining sources of lead air emissions have been industrial sources, including lead smelting and battery recycling operations, and piston-engine small aircraft that use leaded aviation gasoline.[3] Lead accumulates in bones, blood, and soft tissues of the body. Exposure to lead can affect development of the central nervous system in young children, resulting in neurodevelopmental effects such as lowered IQ and behavioral problems.[3]

Carbon Monoxide

Gasoline-fueled vehicles and other on-road and non-road mobile sources are the primary sources of carbon monoxide (CO) in the United States.[7] Exposure to carbon monoxide reduces the capacity of the blood to carry oxygen, thereby decreasing the supply of oxygen to tissues and organs such as the heart. People with several types of heart disease already have a reduced capacity for pumping oxygenated blood to the heart, which can cause them to experience myocardial ischemia (reduced oxygen to the heart), often accompanied by chest pain (angina), when exercising or under increased stress. For these people, short-term CO exposure further affects their body's already compromised ability to respond to the increased oxygen demands of exercise or exertion. Thus people with angina or heart disease are identified as at greatest risk from ambient CO. Other potentially at-risk populations include those with chronic obstructive pulmonary disease, anemia, diabetes, and those in prenatal or elderly lifestages.[7]

The period of fetal development may be one of particular vulnerability for adverse health effects resulting from maternal exposure to some criteria air pollutants. This may occur if maternal exposure to air pollutants is transferred to the fetus during pregnancy; for example, lead and PM have both been shown to cross the placenta and accumulate in fetal tissue during gestation.[3,6] In addition to the findings noted above regarding associations of prenatal PM exposure and adverse birth outcomes (such as low birth weight), limited studies of prenatal exposure to criteria air pollutants have reported that exposure to PM and oxides of nitrogen and sulfur may increase the risk of developing asthma as well as worsen respiratory outcomes among those children that do develop asthma.[25-27] However, it is often difficult to distinguish

the effects of prenatal and early childhood exposure because exposure to air pollutants is often very similar during both time periods.

Additional research indicates that exposure to pollution from traffic-related sources, a mix of criteria air pollutants and hazardous air pollutants, may pose particular threats to a child's respiratory system. Many studies have reported a correlation between proximity to traffic (or to traffic-related pollutants) and occurrence of new asthma cases or exacerbation of existing asthma and other respiratory symptoms, including reduced growth of lung function during childhood.[25,28-35] A report by the Health Effects Institute concluded that living close to busy roads appears to be an independent risk factor for the onset of childhood asthma.[36] The same report also concluded that the evidence was "sufficient" to infer a causal association between exposure to traffic-related pollution and exacerbations of asthma in children.[36] Some studies have suggested that traffic-related pollutants may contribute to the development of allergic disease, either by affecting the immune response directly or by increasing the concentration or biological activity of the allergens themselves.[37-39]

Many of the effects of criteria air pollutants on children can be reduced by limiting outdoor activities on high pollution days.[40] Such avoidance measures can have their own adverse impacts on children's health when they reduce opportunities for play and exercise.

The following three indicators provide different perspectives on children's exposures to criteria air pollutants. Indicator E1 summarizes the percentages of children over time living in counties where measured pollutant concentrations were above the levels of the short- and/or long-term standards for each of the criteria air pollutants.[vii] Indicator E2 provides additional detail on the frequency with which pollutant concentrations were above the levels of the ozone and 24-hour $PM_{2.5}$ standards in one year (2009). Indicator E3 focuses on the frequency with which children were exposed to good, moderate, or unhealthy daily air quality, based on EPA's Air Quality Index.

[vii] For standards with averaging times less than or equal to 24 hours, Indicator E1 includes counties where concentrations were above the level of the standards at least one day per year.

Indicator E1: Percentage of children ages 0 to 17 years living in counties with pollutant concentrations above the level of the current air quality standards, 1999–2009

Indicator E2: Percentage of children ages 0 to 17 years living in counties with 8-hour ozone and 24-hour PM2.5 concentrations above the levels of air quality standards, by frequency of occurrence, 2009

About the Indicators: Indicators E1 and E2 present the percentage of children living in counties where measured ambient concentrations of criteria pollutants were greater than the levels of the Clean Air Act health-based standards at any time during a year. Indicator E1 presents results for each criteria pollutant for each year. Indicator E2 presents more detailed information on the frequency with which measured ambient ozone and fine particle ($PM_{2.5}$) concentrations were greater than the levels of the short-term standards for ozone and $PM_{2.5}$ in 2009. The air quality data used in these indicators are from an EPA database that compiles measurements of pollutants in ambient air from around the country each year.

Air Quality System

State and local environmental agencies that monitor air quality submit their data to EPA. EPA compiles the monitoring data in the national EPA Air Quality System (AQS) database.[viii] AQS contains some monitoring data from the late 1950s and early 1960s, but there is not an appreciable amount of data for lead until 1970, sulfur dioxide until 1971, nitrogen dioxide until 1974, carbon monoxide and ozone until 1975, and PM_{10} until 1987. AQS also contains monitoring data for $PM_{2.5}$ beginning in 1999; $PM_{2.5}$ was measured only infrequently prior to 1999. Indicators E1 and E2 are derived from analysis of air pollution data in AQS.

Air Quality Standards and Concentrations Above the Levels of the Standards

Under the Clean Air Act, EPA has established National Ambient Air Quality Standards (NAAQS) for carbon monoxide, lead, ground-level ozone, particulate matter, nitrogen dioxide, and sulfur dioxide. There are four basic elements of NAAQS that together serve to define each standard: the definition of the pollutant,[ix] the averaging time (e.g., annual average or 24-hour average), the level, and the form of the standard (which defines the air quality statistic compared to the level of the standard in determining whether an area attains the standard—for example, the 24-hour $PM_{2.5}$ standard uses 98th percentile concentrations, averaged over three years). These

[viii] Information on the AQS database is available at http://www.epa.gov/air/data/aqsdb.html.

[ix] In the development of NAAQS, the term "indicator" defines the chemical species or mixture that is to be measured in determining whether an area attains the standard. To avoid confusion with the way in which "indicator" is used throughout *America's* Children *and the Environment*, the term is not used in the following paragraphs, except to refer to the ACE criteria pollutant indicators E1, E2, and E3.

elements must be considered collectively in evaluating the health and welfare protection afforded by the NAAQS.

Indicators E1 and E2 consider the first three elements of a NAAQS: the definition of the pollutant, the averaging time, and the level of the standard. The indicators present percentages of children living in areas with pollutant concentrations above the level of the current standards, using the appropriate averaging time. The indicators do not consider the form of the standard, which often includes considerations for multiple years of air quality data (e.g., 3 years), adjustments for missing data and less-than daily monitoring, and consideration for the frequency and magnitude with which a standard level is exceeded. In considering the form of the NAAQS, these standards are defined to allow some days to be above the level of the standard while limiting the extent to which they are above the level of the standard. Furthermore, determinations of attainment with the NAAQS are generally based on air quality averaged over multiple years. Therefore, air quality in any one-year period, as presented in Indicators E1 and E2, cannot be used to characterize whether air quality does or does not meet the NAAQS. The analyses for Indicators E1 and E2 therefore differ from the analyses used by EPA for the designation of "nonattainment areas" (locations that have not attained the standard) for regulatory compliance purposes.[41] Nonetheless, looking at air quality within a given year, or across many individual years, provides important public health information.

For each of the years 1999–2009, Indicator E1 reflects comparisons of the monitoring data with the levels of the current NAAQS. The indicator for all years therefore incorporates the 2006 revision of the level of the 24-hour $PM_{2.5}$ standard[20] from 65 µg/m^3 to 35 µg/m^3; the 2008 revision of the level of the eight-hour ozone standard[19] from 0.08 ppm to 0.075 ppm;[x] the 2008 revision of the level of the three-month standard[18] for lead from 1.5 µg/m^3 to 0.15 µg/m^3; the establishment of a new one-hour standard[42] for nitrogen dioxide with a level of 100 ppb, issued in 2010; and the establishment of a new one-hour standard[43] for sulfur dioxide with a level of 75 ppb, issued in 2010. Note that EPA promulgated a revised annual $PM_{2.5}$ standard in December 2012, which has not been incorporated into this analysis. Table 1 in the Methods documentation shows the criteria pollutant levels used for the purpose of this indicator to determine whether concentrations were above the standard level for each pollutant.[xi]

[x] In January 2010, the EPA Administrator proposed to reconsider the ozone standard because she believed "that a standard set as high as 0.075 would not be considered requisite to protect public health with an adequate margin of safety, and that consideration of lower levels [was] warranted" (75 FR 2996, January 19, 2010). EPA is currently conducting the next statutorily mandated periodic review of the ozone standards, which the Agency plans to complete in 2014. See http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_index.html for more information on the current and previous ozone NAAQS reviews.

[xi] All criteria pollutants are included in Indicator E1, but for some pollutants with multiple primary standards (reflecting different averaging times), only a single standard is included. For CO only the 8-hour standard is included, because the 1-hour standard is rarely exceeded. For NO_2 only the 1-hour standard is included, because the annual standard is rarely exceeded.

NAAQS are intended to provide public health protection, including providing protection for at-risk populations, with an adequate margin of safety.[xii] EPA's selection of the current standards for ozone, nitrogen dioxide, and sulfur dioxide were intended to protect against respiratory effects in at-risk populations, including children. EPA's selection of the current standards for particulate matter was based primarily on concerns for mortality and cardiovascular effects, as well as respiratory effects. EPA's selection of the current standard for lead was intended to reduce risks of neurodevelopmental effects in children. The standard for carbon monoxide is intended primarily to protect against potential effects in people with heart disease. The Clean Air Act does not require the EPA Administrator to establish a primary NAAQS at a zero-risk level or at background concentration levels, but rather at a level that reduces risk sufficiently so as to protect health with an adequate margin of safety. However, pollutant concentrations that are lower than the levels of the standards are not necessarily without risk for all individuals. No risk-free level of exposure has been determined for any of the criteria pollutants.

Data Presented in the Indicators

Indicator E1 presents the percentage of children living in counties with measured pollutant concentrations above the level of a NAAQS for any of the criteria pollutants, for each year from 1999–2009.[xiii] The indicator begins with data for 1999 because, as noted above, this was the first year of widespread monitoring for $PM_{2.5}$. In addition to presenting data for each of the criteria pollutants separately, the indicator also presents the percentage of children living in counties with measured concentrations above the level of a NAAQS for any criteria air pollutant (i.e., exceedance of standard levels for one or more criteria air pollutants).

Indicator E1 does not differentiate between counties in which concentrations were above standard levels frequently or by a large margin, and areas in which concentrations were above standard levels only rarely or by a small margin. It also assumes that air pollutant concentrations are consistent throughout a county. Some pollutants, such as ozone and $PM_{2.5}$, tend to be well dispersed and generally have limited spatial variation within a county, whereas other pollutants such as lead might have higher concentrations within relatively smaller areas. The indicator is based on concentrations of individual pollutants compared with individual standard levels, and does not reflect any combined effect of exposure to multiple criteria pollutants.

All children living in all counties are considered in the indicator; however, many counties do not have air pollution monitors. Monitoring networks are typically designed to focus on areas that

[xii] The legislative history of section 109 of the Clean Air Act indicates that a primary standard is to be set at "the maximum permissible ambient air level… which will protect the health of an [sensitive] group of the population," and that for this purpose, "reference should be made to a representative sample of persons comprising the sensitive group rather than to a single person in such a group" S. Rep. No. 91-1196, 91st Cong., 2d Sess. 10 (1970).

[xiii] For standards with averaging times less than or equal to 24 hours, Indicator E1 includes counties where concentrations were above the level of the standards at least one day per year.

are expected to have higher concentrations or that have larger populations. If any of the unmonitored counties have concentrations above the levels of the NAAQS, Indicator E1 will understate the percentage of children living in counties with concentrations above standard levels. The indicator thus represents the percentage of all children who lived in counties with confirmed pollutant concentrations above the levels of the standards each year, where confirmation is provided by a valid monitor value in that year. The percentages of children in unmonitored counties in 2009 range from about 30% for ozone and $PM_{2.5}$ to about 50% for PM_{10}, carbon monoxide, sulfur dioxide and nitrogen dioxide, and about 80% for lead.[xiv] These percentages have been fairly stable from 1999–2009, though there are some limited changes in monitoring from year to year. Those limited changes in monitoring mean that there are some small changes in data available for calculation of the indicator over time.

The supplemental data tables E1a and E1b show the percentage of children living in counties with concentrations above the levels of the air quality standards in 2009 by race/ethnicity (Table E1a) and family income (Table E1b).

Ambient concentrations were more frequently above the levels of the 8-hour ozone and the 24-hour $PM_{2.5}$ standards than the levels of the standards for other criteria pollutants. Indicator E2 provides information on the frequency with which concentrations were above the levels of these two standards in 2009. Counties were classified by the number of days during 2009 that measured pollutant concentrations were above the levels of the 8-hour ozone and 24-hour $PM_{2.5}$ standards. This indicator, therefore, shows the percentage of children living in counties in which concentrations were measured above the levels of these two short-term standards a few times, as well as the percentage in counties with more frequent measurements above the levels of the standards. The percentage of children in counties without monitors for these two pollutants in 2009 is also shown in Indicator E2. The data table for this indicator (Table E2) also provides the same information for each year 1999–2009, using the current level of the standards for each year's calculation.

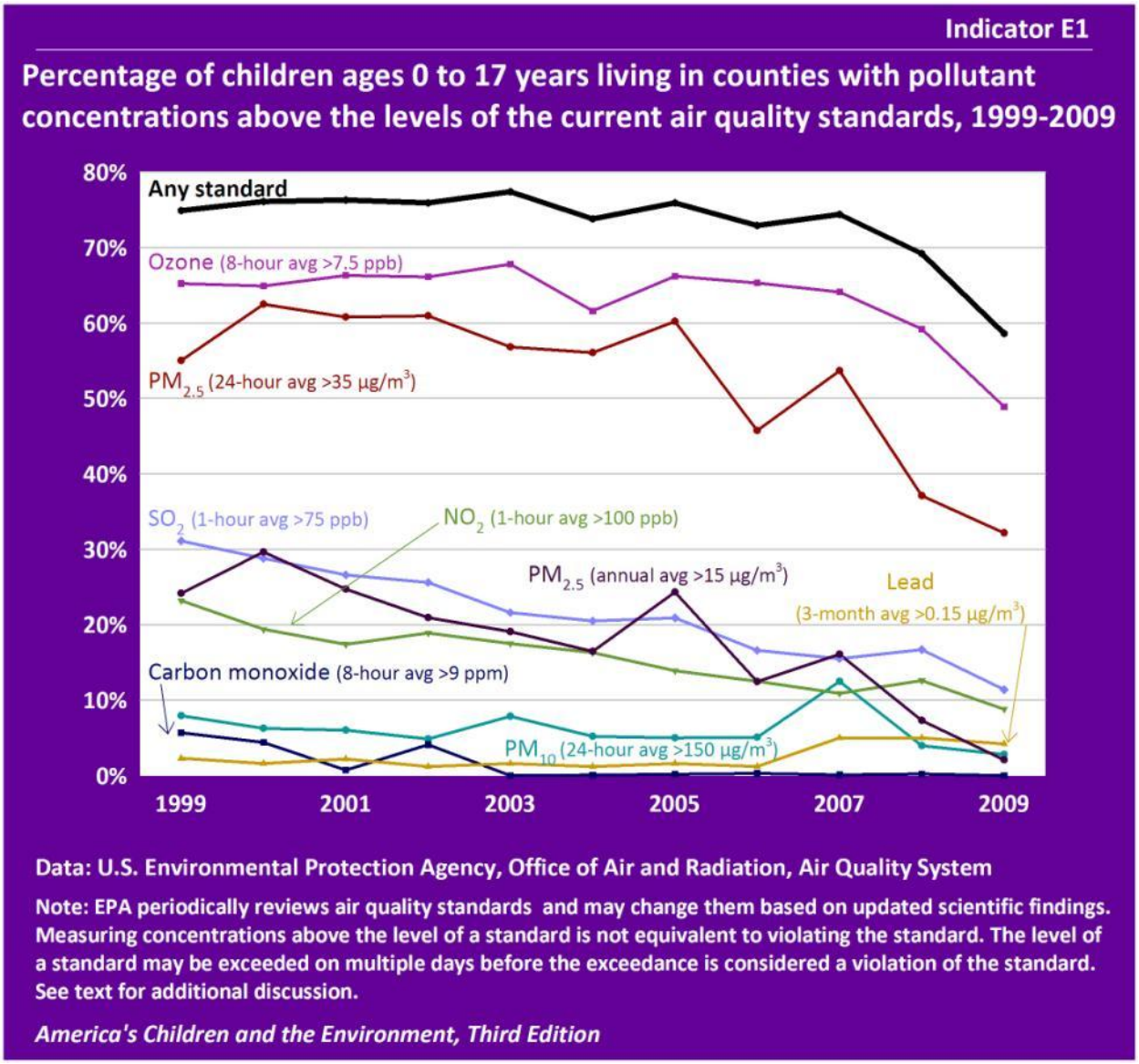
Values in this indicator may be understated due to the fact that most monitors do not operate every day. Ozone monitors operate daily during the ozone season, which lasts from 6 to 7 months in most locations but can be between 5 and 12 months (based on ranges of dates when high temperatures associated with high ozone concentrations may occur). $PM_{2.5}$ monitors operate year round, but may collect measurements daily or every third or every sixth day. EPA requires areas that measure concentrations within 5% of the 24-hour $PM_{2.5}$ standard to monitor daily. Monitors for other criteria pollutants operate year round.

[xiv] EPA issued increased requirements for lead monitoring in December 2010.[44]

Statistical Testing

Statistical analysis has been applied to Indicator E1 to evaluate trends over time in the percentage of children living in counties with concentrations above the standard levels each year. These analyses use a 5% significance level, meaning that a conclusion of statistical significance is made only when there is no more than a 5% probability that the observed trend occurred by chance ($p \leq 0.05$). The statistical analysis of trends over time is dependent on how the annual values vary as well as on the number of annual values. For example, the statistical test is more likely to detect a trend when data have been obtained over a longer period. It should be noted that conducting statistical testing for multiple air quality standards increases the probability that some trends identified as statistically significant may actually have occurred by chance.

A finding of statistical significance is useful for determining that an observed trend was unlikely to have occurred by chance. However, a determination of statistical significance by itself does not convey information about the magnitude of the increase or decrease in indicator values. Furthermore, a lack of statistical significance means only that occurrence by chance cannot be ruled out. Thus, a conclusion about statistical significance is only part of the information that should be considered when determining the public health implications of trends.



Data characterization

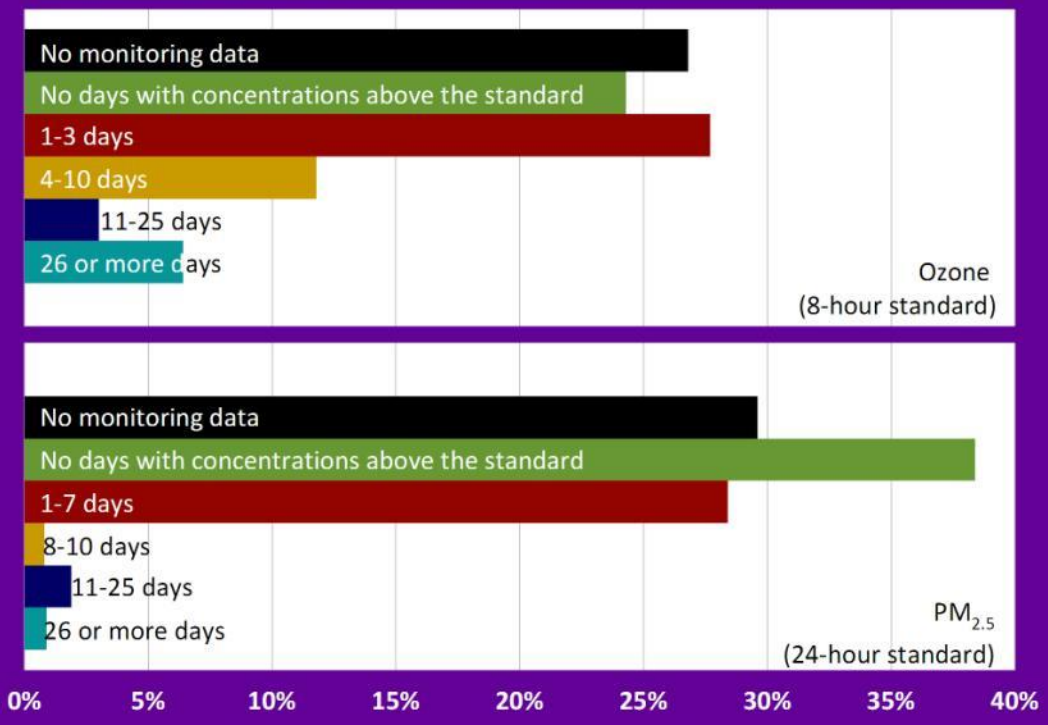
- Data for this indicator are obtained from EPA's database of air quality monitoring measurements.
- Air pollution monitors are placed in locations throughout the country, with an emphasis on areas expected to have higher pollutant concentrations or that have larger populations. Not all counties in the United States have air pollution monitors, and the number of counties with monitors has changed over time.
- Monitors generally tend to stay in the same location over many years, but there may be some limited changes in the number or location of monitors providing data from year to year.

- From 1999 to 2009, the proportion of children living in counties with measured pollutant concentrations above the levels of one or more national ambient air quality standards decreased from 75% to 59%. This includes both concentrations above the level of any

current short-term standard at least once during the year as well as average concentrations above the level of any current long-term standards.
    - The decreasing trend over the years 1999–2009 was statistically significant.
- From 1999–2009, the percentage of children living in counties with measured ozone concentrations above the level of the current 8-hour ozone standard at least one day during the year decreased from 65% to 49%.
    - The decreasing trend for ozone over the years 1999–2009 was statistically significant.
- From 1999–2009, the percentage of children living in counties with measured $PM_{2.5}$ concentrations above the level of the current 24-hour $PM_{2.5}$ standard at least once per year decreased from 55% to 32%. Over the same years, the percentage of children living in counties with a measured annual average concentration above the level of the current annual $PM_{2.5}$ standard declined from 24% to 2%.
    - The decreasing trends for $PM_{2.5}$ were statistically significant.
- From 1999–2009, the percentage of children living in counties with measured sulfur dioxide concentrations above the level of the current one-hour standard for sulfur dioxide at least one day per year declined from 31% to 11%. Over the same years, the percentage of children living in counties with measured concentrations above the level of the current one-hour standard for nitrogen dioxide at least one day per year decreased from 23% to 9%.
    - The decreasing trends for both sulfur dioxide and nitrogen dioxide were statistically significant.
- In each year since 1999, between 1 and 5% of children lived in counties with measured ambient lead concentrations above the level of the current three-month standard for lead. In 2009, 8 counties with 4% of U.S. children reported concentrations above the level of the three-month standard for lead.
- In 2009, 3% of children lived in counties with measured PM_{10} concentrations above the level of the current 24-hour standard for PM_{10} at least one day per year, and no children lived in counties with measured concentrations above the level of the current standard for carbon monoxide.

Indicator E2

Percentage of children ages 0 to 17 years living in counties with 8-hour ozone and 24-hour $PM_{2.5}$ concentrations above the levels of air quality standards, by frequency of occurrence, 2009



Data: U.S. Environmental Protection Agency, Office of Air and Radiation, Air Quality System

Note: EPA periodically reviews air quality standards and may change them based on updated scientific findings. Measuring concentrations above the level of a standard is not equivalent to violating the standard. The level of a standard may be exceeded on multiple days before the exceedance is considered a violation of the standard. See text for additional discussion.

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Data characterization

- Data for this indicator are obtained from EPA's database of air quality monitoring measurements.
- Air pollution monitors are placed in locations throughout the country, with an emphasis on areas expected to have higher pollutant concentrations or that have larger populations. Not all counties in the United States have air pollution monitors.
- Some air pollution monitors do not operate every day, so some days with pollutant concentrations above the levels of the air quality standards may not be identified.
- In 2009, 27% of children lived in counties with no monitoring data for ozone, and 30% lived in counties with no monitoring data for $PM_{2.5}$.

- In 2009, 6% of children lived in counties with measured ozone concentrations above the level of the 8-hour ozone standard on more than 25 days. An additional 3% of children lived in counties with measured concentrations above the level of the ozone standard between 11 and 25 days, and 12% of children lived in counties where concentrations were above the level of the standard between 4 and 10 days.
- In 2009, 1% of children lived in counties with measured $PM_{2.5}$ concentrations above the level of the 24-hour $PM_{2.5}$ standard on more than 25 days. An additional 2% of children lived in counties with measured concentrations above the level of this standard between 11 and 25 days, and 1% of children lived in counties with measured concentrations above the level of the 24-hour $PM_{2.5}$ standard between 8 and 10 days.
- In 1999, 23% of children lived in counties with measured ozone concentrations above the level of the current 8-hour ozone standard on more than 25 days. An additional 27% of children lived in counties with measured concentrations above the level of the ozone standard between 11 and 25 days, and 11% of children lived in counties where concentrations were above the level of the standard between 4 and 10 days. (See Table E2.)
- In 1999, 6% of children lived in counties with measured $PM_{2.5}$ concentrations above the level of the current 24-hour $PM_{2.5}$ standard more than 25 days. An additional 9% of children lived in counties with measured concentrations above the level of this standard 11 and 25 days, and 3% of children lived in counties with measured concentrations above the level of the 24-hour $PM_{2.5}$ standard between 8 and 10 days. (See Table E2.)

Indicator E3: Percentage of days with good, moderate, or unhealthy air quality for children ages 0 to 17 years, 1999–2009

About the Indicator: Indicator E3 presents data from EPA's Air Quality Index (AQI). The AQI produces a rating of the air quality for each county on each day, considering all monitoring results available on that day for carbon monoxide, ozone, nitrogen dioxide, particulate matter, and sulfur dioxide. Air quality in each county is considered to be "good," "moderate," or "unhealthy" based on comparison of the monitored pollutant concentrations to breakpoints defined by the AQI. The indicator is calculated by considering the number of children in counties with each rating for each day of the year, then summing the number of children for all days in the year.

Air Quality Index

EPA's Air Quality Index (AQI)[xv] represents air quality for each individual day and is widely reported in newspapers and other media outlets in metropolitan areas. The AQI is based on daily measurements of up to five of the six air quality criteria pollutants (carbon monoxide, ozone, nitrogen dioxide, particulate matter, and sulfur dioxide). The standard for lead is not included in the AQI because it requires averaging concentrations over a three-month period, and it can take several weeks to collect and analyze lead samples.

The specific pollutants considered in the AQI for each metropolitan area depend on the pollutants monitored in that area each day. Each pollutant concentration is given a value on a scale relative to the air quality standard for that pollutant. The daily AQI is based on the single pollutant with the highest index value that day. An AQI value of 100 corresponds to the level of the short-term (e.g., daily or hourly) NAAQS for each criteria pollutant. An AQI value of 50 is defined either as the level of the annual standard, if one has been established (e.g., $PM_{2.5}$, NO_2), or as a concentration equal to one-half the value of the short-term standard used to define an index value of 100 (e.g., CO).

EPA has divided the AQI scale into categories. Air quality is considered "good" (referred to as "code green") if the AQI is between 0 and 50, posing little or no risk. Air quality is considered "moderate" ("code yellow") if the AQI is between 51 and 100. Some pollutants at this level may present a moderate health concern for a small number of individuals. Air quality is considered "unhealthy for sensitive groups" if the AQI is between 101 and 150 (referred to as "code orange"). On code orange days, members of at-risk populations such as children may experience health effects, but the rest of the general population is unlikely to be affected. Air quality is considered "unhealthy" if the AQI is between 151 and 200 ("code red"). The general population may begin to experience health effects, and members of at-risk populations may

[xv] Available at http://www.airnow.gov/.

experience more serious health effects. Values of 201 to 300 are designated as "very unhealthy" ("code purple"), while values of 301 to 500 are considered "hazardous" ("code maroon"). Decisions about the pollutant concentrations at which to set the various AQI breakpoints that delineate the various AQI categories draw directly from the underlying health information that supports the reviews of the NAAQS.

For $PM_{2.5}$, the AQI values used in preparing Indicator E3 were calculated with a 24-hour concentration of 40 µg/m^3 used to define air quality as "unhealthy for sensitive groups" (i.e., an AQI value of 100), rather than the level of the current 24-hour $PM_{2.5}$ standard of 35 µg/m^3. As a consequence, Indicator E3 likely overstates the days with moderate air quality and understates the days with unhealthy air quality.[xvi]

Data Presented in the Indicator

Indicator E3 is based on the reported AQI for counties in the United States. EPA calculates an AQI value each day in each county for five major air pollutants regulated by the Clean Air Act: ozone, particulate matter, carbon monoxide, sulfur dioxide, and nitrogen dioxide. The highest of these pollutant-specific AQI values is reported as the county's AQI value for that day.

Indicator E3 was developed by reviewing the AQI designation for each day for each county and weighting the daily designations by the number of children living in each county. The calculation, therefore, is a summation of the AQI values for all children in the United States, based on county of residence, for each day of the year. For example, the number of days of good air quality during the year is counted up for each child in the population based on the daily air quality in the county where they live. The overall indicator reports the percentage of children's days in each year considered to be of good (AQI 0–50; code green), moderate (AQI 51–100; code yellow), or unhealthy (AQI greater than 100; codes orange, red, purple, and maroon combined) air quality.[xvii] The percentage of children's days with no AQI value available (representing the absence of monitoring data) are also reported in Indicator E3.

Whereas Indicator E1 presents an annual analysis of counties in which concentrations were above the level of a standard for a pollutant, the AQI data used in Indicator E3 are based on the concentrations for all pollutants for which an AQI has been established in each county over the course of a year. The E3 method uses data on the air quality category for each day, rather than simply reporting whether a county ever exceeds the standard for each pollutant during the

[xvi] In December 2012, EPA promulgated a rule to change the AQI to use 35 µg/m^3 for defining the AQI value of 100 for $PM_{2.5}$. Prior to this rule, although the AQI had not formally been changed, an EPA guidance document[45] recommended use of 35 µg/m^3 for defining the AQI value of 100 for $PM_{2.5}$. States have generally been using 35 µg/m^3 in calculating and reporting their daily AQI values.

[xvii] As discussed above, an AQI value of 100 generally corresponds to the level of a short-term national ambient air quality standard. When AQI values are above 100, air quality is considered to be unhealthy—at first for certain sensitive groups of people (101 to 150), then for everyone as AQI values get higher.

year. However, the AQI method has some limitations. The AQI is based on the single pollutant with the highest value for each day; it does not reflect any combined effect of multiple pollutants or the effects of pollutants that were not measured on a given day.

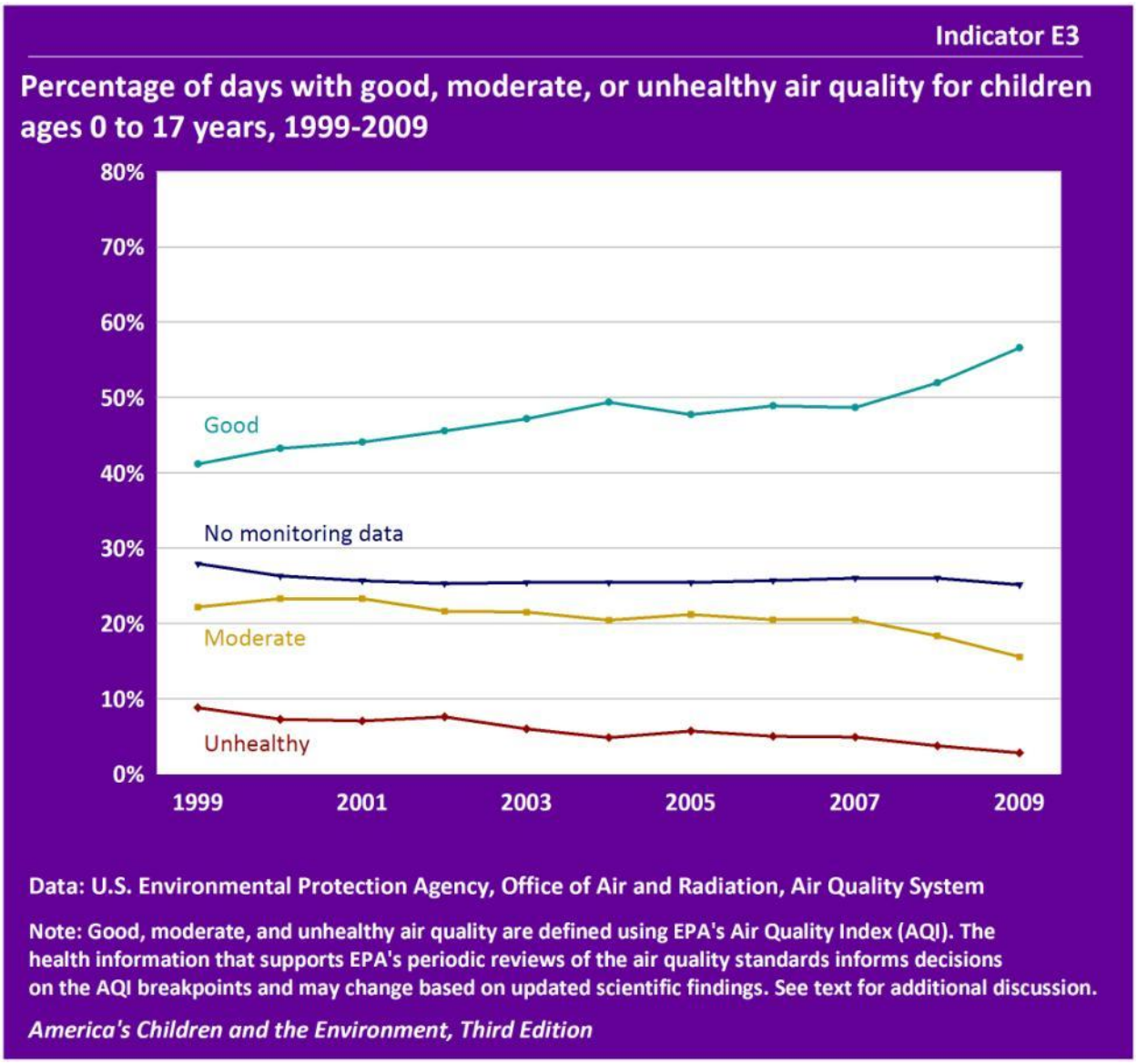
Indicator E3 starts in 1999 because this was the first year of widespread monitoring for $PM_{2.5}$. The indicator uses a consistent set of pollutant concentrations to define good, moderate, or unhealthy air quality for all years shown, 1999–2009, but as noted above, the level of the current 24-hour standard for $PM_{2.5}$ has not been incorporated into calculation of the indicator.

Tables E3a and E3b show the percentage of children's days of exposure to good, moderate, or unhealthy air quality in 2009 by race/ethnicity (Table E3a) and family income (Table E3b). These calculations do not account for any possible variation in air quality within a county, and thus may not fully reflect the variability in air quality among children of different race/ethnicity and income.

Statistical Testing

Statistical analysis has been applied to Indicator E3 to evaluate trends over time in the percentage of children's days of with good, moderate, or unhealthy air quality. These analyses use a 5% significance level, meaning that a conclusion of statistical significance is made only when there is no more than a 5% probability that the observed trend occurred by chance ($p \leq 0.05$). The statistical analysis of trends over time is dependent on how the annual values vary as well as on the number of annual values. For example, the statistical test is more likely to detect a trend when data have been obtained over a longer period.

A finding of statistical significance is useful for determining that an observed trend was unlikely to have occurred by chance. However, a determination of statistical significance trend over time does not imply anything about the magnitude of the increase or decrease in indicator values. Furthermore, a lack of statistical significance means only that occurrence by chance cannot be ruled out. Thus, a conclusion about statistical significance is only part of the information that should be considered when determining the public health implications of trends.



Data characterization

- Data for this indicator are obtained from EPA's database of daily Air Quality Index (AQI) values for each county in the United States.
- Air pollution monitors are placed in locations throughout the country, with an emphasis on areas expected to have higher pollutant concentrations or that have larger populations.
- AQI values are based on daily monitoring data for up to five criteria air pollutants. Some counties do not have monitors, and some monitors do not operate every day, so some days do not have AQI values.
- For this indicator, the available monitoring data are used to assign a value of "good," "moderate," "unhealthy," or "no monitoring data" for each day in each U.S. county.

- The percentage of children's days that were designated as having "unhealthy" air quality decreased from 9% in 1999 to 3% in 2009. The percentage of children's days with "good" air quality increased from 41% in 1999 to 57% in 2009. The percentage of children's days with "moderate" air quality was approximately constant at 21–23% from 1999 to 2007, and then decreased to 16% in 2009.
  - The 1999 to 2009 trends in "unhealthy," "good," and "moderate" air quality days were statistically significant.

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Hazardous Air Pollutants

Hazardous Air Pollutants (HAPs) are air contaminants, frequently referred to as "air toxics," that are known or suspected to cause serious human health effects or adverse environmental effects.[1] The Clean Air Act identifies 187 substances as HAPs. Examples include benzene, trichloroethylene, mercury, chromium, and dioxin. The "criteria" air pollutants such as ozone and particulate matter are excluded from the HAPs list.[xviii]

HAPs are emitted into ambient air from a diverse range of facilities, businesses, and vehicles that are grouped into three general categories: major sources, area sources, and mobile sources. Major sources typically are large industrial facilities such as chemical manufacturing plants, refineries, and waste incinerators. These sources may release air toxics from equipment leaks, when materials are transferred from one location to another, or during discharge through emission stacks or vents. Area sources typically are smaller stationary facilities such as dry cleaners, auto body repair shops, and small manufacturing operations. Though emissions from individual area sources often are relatively small, collectively they can be of concern—particularly where large numbers of sources are located in heavily populated areas. Mobile sources include both on-road sources, such as cars, light trucks, large trucks, and buses, and non-road sources such as farm and construction equipment, lawn and garden equipment, marine engines, aircraft, and locomotives. Some HAPs are also emitted from natural sources such as volcanoes. Health effects associated with HAPs include cancer, asthma and other respiratory ailments, birth defects, reproductive effects, and neurodevelopmental effects.[2-9]

In some cases, health concerns are based on studies of workers exposed to high levels of particular HAPs on the job. For example, EPA has determined that HAPs such as benzene; 1,3-butadiene; chromium; nickel; and vinyl chloride are carcinogenic to humans, based on findings in occupational studies.[10-14] Similarly, toluene diisocyanate exposure has been associated with effects on the lung, and manganese exposure with neurological effects, in occupational studies.[15,16]

A limited number of HAPs have also been studied in human populations that have been exposed in their day-to-day lives. For examples, several studies have reported associations between formaldehyde exposure (usually indoors at home or at school) and childhood asthma.[3] In addition, a series of recent studies conducted in New York City reported that children of women who were exposed to increased levels of polycyclic aromatic hydrocarbons (PAHs, produced when gasoline and other materials are burned) during pregnancy are more likely to have experienced adverse effects on neurological development (such as reduced intelligence quotient (IQ) or behavioral problems[6,7]), as well as respiratory effects.[17-19]

[xviii] Lead is an exception: it is regulated as a criteria pollutant, and "lead compounds" are included on the list of HAPs. Note that criteria pollutants are discussed further in the *Criteria Air Pollutants* topic.

For the majority of HAPs, however, there are no human epidemiological studies, or very few, and concern for health effects is based on findings from animal studies. For example, many HAPs, such as PAHs,[20] acetaldehyde[21] and carbon tetrachloride[22] are considered likely to be carcinogenic to human based primarily on evidence from long-term laboratory animal studies.

Although many HAPs are of concern due to their potential to cause cancer, a substantial number of HAPs lack evidence of cancer—either because the relevant long-term studies have not been conducted, or because studies have been conducted and do not indicate carcinogenic potential. An example of a HAP that is not associated with cancer is acrolein; there are no appropriate human or animal studies with which to assess the carcinogenic potential of acrolein. However, acrolein has been identified as a HAP of particular concern for effects other than cancer.[23,24] Health concerns for acrolein include respiratory effects and irritation of the eyes, nose, and throat, based on animal studies and on short-term studies of small groups of humans intentionally exposed to high levels of acrolein.[25]

EPA relies on both monitoring and modeled data to characterize ambient air concentrations of HAPs, and to estimate potential human exposure and risk of adverse health effects associated with these toxics. EPA and state monitoring programs do not cover all the places where people live in the United States. For this reason, the following indicator relies on modeled data from the National Air Toxics Assessment.[26] The indicator presents the percentage of children living in census tracts with estimated HAP concentrations greater than benchmark comparison levels derived from health effects information.

In addition to their presence in ambient air, many HAPs also have indoor sources, and the indoor sources may frequently result in greater exposure than the presence of HAPs in ambient air. Sufficient data are not available to develop an indicator considering the combined exposure to HAPs from both indoor and outdoor sources; therefore the following indicator considers only levels of HAPs in ambient air.[xix]

[xix] Indoor sources of HAPs are further discussed in the *Indoor Environments* and *Contaminants in Schools and Child Care Facilities* topics, and in several of the biomonitoring topics.

Indicator E4: Percentage of children ages 0 to 17 years living in census tracts where estimated hazardous air pollutant concentrations were greater than health benchmarks in 2005

About the Indicator: Indicator E4 presents estimates of the percentage of children living in census tracts with ambient hazardous air pollutant (HAP) concentrations greater than benchmark values representing levels of concern for health effects. The HAP concentrations are computer model estimates for 2005, representing all identified sources of HAP emissions, including factories and motor vehicles. The health benchmarks are based on concerns for cancer and other adverse health effects that may be associated with HAP exposure.

National Air Toxics Assessment

EPA's National Air Toxics Assessment (NATA) provides estimated concentrations of 181 HAPs in ambient air for the year 2005. NATA is the most comprehensive resource on potential human exposure to and risk of adverse health effects from HAPs in the United States. Monitoring data are insufficient to characterize HAP concentrations across the country because of the limited number of monitors, and because concentrations of many HAPs may vary considerably within a metropolitan area or region.

Under NATA, EPA develops modeled estimates of ambient concentrations of HAPs using estimated emissions data from major, area, onroad mobile, and non-road mobile sources. These emissions data are collected and updated periodically, and are maintained in an emissions inventory. The original NATA was developed using emissions data for the year 1996. Since the initial release, EPA has developed additional estimates of ambient air concentrations of HAPs using updated emissions inventories for 1999, 2002, and 2005. NATA's computer modeling approach has the advantage of allowing estimation of HAP concentrations at locations throughout the United States, rather than in just those locations that have HAP monitors. However, compared with monitoring, the computer model requires estimating quantities of HAP emissions, estimating locations of HAP emissions sources, and modeling the dispersion of HAPs in the atmosphere after they have been emitted.

The most recent assessment developed estimated ambient concentrations of 179 air toxics for the year 2005. A computer model provided estimates for every census tract in the United States. The modeled estimates generally are consistent with the limited set of ambient air toxics monitoring data, although at many locations the model estimates for some HAPs are lower than measured concentrations.[27] The 2005 NATA estimates do not reflect any changes in emissions that may have occurred since 2005 due to new regulations, new technologies, changes in economic activity, or changes in the vehicle fleet and vehicle miles traveled.

Health Benchmarks for Hazardous Air Pollutants

Indicator E4 presents comparisons of modeled concentrations of HAPs in ambient air for 2005 with three health benchmark concentrations derived from scientific assessments conducted by EPA and other environmental agencies.[28] EPA uses the three benchmark risk levels to identify HAPs that are of priority concern.[29]

Two benchmarks reflect potential cancer risks, at levels of 1-in-100,000 risk and 1-in-10,000 risk. If a particular hazardous air pollutant is present in ambient air at a 1-in-100,000 benchmark concentration, for example, it is estimated that one additional case of cancer would occur in a population of 100,000 people exposed for a lifetime. The comparison to the cancer risk benchmark incorporates data for all HAPs considered carcinogenic to humans, likely carcinogenic to humans, or with suggestive evidence of carcinogenicity. The majority of HAPs included in the comparison to the cancer risk benchmarks are considered "carcinogenic to humans" or "likely carcinogenic to humans."[30]

The third benchmark concentration corresponds to the level at which exposure to the hazardous air pollutant is estimated to be of minimal risk for adverse non-cancer health effects; exposures above this benchmark may be associated with adverse health effects such as respiratory or neurological effects. Due to variation in human response to HAP exposure and uncertainty in the benchmark values, it is not necessarily the case that a person living in a location where this benchmark is exceeded will experience adverse effects. It is also possible that individuals may experience effects at levels below the benchmark level.

The health benchmarks are generally derived from laboratory animal studies, although for some HAPs they are derived from human epidemiological studies of workers exposed on the job. For some HAPs, even the animal studies are very limited and no benchmark has been derived. Health benchmarks were available to assess 87 HAPs as cancer-causing agents and 105 HAPs as agents that cause adverse health effects other than cancer. Some HAPs had benchmarks for both cancer and non-cancer health endpoints; a total of 141 air toxics were used in calculating the indicator.

Because they are typically based on studies of adults or mature laboratory animals, the three benchmarks generally reflect health risks to adults, rather than potential risks to children or risks in adulthood stemming from childhood exposure. Benchmarks for non-cancer effects incorporate assumptions that are based on adult respiratory physiology (i.e., breathing rates and lung structure); benchmarks for some HAPs would be lower if they were adjusted for children's respiratory physiology.[31]

Under a policy adopted in 2005, EPA adjusts risk estimates for certain carcinogens to account for increased risks associated with exposures during early life.[32] This adjustment has been applied to the cancer benchmarks for PAHs, acrylamide, benzidine, and ethyl carbamate. Benchmark values for other HAPs that are suspected carcinogens receive no adjustment for

potential elevated risks from early-life exposures because they do not meet the criteria of the EPA policy or lack sufficient data to support application of the adjustment.

Further, the benchmarks reflect risks of continuous exposure over the course of a lifetime. Potential risks from higher concentrations experienced over a short amount of time (one day, one hour, or less) may in some cases trigger immediate responses, such as asthma attacks or effects on the central nervous system are not addressed by these benchmarks.

Finally, the benchmark values for HAP s are uncertain to varying degrees, due to data limitations and the lag in time between when new studies become available and the completion of updated assessments by EPA and other government agencies.

Data Presented in the Indicator

Indicator E4 presents the percentage of children living in census tracts where estimated 2005 HAP concentrations exceeded benchmark levels for cancer (at levels of 1-in-100,000 risk and 1-in-10,000 risk) and for other (non-cancer) adverse health effects. The indicator is calculated by comparing the estimated HAP concentrations for each U.S. census tract in 2005 to each of the benchmark concentrations. Census tracts are geographic areas within U.S. counties that vary in size and generally have 1,500 to 8,000 residents, with a typical population of 4,000 residents.

The comparison to the cancer risk benchmark sums up data for all carcinogenic HAPs. The comparison to the benchmark for other adverse health effects considers only individual HAPs; that is, a county is considered to exceed this benchmark if the modeled concentration for any single HAP exceeds the corresponding non-cancer benchmark for that HAP, but it does not consider adverse effects of combinations of HAPs.

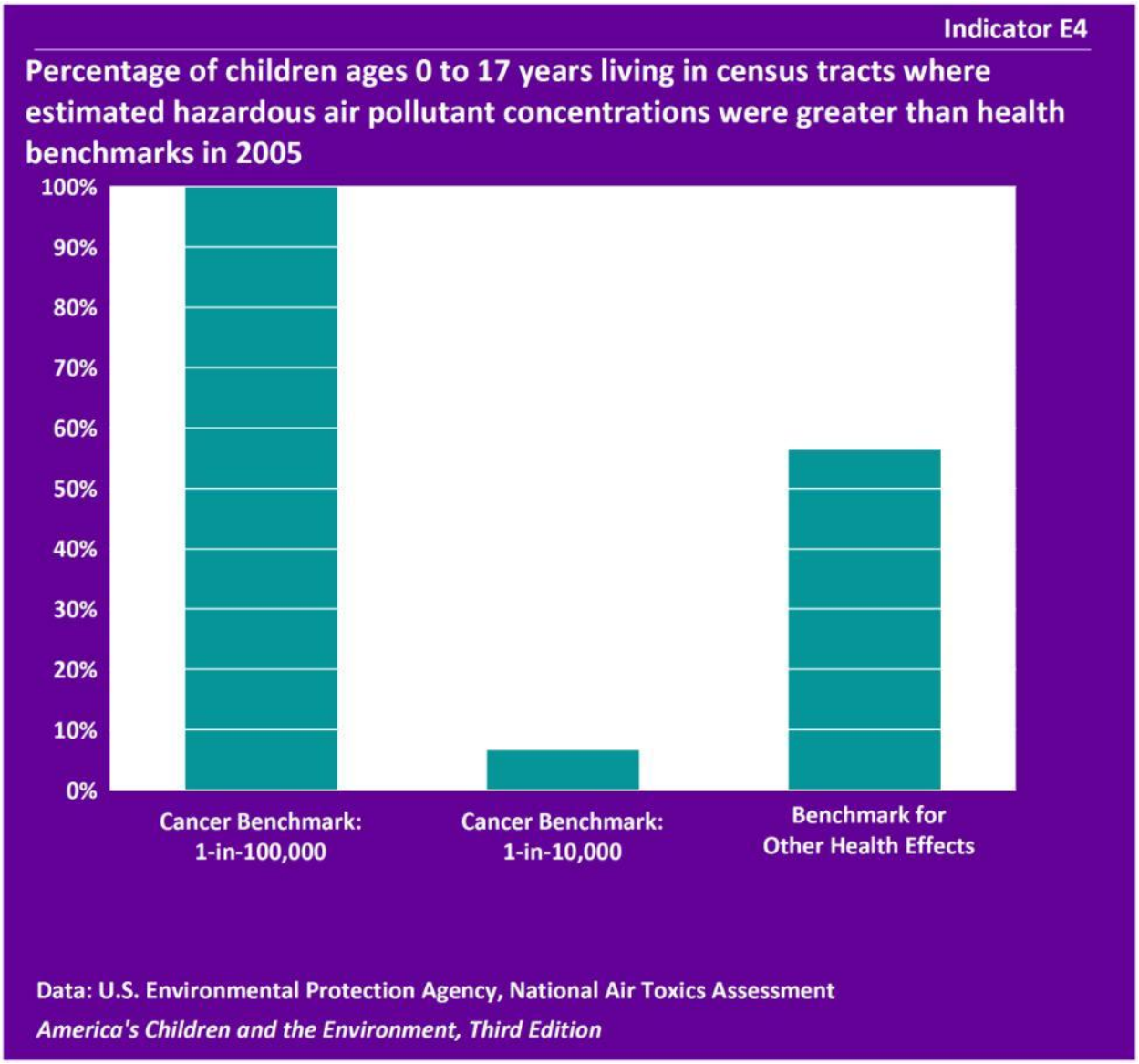
Available information indicates that the NATA estimates of ambient HAP concentrations tend to be similar to or lower than actual HAP concentrations.[27] To the extent that underestimation occurs, the percentage of children living in census tracts exceeding the benchmark levels may be understated. In addition, the indicator does not differentiate between census tracts in which the benchmarks are exceeded by a large margin and those in which estimated HAP concentrations are just above the benchmark concentrations. The indicator presents results only for 2005, and does not compare results across assessment years, such as between 1999 and 2005, because each update of the assessment brings new improvements to methods. For example, improvements to the emissions estimation methodologies made in the 2005 assessment were not applied to the earlier versions, so the ambient concentration estimates are not entirely comparable between years.

Actual exposures may differ from ambient concentrations. Indoor concentrations of HAPs from outdoor sources may be slightly lower than ambient concentrations, although they can be significantly higher if any indoor sources are present.[33-36] Levels of some hazardous pollutants may be substantially higher inside cars and school buses,[37-39] and those higher levels would increase the risks.

In addition, this indicator only considers exposures to air toxics that occur by inhalation. For many air toxics, dietary exposures are also important. Air toxics that are persistent in the environment settle out of the atmosphere onto land and water, and then may accumulate in fish and other animals in the food web. For HAPs that are persistent in the environment and accumulate significantly in food, exposures through food consumption typically are greater than inhalation exposures. HAPs for which food chain exposures are important include mercury, dioxins, and PCBs.[40-42]

The comparison of ambient HAP concentrations in 2005 to the health benchmarks is not equivalent to an estimate of risk to the population from chronic HAP exposure. Actual risks to health depend on concentrations of HAPs in many environments over an extended period of time. Ambient concentrations will change over time as the mix of sources changes (e.g., due to businesses opening and closing), vehicle use changes (e.g., more cars and trucks traveling longer distances), and regulatory controls are applied. In addition, children spend most of their time indoors at home, at school, or at child care centers, and pollutant concentrations in indoor environments may be greater or lesser than the modeled ambient concentrations.

In addition to the indicator presented in the figure, which is based on where children live, the same statistics are calculated based on where children's schools are located (see data tables). Exposures at school are an important consideration, as children spend an average of 33 hours per week in school.[43] The data tables also provide indicator values by race/ethnicity and income, based on where children live.



Data characterization

- Data for this indicator are obtained from EPA's National Air Toxics Assessment computer model predictions of hazardous air pollutant (HAP) concentrations in outdoor air.
- The model produces estimates of HAP concentrations from emissions data for all census tracts in the United States (census tracts typically have about 4000 residents each).

- In 2005, nearly all children lived in census tracts in which HAP concentrations combined to exceed the 1-in-100,000 cancer risk benchmark.
- Seven percent of children lived in census tracts in which HAPs combined to exceed the 1-in-10,000 cancer risk benchmark. The pollutants that contributed most to this result were formaldehyde, benzene, acetaldehyde, carbon tetrachloride, and hexavalent chromium. Formaldehyde, benzene, and hexavalent chromium are considered to be carcinogenic to

humans,[5,10,13] and acetaldehyde and carbon tetrachloride are considered likely to be carcinogenic to humans.[21,22]

- Fifty-six percent of children lived in census tracts in which at least one HAP exceeded the benchmark for health effects other than cancer. In almost all cases, this result was attributable to the pollutant acrolein, which is a respiratory irritant. More than 90% of acrolein emissions are from wood-burning fires and mobile sources such as cars, trucks, buses, planes, and construction equipment.
- Exposures to diesel particulate matter from diesel engine emissions are not included in this indicator due to uncertainty regarding the appropriate values to use as cancer benchmarks. Some studies have found that cancer risks from diesel particulate matter exceed those of the HAPs considered in this indicator.[44] Although EPA does not endorse any particular cancer benchmark value for diesel particulate matter, if the State of California's benchmark for diesel particulate matter were used in this analysis, 73% of children would live in census tracts where HAP estimates combined to exceed the 1-in-10,000 cancer risk benchmark.
- In 2005, all children's schools were located in census tracts where HAPs concentrations combined to exceed the 1-in-100,000 cancer risk benchmark. Six percent of children attended schools in census tracts where the HAPs concentrations exceeded the higher 1-in-10,000 cancer risk benchmark.
- Fifty-seven percent of children attended schools that were located in census tracts where at least one HAP exceeded the benchmark for health effects other than cancer.

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Indoor Environments

Children spend most of their time in indoor environments, including homes, schools, child care facilities, and other buildings.[1] The chemicals found indoors or measured in indoor air are numerous and diverse. Hundreds of chemicals have been measured in indoor air, including multiple pesticides, fragrance-related compounds, polychlorinated biphenyls (PCBs), phthalates, combustion byproducts, carbon monoxide, benzene, formaldehyde, and other compounds.[2-4] Pollutants in indoor environments can come from many different sources, including combustion sources such as furnaces, gas stoves, fireplaces, and cigarettes; building materials and furnishings such as treated wood, paints, furniture, carpet, and fabrics; consumer goods such as electronics and toys; cleaning products, pesticides, and other products used for maintenance of the home or building; and products used for hobbies, science projects, arts and crafts projects, and other activities.

Children may also be routinely exposed to chemical contaminants that accumulate in dust, including lead, nicotine, pesticides, brominated flame retardants, phthalates, and perfluorinated chemicals.[3,5-9] Many pesticides and other chemicals that break down relatively quickly outdoors are much more persistent and long-lasting indoors, where they are less exposed to natural elements such as sunlight, moisture, and microorganisms that can accelerate the breakdown of chemicals.[10-12]

Infants and small children may have the highest exposure to house dust contaminants due to their frequent and extensive contact with floors, carpets, and other surfaces where dust gathers, as well as their frequent hand-to-mouth activity. However, children of all ages (as well as adults) are likely to be exposed to dust contaminants through hand-to-mouth activity[1,13] and other ingestion pathways, such as the settling of dust onto food and food preparation surfaces in the kitchen.

The indoor environments of personal cars and school buses are also important to children's exposure, as a child can spend up to an average of 84 minutes per day in a vehicle, depending on his or her age.[1] School bus cabins can have levels of fine particulate matter ($PM_{2.5}$) four times higher than levels in ambient air.[14] In addition, children riding school buses in urban areas are likely to be exposed to elevated levels of benzene, formaldehyde, and other pollutants in motor vehicle emissions. It is estimated that school buses commuting through congested urban areas may contribute up to 30% of a child's daily exposure to diesel engine-related pollutants.[15]

Adult smoking in personal cars can have a significant impact on children's environmental tobacco smoke exposures, as the air in smokers' cars tends to have significantly higher nicotine concentrations than that in non-smokers' cars.[16] Smoking in cars also leaves nicotine residues that may linger in dust and surfaces within smokers' cars, leading to continued exposure even after the practice of smoking within the car has ceased.[17]

Pollutants in indoor environments can also come from outside sources. For example, pollutants in outdoor air will penetrate to the indoor environment,[18,19] and contaminants from

workplaces, streets, or lawns may be carried into the home on people's shoes or clothing.[20,21] Some contaminants in drinking water can enter indoor air through uses of hot water such as showering.[22,23] In areas where groundwater is contaminated, chemicals may enter indoor environments via vapor intrusion.[24,25] Radon, a gaseous radioactive element that causes lung cancer, is found in soils and can enter homes through cracks in the foundation and other entry points.[26]

Indoor air pollutants from biological sources such as mold; dust mites; pet dander (skin flakes); and droppings and body parts from cockroaches, rodents, and other pests or insects are commonly found in children's homes.[27-30] These contaminants are important because they can lead to allergic reactions, exacerbate existing asthma, and have been associated with the development of respiratory symptoms.[28-31]

Two indoor environmental contaminants for which there is extensive evidence of children's health effects are environmental tobacco smoke and lead. The following indicators present data on environmental tobacco smoke and lead dust hazards in children's homes, because they are well-established indoor hazards to children's health and because they have nationally representative data available for more than one point in time. Other indoor environmental hazards in children's homes generally lack nationally representative data necessary for development of indicators that can identify any changes over time. Unlike many outdoor pollutants, indoor pollutants are not regulated or systematically monitored in residential settings, and data collection for indoor pollutants is much more limited. Indicator E5 presents data on environmental tobacco smoke, based on national survey data of homes with young children where someone smokes regularly. Indicator E6 presents data on lead dust hazards in children's homes. Further information on these issues is provided in the following sections. In addition, indoor environments in children's schools and in child care facilities are discussed in the Supplementary Topics section of this report.

Environmental Tobacco Smoke

Environmental tobacco smoke (ETS), commonly referred to as secondhand smoke, is a complex mixture of gases and particles and includes smoke from burning cigarettes, cigars, and pipe tobacco (sidestream smoke), as well as exhaled mainstream smoke.[32] There are at least 250 chemicals in ETS that are known to be toxic or carcinogenic, including acrolein, ammonia, benzene, carbon monoxide, formaldehyde, hydrogen cyanide, nicotine, nitrogen oxides, and sulfur dioxide.[32,33] In 1992, EPA classified ETS as a known human carcinogen.[34] Children can be exposed to ETS in their homes or in places where people are allowed to smoke, such as some restaurants in some locations throughout the United States.

According to the U.S. Surgeon General, there is no safe level of exposure to ETS, and breathing even a small amount can be harmful to human health.[32] The Surgeon General has concluded that exposure to ETS causes sudden infant death syndrome (SIDS), acute lower respiratory infection, ear problems, and more severe asthma in children. Smoking by parents causes

respiratory symptoms and slows lung growth in their children.[32] Young children appear to be more susceptible to the respiratory effects of ETS than are older children.[29,34,35]

The exposure of a pregnant woman to ETS can also be harmful to her developing fetus. The Surgeon General has determined that exposure of pregnant women to ETS causes a small reduction in mean birth weight, and that the evidence is suggestive (but not sufficient to infer causation) of a relationship between maternal exposure to environmental tobacco smoke during pregnancy and preterm delivery.[32] In addition, the Surgeon General concluded the evidence is suggestive but not sufficient to infer a causal relationship between prenatal and postnatal exposure to ETS and childhood cancer.[32]

Exposure to ETS in the home is influenced by adult behaviors, including the decisions to smoke at home and to allow visitors to smoke inside the home. Children living in homes with smoking bans have significantly lower levels of cotinine (a biological marker of exposure to ETS) in urine than children living in homes without smoking bans.[36] Household smoking bans can significantly decrease children's exposures to ETS, but do not completely eliminate them, especially in multi-unit housing where ETS from other apartments may infiltrate through seepage in walls or shared ventilation systems.[37-39] Furthermore, children may be exposed to toxic residues that remain from ETS in dust and on surfaces inside the home for weeks or months after smoke has cleared from the air.[6,40-43] These residues, referred to as "third-hand smoke," may be re-emitted into the gas phase or may react with other compounds to form secondary pollutants.[40,43] The risk of exposure to third-hand smoke may be particularly high for infants, due to their close proximity to contaminated objects such as blankets, carpets, and floor surfaces, and their frequent hand-to-mouth activity.[6]

Parental smoking status inside the home greatly affects children's exposures to ETS, but research suggests a difference in impact between maternal and paternal smoking. Maternal smoking is associated with higher cotinine levels in children, and maternal smoking appears to have a greater effect on lower respiratory illnesses than does paternal smoking.[32]

In recent years there has been a significant decline in children's exposures to ETS.[44] This reduction is in part attributable to a decline in the percentage of adults who smoke, and is likely related to increased restrictions on smoking at workplaces and other public places, as well as efforts to reduce the exposure of nonsmokers in homes.[44] In 2010, an estimated 19.3% of adults were current smokers, down from 24.7% in 1997.[45,46] In addition, the prevalence of smoke-free households increased from 43% of U.S. homes in 1992–1993 to 72% in 2003.[47] Children living in homes with smoking bans have significantly lower levels of cotinine than children living in homes without smoking bans.[36] Recent studies also suggest that smoking bans in workplaces and other public places can reduce the number of asthma-related emergency room visits and hospitalizations, including among children when legal bans lead to an increase in voluntary smoking bans in homes.[48,49] However, despite the increasing numbers of adults disallowing smoking in the home, approximately 34% of children live in a home with at least one smoker as of 2009.[50]

Lead in House Dust

The ingestion of lead-contaminated house dust, soil, and water is the primary pathway of current childhood exposure to lead.[51] Children have a greater risk of exposure to lead-contaminated dust than that of adults, due to their frequent and extensive contact with floors, carpets, and other surfaces where dust gathers, as well as their high rate of hand-to-mouth activity. Additionally, lead-contaminated dust particles are more readily absorbed into the body than soil or paint chips, and children's bodies absorb up to 10 times more ingested lead than adults do as a result of their less-developed gastrointestinal pathways.[52] Children living in homes with higher levels of lead-contaminated dust tend to have higher blood lead levels.[53-58]

Lead dust is composed of fine particles of soil, paint, and other settled industrial or automotive emissions from the outdoor and indoor air.[59] Residences with deteriorated lead-based paint tend to have higher levels of lead in house dust and the surrounding soil.[51,60] Deteriorated lead-based paint that is cracked, peeling, or chipped can be ingested directly by children or can mix with and contaminate house dust, which can also be ingested.[61] Normal wear as the result of cleaning activities or repeated surface friction can lead to further deterioration and the release of lead-based paint particles.[62] Any house built before 1978 may contain lead-based paint. As of the year 2000, approximately 38 million older housing units in the United States still contained lead-based paint.[51]

Home maintenance and renovation activities that disturb lead-based paint, such as sanding, scraping, cutting, and demolition, create hazardous lead dust and chips and have been associated with higher levels of lead dust and blood lead in children.[60,63] Beginning in April 2010, all contractors performing renovation, repair, and painting projects that disturb lead-based paint in pre-1978 homes and child-occupied facilities, such as child care facilities and preschools, must be certified and follow specific work practices to prevent lead contamination.[60] Lead-contaminated soil is another contributor to lead in house dust. Known sources of lead in soil include historical airborne emissions from leaded gasoline use, emissions from industrial sources such as smelters, and lead-based paint. Current sources of lead in ambient air in the United States include smelters, ore mining and processing, lead acid battery manufacturing, and coal combustion activities, such as electricity generation.[58] Lead-contaminated dust and soil from the outdoors can be transported into the home after becoming airborne via soil resuspension, or can be tracked into the home by occupants or family pets.[52]

The National Toxicology Program (NTP) has concluded that childhood lead exposure is associated with reduced cognitive function.[64] Children with higher blood lead levels generally have lower scores on IQ tests[55,65-70] and reduced academic achievement.[64] The NTP has also concluded that childhood lead exposure is associated with attention-related behavioral problems (including inattention, hyperactivity, and diagnosed attention-deficit/hyperactivity disorder) and increased incidence of problem behaviors (including delinquent, criminal, or antisocial behavior).[64]

Until recently, the Centers for Disease Control and Prevention (CDC) defined a blood lead level of 10 micrograms per deciliter (µg/dL) as "elevated." This definition was used to identify children for blood lead case management.[71,72] However, no level of lead exposure has been identified that is without risk of deleterious health effects.[58] CDC's Advisory Committee on Childhood Lead Poisoning Prevention (ACCLPP) recommended in January 2012 that the 97.5th percentile of children's blood lead distribution (currently 5 $\mu g/dL$) be defined as "elevated" for purposes of identifying children for follow up activities such as environmental investigations and ongoing monitoring.[73] CDC has adopted the ACCLPP recommendation.[74] CDC specifically notes that "no level of lead in a child's blood can be specified as safe,"[75] and the NTP has concluded that there is sufficient evidence for adverse health effects in children at blood lead levels less than 5 $\mu g/dL$.[64]

The current federal standards indicate that floor and window lead dust should not exceed 40 micrograms of lead per square foot (µg/ft^2) and 250 µg/ft^2, respectively, in order to protect children from developing "elevated" blood lead levels as formerly defined by the CDC. EPA is currently reviewing the lead dust standards to determine whether they should be lowered, based on indications from more recent epidemiological studies that the current standards may not be sufficiently protective of children.[76]

Childhood blood lead and house dust lead levels in the United States differ across groups in the population, such as those defined by socioeconomic status, race/ethnicity,[51,53,77] and geographic location. Children living in poverty and Black non-Hispanic children tend to have higher blood lead levels[53,78] and higher levels of lead-contaminated dust in the home than do White non-Hispanic children.[77] Blood lead levels tend to be higher for children living in older housing, because older housing units are more likely to contain lead-based paint.[77,79] Additionally, housing in the Northeast and Midwest has twice the prevalence of lead-based paint hazards compared with housing in the South and West,[59] because of the older housing stock in those areas.

Indicator E5: Percentage of children ages 0 to 6 years regularly exposed to environmental tobacco smoke in the home, by family income, 1994, 2005, and 2010

About the Indicator: Indicator E5 presents the percentage of children ages 0 to 6 years regularly exposed to environmental tobacco smoke (ETS) in the home. The data are from a national survey that collects health information from a representative sample of the population. The survey provides data on children exposed to ETS in the home on four or more days per week for the years 1994, 2005, and 2010. The focus is on children ages 6 years and under because these younger children have been specifically identified as more susceptible to the effects of tobacco smoke.

National Health Interview Survey

Comparable, nationally representative data on children living in homes where someone smokes regularly come from the National Health Interview Survey (NHIS) for 1994, 2005, and 2010. The NHIS is a large-scale household interview survey of a representative sample of the civilian noninstitutionalized U.S. population, conducted by the National Center for Health Statistics. In 1994, interviews were conducted with household adults representing about 5,450 children ages 0 to 6 years, and ETS exposure information was reported for about 5,390 of those children. In 2005, interviews were conducted with household adults representing about 10,100 children ages 0 to 6 years, and ETS exposure information was reported for about 7,800 of those children. In 2010, interviews were conducted with household adults representing about 9,350 children ages 0 to 6 years, and ETS exposure information was reported for about 6,900 of those children. Questions related to smoking in the home are included in the NHIS only in selected years. In 1994, the NHIS asked, "Does anyone who lives here smoke cigarettes, cigars, or pipes anywhere inside this home?" Similarly, in 2005 and 2010, the NHIS asked, "In a usual week, does ANYONE who lives here, including yourself, smoke cigarettes, cigars, or pipes anywhere inside this home?" If the answer was positive, participants were asked how many days per week smoking usually occurred anywhere inside the home. The NHIS also included questions about smoking in the home in the 1998 survey, but the questions used in 1998 provide data that are not directly comparable to the 1994, 2005, and 2010 data.

Data Presented in the Indicator

Indicator E5 presents data from NHIS for the percentage of children ages 0 to 6 years living in homes where someone smokes on a regular basis (defined as four days or more per week). Studies have found that questionnaire data on smoking in the home are relatively accurate in predicting serum levels of cotinine (a metabolite of nicotine used as a marker of ETS exposure) in children,[80,81] and researchers have used these data to associate ETS exposure with adverse effects on childhood lung function and other health outcomes.[32] However, comparisons of questionnaire data with measures of serum cotinine in children suggest that questionnaires may underestimate actual exposure to ETS, particularly in multi-unit housing or in cases where visitors and other non-family members may smoke in the home.[32,39,82-84]

While the indicator provides information on the presence and number of days per week of smoking in the home, it does not indicate the intensity of smoking (e.g., the number of cigarettes smoked in the home per day). Furthermore, children exposed to ETS at home fewer than four days per week are not included in this indicator, but may also experience adverse health effects since no level of exposure to ETS is without a risk to health.

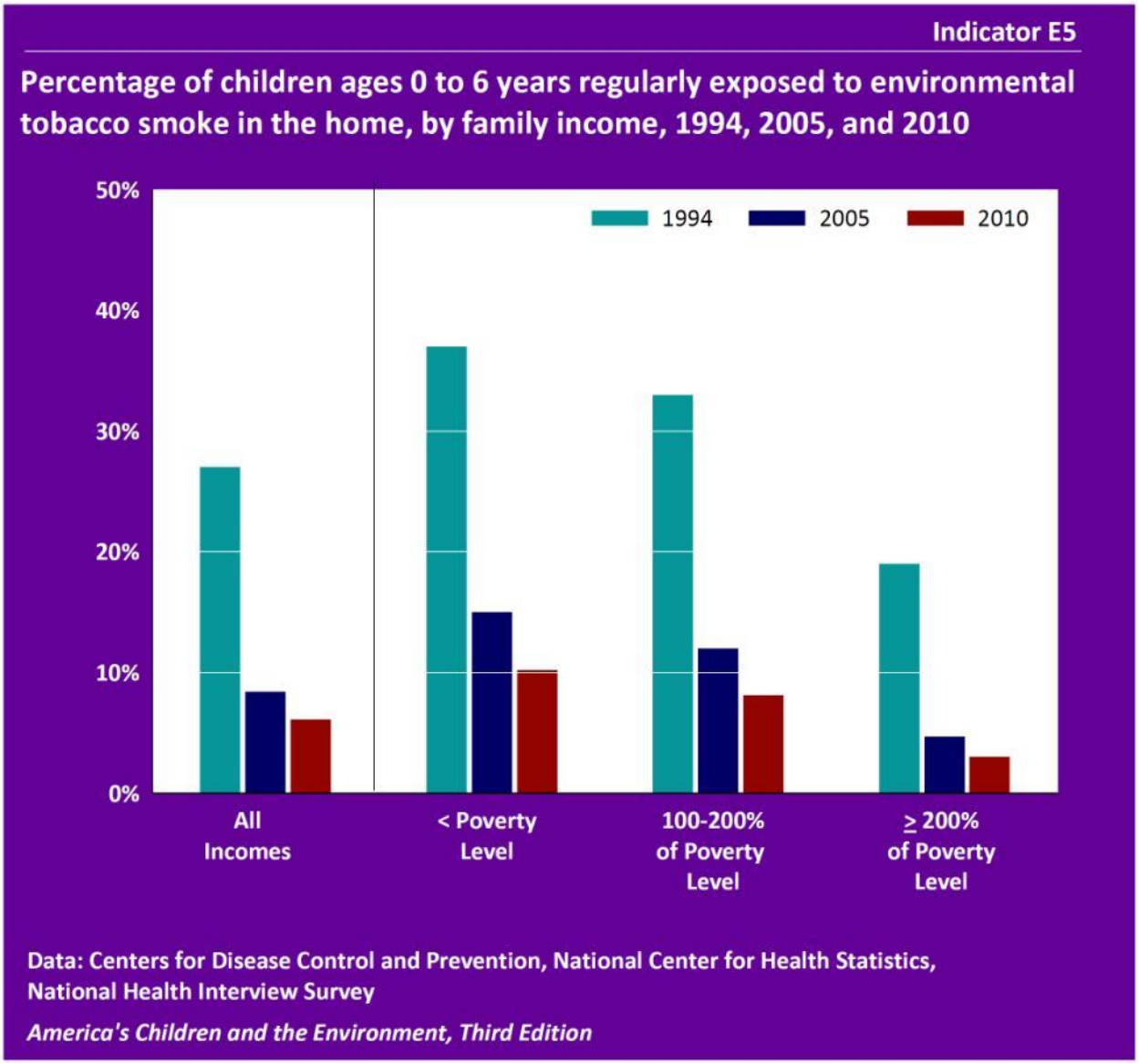
We focus on children ages 0 to 6 years because these younger children have been specifically identified as more susceptible to the effects of tobacco smoke and are targeted by the indicator used in the federal government's Healthy People 2010 initiative.[85] Children ages 6 years and under also have less control over their environment and are likely to spend more time in close proximity to adult caregivers.[32] Children of all ages, however, may be affected by exposure to ETS.

The indicator presents data on children's exposures to ETS in the home for 1994, 2005, and 2010, based on family income level. Additional information regarding ETS exposures for different race/ethnicity groups is presented in Table E5a.

Statistical Testing

Statistical analysis has been applied to the 2010 data to evaluate differences in indicator values between demographic groups. These analyses use a 5% significance level, meaning that a conclusion of statistical significance is made only when there is no more than a 5% probability that the observed difference occurred by chance ($p \leq 0.05$). A finding of statistical significance depends on the numerical difference in the indicator value between two groups, the number of observations in each group, and various aspects of the survey design. For example, the statistical test is more likely to detect a difference between two groups when data have been obtained from a larger number of people in those groups. It should be noted that when statistical testing is conducted for differences among multiple demographic groups (for example, considering both race/ethnicity and income level), the large number of comparisons involved increases the probability that some differences identified as statistically significant may actually have occurred by chance.

A finding of statistical significance is useful for determining that an observed difference was unlikely to have occurred by chance. However, a determination of statistical significance by itself does not convey information about the magnitude of the difference in indicator values or the potential difference in risk of associated health outcomes. Furthermore, a lack of statistical significance means only that occurrence by chance cannot be ruled out. Thus a conclusion about statistical significance is only part of the information that should be considered when determining the public health implications of differences in indicator values.



Data characterization

- Data for this indicator are obtained from an ongoing annual survey conducted by the National Center for Health Statistics.
- Survey data are representative of the U.S. civilian noninstitutionalized population.
- In 1994, 2005, and 2010, an adult survey participant in each sampled household was asked whether any resident smokes inside the home and the number of days per week that smoking occurred.

- In 2010, 6% of children ages 0 to 6 years lived in homes where someone smoked regularly, compared with 27% in 1994.
- Children living in homes with family incomes below the poverty level were more likely than their peers at higher income levels to be living in homes where someone smoked regularly. In 2010, 10% of children below the poverty level lived in homes where someone smoked

regularly, compared with 8% of children in homes with incomes between 100–200% of poverty level, and 3% of children in homes with incomes at least twice the poverty level.

- The differences between children in homes with family incomes below the poverty level and children in homes with family incomes at or above the poverty level were statistically significant.
- In 2010, 20% of White non-Hispanic children below poverty lived in homes where someone smoked regularly, compared with 10% of Black non-Hispanic children and 2% of Hispanic children living below poverty. (See Table E5a.) These differences were statistically significant.

Indicator E6: Percentage of children ages 0 to 5 years living in homes with interior lead hazards, 1998–1999 and 2005–2006

About the Indicator: Indicator E6 shows the percentage of children ages 0 to 5 years who lived in homes with interior lead-based paint hazards. The data are from two nationally representative surveys of homes conducted in 1998–1999 and 2005–2006. The surveys involved collection of dust, soil, and paint samples from homes and measurement of the lead levels in these samples. The focus of the indicator is on children ages 0 to 5 years, due to the elevated exposures that occur during early childhood and the sensitivity of the developing brain to the effects of lead.

NSLAH/AHHS

The United States Department of Housing and Urban Development (HUD) has conducted two nationally representative surveys of housing in the United States to assess children's potential household exposure to lead and other contaminants. The American Healthy Homes Survey (AHHS) was conducted from 2005–2006 to update the National Survey of Lead and Allergens in Housing (NSLAH), which was conducted from 1998–1999. AHHS also included measurements of arsenic, pesticides, and mold; however, these substances were not measured in the earlier NSLAH.

Samples of paint, dust, and soil were taken from 831 total housing units (184 units with children ages 0 to 5 years) in NSLAH, and 1,131 total housing units (206 units with children ages 0 to 5 years) in AHHS. The lead sampling components of AHHS were designed to be very similar to NSLAH so that results of the two studies could be compared.

Lead-Based Paint Hazards

Samples collected from the housing units surveyed in NSLAH and AHHS were analyzed to determine their lead content. HUD then compared these measured lead levels to federal guidelines to identify homes with lead-contaminated dust, deteriorated lead-based paint, and lead-contaminated soil hazards.

EPA has established Residential Lead Hazard Standards under Title X of the Toxic Substances Control Act (TSCA), section 403, for identifying lead-based paint hazards in all housing built before 1978. These standards were adopted by HUD under the Lead Safe Housing Act, which applies to all federally owned or assisted housing in the United States. According to these regulations, a lead-based paint hazard is the presence of deteriorating lead-based paint, lead-contaminated dust, or lead-contaminated soil above federal standards.

For lead-contaminated dust, there are separate standards for dust on the floor and dust on windowsills. Floor dust samples should not have more than 40 micrograms of lead per square foot ($\mu g/ft^2$) and window dust samples should not have more than 250 $\mu g/ft^2$.[61,86]

Additionally, current federal standards qualify a significantly deteriorated lead-based paint hazard as the deterioration of an area of lead-based paint greater than 20 square feet (exterior) and 2 square feet (interior) for large-surface items, such as walls and doors; or damage to more than 10% of the total surface area of small-surface components—such as windowsills, baseboards, and trim—with lead-based paint.

The level of deterioration is an important variable in determining exposure. The presence of lead-based paint alone is not necessarily indicative of a significant hazard; except during renovations, maintenance, and similar disturbances, intact lead-based paint is believed to pose very little risk to occupants.[87] However, deteriorated lead-based paint that is cracked, peeling, or chipped can be ingested directly by children or can contaminate house dust that can be inhaled or ingested by children.[61]

Data Presented in the Indicator

Indicator E6 presents the percentage of children ages 0 to 5 years who lived in homes with interior lead-based paint hazards, using data from NSLAH and AHHS and three hazard definitions.

The first hazard definition, "interior lead dust," presents the percentage of children ages 0 to 5 years living in homes with a lead dust hazard, based on the number of homes with dust containing levels of lead that exceeded the levels defined by EPA's Residential Lead Hazard Standards (established under Title X of TSCA, section 403). The second hazard definition, "interior deteriorated lead-based paint," displays the percentage of children ages 0 to 5 years who lived in homes with significantly deteriorated lead-based paint indoors as defined by EPA's Residential Lead Hazard Standards. The last definition, "either interior lead dust or interior deteriorated lead-based paint," represents the percentage of children living in homes with an interior dust hazard, a deteriorated lead-based paint hazard, or both.

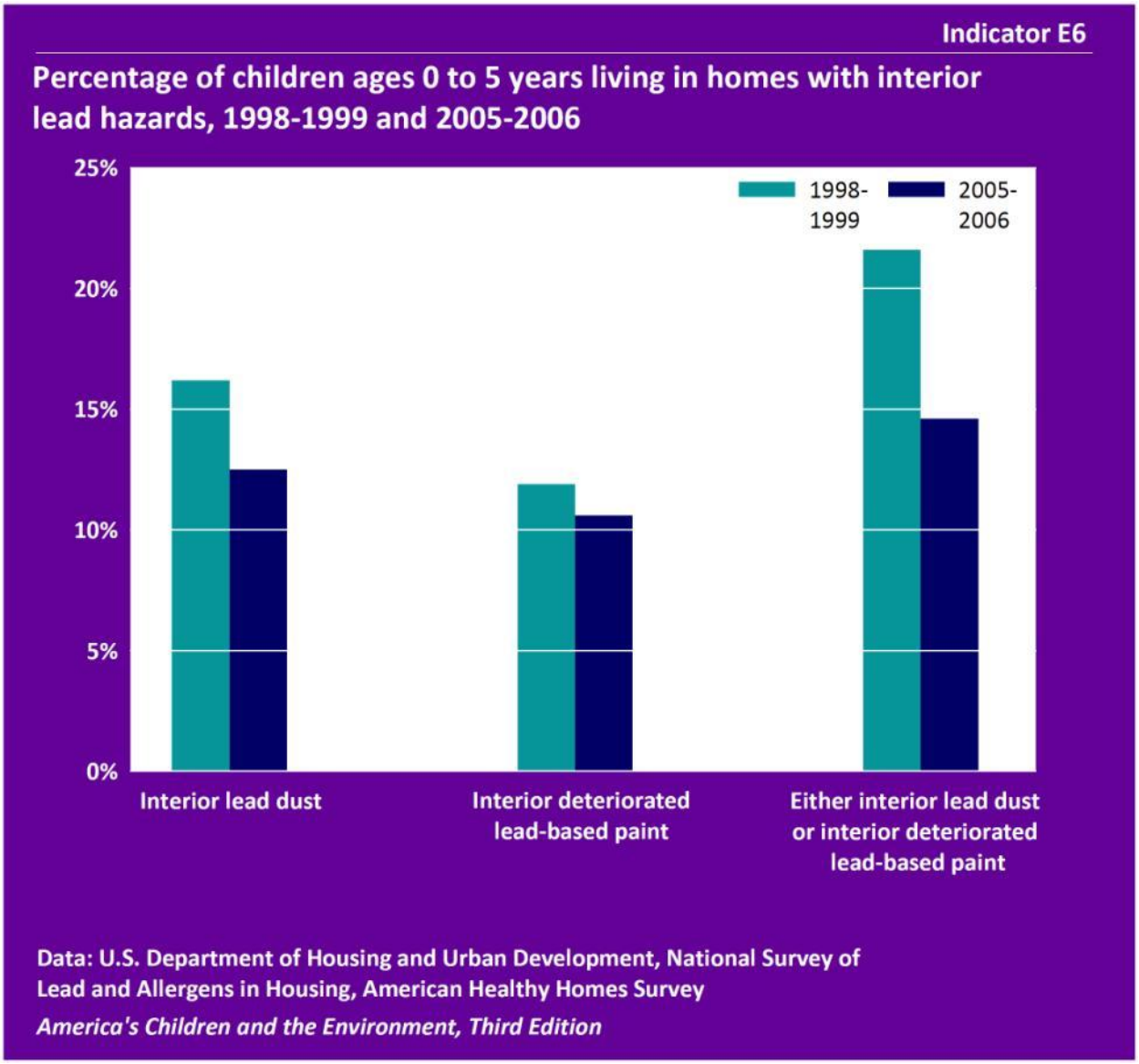
This indicator represents the potential for children's indoor exposure to lead based solely on the percentage of children ages 0 to 5 years living in homes with levels of lead-based paint and dust above federal standards. The indicator does not represent differences in paint lead levels, paint deterioration levels, or the amount of lead in the dust above the standards. It also does not account for the possibility that children living in homes with levels of lead-based paint and dust below federal standards may still have some exposure to lead. Furthermore, while this indicator focuses on children ages 0 to 5 years, older children may also experience health effects from exposure to lead.

Survey records identify the race/ethnicity and income level of survey respondents; however, estimates of lead hazards in the home for children ages 0 to 5 years broken out by race/ethnicity and income are not statistically reliable, due to the relatively small number of homes in each group. Therefore, the indicator provides data only for all children ages 0 to 5 years combined.

Statistical Testing

Statistical analysis has been applied to Indicator E6 to evaluate differences over time in the indicator values (for example, percentage of children living in homes with lead-contaminated dust). These analyses use a 5% significance level, meaning that a conclusion of statistical significance is made only when there is no more than a 5% probability that the observed difference occurred by chance ($p \leq 0.05$). The statistical analysis depends on the numerical difference in the indicator value over time, the number of observations in each time period, and various aspects of the survey design. For example, the statistical test is more likely to detect a change over time when data have been obtained from a larger number of people in each time period.

A finding of statistical significance is useful for determining that an observed difference was unlikely to have occurred by chance. However, a determination of statistical significance by itself does not convey information about the magnitude of the difference in indicator values or the potential difference in risk of associated health outcomes. Furthermore, a lack of statistical significance means only that occurrence by chance cannot be ruled out. Thus a conclusion about statistical significance is only part of the information that should be considered when determining the public health implications of changes over time.



Data characterization

- Data for this indicator are obtained from two surveys of U.S. homes conducted by the Department of Housing and Urban Development.
- Surveyed homes were representative of permanently occupied, non-institutional housing units in the United States in which children may live. Only surveyed homes with children ages 0 to 5 years were included in calculation of this indicator.
- Lead was measured in samples of paint and dust collected from the surveyed homes.

- In 2005–2006, 13% of children ages 0 to 5 years lived in homes with an interior lead dust hazard, compared with 16% in 1998–1999.
- In 2005–2006, 11% of children ages 0 to 5 years lived in homes with an interior deteriorated lead-based paint hazard, compared with 12% in 1998–1999.

- In 2005–2006, 15% of children ages 0 to 5 years lived in homes with either an interior lead dust hazard or an interior deteriorated lead-based paint hazard, compared with 22% in 1998–1999.
- Changes in percentages between the two surveys were not statistically significant.

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Drinking Water Contaminants

Drinking water sources may contain a variety of contaminants that, at elevated levels, have been associated with increased risk of a range of diseases in children, including acute diseases such as gastrointestinal illness, developmental effects such as learning disorders, endocrine disruption, and cancer.[1-3] Because children tend to take in more water relative to their body weight than adults do, children are likely to have higher exposure to drinking water contaminants.

Drinking water sources include surface water, such as rivers, lakes, and reservoirs;[4] and groundwater aquifers, which are subsurface layers of porous soil and rock that contain large collections of water.[5] Groundwater and surface water are not isolated systems and are continually recharged by each other as well as by rain and other natural precipitation.[6]

Several types of drinking water contaminants may be of concern for children's health. Examples include microorganisms, (e.g., *E. coli, Norovirus, and Giardia)*, inorganic chemicals (e.g., lead, arsenic, nitrates, and nitrites), organic chemicals (e.g., atrazine, glyphosate, trichloroethylene, and tetrachloroethylene), and disinfection byproducts (e.g., chloroform). EPA and the Food and Drug Administration (FDA) are both responsible for the safety of drinking water. FDA regulates bottled drinking water, while EPA regulates drinking water provided by public water systems. EPA sets enforceable drinking water standards for public water systems, and unless otherwise specified, the term "drinking water" in this text refers to water provided by these systems. The drinking water standards include maximum contaminant levels and treatment technique requirements for more than 90 chemical, radiological, and microbial contaminants, designed to protect people, including sensitive populations such as children, against adverse health effects.[2,7] Microbial contaminants, lead, nitrates and nitrites, arsenic, disinfection byproducts, pesticides, and solvents are among the contaminants for which EPA has set health-based standards.

Microbial contaminants include bacteria, viruses, and protozoa that may cause severe gastrointestinal illness.[2] Children are particularly sensitive to microbial contaminants, such as *Giardia, Cryptosporidium, Norovirus*, and *E. coli*, because their immune systems are less developed than those of most adults.[8-14]

Drinking water is a known source of lead exposure among children in the United States, particularly from corrosion of pipes and other elements of the drinking water distribution systems.[15-17] Exposure to lead via drinking water may be particularly high among very young children who consume baby formula prepared with drinking water that is contaminated by leaching lead pipes.[15] The National Toxicology Program has concluded that childhood lead exposure is associated with reduced cognitive function, reduced academic achievement, and increased attention-related behavioral problems.[18]

Fertilizer, livestock manure, and human sewage can be significant contributors of nitrates and nitrites in groundwater sources of drinking water.[19,20] High levels of nitrates and nitrites can cause the blood disorder methemoglobinemia (blue baby syndrome)[21-23] and have been associated with thyroid dysfunction in children[24,25] and pregnant women.[24,26,27] Moderate deficits in maternal thyroid hormone levels during early pregnancy have been linked to reduced childhood IQ scores and other neurodevelopmental effects, as well as unsuccessful or complicated pregnancies.[28]

Arsenic enters drinking water sources from natural deposits in the earth, which vary widely from one region to another, or from agricultural and industrial sources where it is used as a wood preservative and a component of fertilizers, animal feed, and a variety of industrial products.[29] Population studies of health effects associated with arsenic exposure have been conducted primarily in countries such as Bangladesh, Taiwan, and Chile, where arsenic levels in drinking water are generally much higher than in the United States due to high levels of naturally occurring arsenic in groundwater.[30] Long-term consumption of arsenic-contaminated water has been associated with the development of skin conditions and circulatory system problems, as well as increased risk of cancer of the bladder, lungs, skin, kidney, nasal passages, liver, and prostate.[29,31] In many cases, long-term exposure to arsenic begins during prenatal development or childhood, which increases the risk of mortality and morbidity among young adults exposed to arsenic long-term.[32] A review of the literature concluded that epidemiological studies of associations between exposure to arsenic and some adverse health outcomes pertinent to children's health have mixed findings. These include studies of associations between high levels of exposure to arsenic and abnormal pregnancy outcomes, such as spontaneous abortion, still-births, reduced birth weight, and infant mortality, as well as associations between early-life exposure to arsenic and increased incidence of childhood cancer and reduced cognitive function.[33]

Water can contain microorganisms such as parasites, viruses, and bacteria; the disinfection of drinking water to reduce water-borne infectious disease is one of the major public health advances of the 20th century.[34] The method by which infectious agents are removed or chemically inactivated depends on the type and quality of the drinking water source and the volume of water to be treated. Surface water systems are more exposed than groundwater systems to weather and runoff; therefore, they may be more susceptible to contamination.[4,35] Surface and groundwater systems use filtration and other treatment methods to physically remove particles. Disinfectants, such as chlorine and chloramine, ultraviolet radiation, and ozone are added to drinking water provided by public water systems to kill or neutralize microbial contaminants.[36] However, this process can produce disinfection byproducts, which form when chemical disinfectants react with naturally occurring organic matter in water.[37] The most common of these disinfection byproducts are chloroform and other trihalomethanes. Consumption of drinking water from systems in the United States and other industrialized countries with relatively high levels of disinfection byproducts has been associated with bladder cancer and developmental effects in some studies.[38-41] Some individual epidemiological studies have reported associations between the presence of disinfection byproducts in drinking water

and increased risk of birth defects, especially neural tube defects and oral clefts; however, recent articles reviewing the body of literature determined that the evidence is too limited to make conclusions about a possible association between exposure to disinfection byproducts and birth defects.[38,42-45]

Some of the most widely used agricultural pesticides in the United States, such as atrazine and glyphosate, are also drinking water contaminants.[46,47] Pesticides can enter drinking water sources as runoff from crop production in agricultural areas and enter groundwater through abandoned wells on farms.[48] Some epidemiological studies have reported associations between prenatal exposure to atrazine and reduced fetal growth.[49-52]

The use of glyphosate, an herbicide used to kill weeds, has increased dramatically in recent years because of the growing popularity of crops genetically modified to survive glyphosate treatment.[53] Previous safety assessments have concluded that glyphosate does not affect fertility or reproduction in laboratory animal studies.[54,55] However, more recent studies in laboratory animals have found that male rats exposed to high levels of glyphosate, either during prenatal or pubertal development, may suffer from reproductive problems, such as delayed puberty, decreased sperm production, and decreased testosterone production.[56,57] Very few epidemiological human studies have investigated effects of glyphosate exposure on reproductive endpoints. In contrast to the results of animal studies, one such epidemiological study of women living in regions with different levels of exposure to glyphosate found no associations between glyphosate exposure and delayed time to pregnancy.[58]

A variety of other chemical contaminants can enter the water supply after use in industry.[47] Examples include trichloroethylene and tetrachloroethylene (also known as perchloroethylene), which are solvents widely used in industry as degreasers, dry cleaning agents, paint removers, chemical extractors, and components of adhesives and lubricants.[59-61] Potential health concerns from exposure to trichloroethylene, based on limited epidemiological data and evidence from animal studies, include decreased fetal growth and birth defects, particularly cardiac birth defects.[61] A study conducted in Massachusetts reported associations between birth defects and maternal exposure to drinking water contaminated with high levels of tetrachloroethylene around the time of conception.[62] An additional study reported that older mothers or mothers who had previously miscarried, and who were exposed to high levels of tetrachloroethylene in contaminated drinking water, had a higher risk of delivering a baby with reduced birth weight.[63] However, other studies did not find associations between maternal exposure to tetrachloroethylene and pregnancy loss, gestational age, or birth weight.[64,65] Studies in laboratory animals indicate that mothers exposed to high levels of tetrachloroethylene can have spontaneous abortion, and their fetuses can suffer from altered growth and birth defects.[60]

EPA has not determined whether standards are necessary for some drinking water contaminants, such as personal care products. Personal care products, such as cosmetics, sunscreens, and fragrances; and pharmaceuticals, including prescription, over-the-the counter,

and veterinary medications, can enter water systems after use by humans or domestic animals[66] and have been measured at very low levels in drinking water sources.[67] Many concentrated animal feeding operations treat livestock with hormones and antibiotics, and can be one significant source of pharmaceuticals in water.[35] Other major sources of pharmaceuticals in water are human waste, manufacturing plants and hospitals, and other human activities such as showering and swimming.[66] Any potential health implications of long-term exposure to levels of pharmaceuticals and personal care products found in drinking water are unclear.

Manganese is a naturally occurring mineral that can enter drinking water sources from rocks and soil or from human activities.[68] While manganese is an essential nutrient at low doses, chronic exposure to high doses may be harmful, particularly to the nervous system. Many of the reports on adverse effects from manganese exposure are based on inhalation exposures in occupational settings. Fewer studies have examined health effects associated with oral exposure to manganese.[68] However, some recent epidemiological studies have reported associations between long-term exposure to high levels of manganese in drinking water during prenatal development or childhood and intellectual impairment; decreased non-verbal memory, attention, and motor skills; hyperactivity; and other behavioral effects.[69-73] Most studies on the health effects of manganese have been conducted in countries where manganese exposure is generally higher than in the United States. However, two individual studies conducted in specific areas of relatively high manganese contamination in the United States reported associations between prenatal or childhood manganese exposure and problems with general intelligence, memory, and behavior.[74,75] Although there is no health-based regulatory standard for manganese in drinking water, EPA has set a voluntary standard for manganese as a guideline to assist public water systems in managing their drinking water for aesthetic considerations, such as taste, color and odor.[7]

Perchlorate is a naturally occurring and man-made chemical that has been found in surface and groundwater in the United States.[76-78] Perchlorate is used in the manufacture of fireworks, explosives, flares, and rocket fuel.[78] Perchlorate was detected in just over 4% of public water systems in a nationally representative monitoring study conducted from 2001–2005.[78] Some infant formulas have been found to contain perchlorate, and the perchlorate content of the formula is increased if it is prepared with perchlorate-contaminated water.[79-82] Exposure to elevated levels of perchlorate can inhibit iodide uptake into the thyroid gland, possibly disrupting the function of the thyroid and potentially leading to a reduction in the production of thyroid hormone.[83,84] As noted above, thyroid hormones are particularly important for growth and development of the central nervous system in fetuses and infants.

In January 2009, EPA issued an interim health advisory level to help state and local officials manage local perchlorate contamination issues in a health-protective manner, in advance of a final EPA regulatory determination.[78,85] In February 2011, EPA decided to develop a federal drinking water standard for perchlorate, based on the concern for effects on thyroid hormones and the development and growth of fetuses, infants, and children.[78] The process for developing

the standard will include receiving input from key stakeholders as well as submitting any formal rule to a public comment process.

The two indicators that follow use the best nationally representative data currently available to characterize the performance of water systems in meeting EPA's health-based drinking water standards and in reporting monitoring results over time. Indicator E7 estimates the percentage of children served by community water systems that did not meet all applicable health-based drinking water standards. Indicator E8 estimates the percentage of children served by systems with violations of drinking water monitoring and reporting requirements. Monitoring and reporting violations occur when a water system does not monitor, does not report monitoring results, or was late in reporting results.[86] Such violations in monitoring and reporting may mean that some health-based violations were not reported; this could cause the percentages shown in Indicator E7 to be underestimated.

Indicator E7: Estimated percentage of children ages 0 to 17 years served by community water systems that did not meet all applicable health-based drinking water standards, 1993–2009

Indicator E8: Estimated percentage of children ages 0 to 17 years served by community water systems with violations of drinking water monitoring and reporting requirements, 1993–2009

About the Indicators: Indicators E7 and E8 estimate the percentage of children served by community water systems that did not meet all health-based drinking water standards or failed to adhere to monitoring and reporting requirements. The data are from an EPA database that compiles drinking water violations reported by public water systems. Indicator E7 shows the estimated percentage of children served by community water systems that did not meet health-based drinking water standards in each year from 1993 to 2009. Indicator E8 shows the estimated percentage of children served by community water systems that did not adhere to monitoring and reporting requirements in each year.

SDWIS/FED

EPA's Safe Drinking Water Information System, Federal Version (SDWIS/FED) provides information on violations of drinking water standards. Public drinking water systems in the United States are required to monitor the presence of certain individual contaminants at specific time intervals and locations to assess whether they are complying with drinking water standards. These standards include Maximum Contaminant Levels (MCLs), which are numerical limits on how much of a contaminant may be present in drinking water; as well as mandatory treatment techniques and processes, such as those intended to prevent microbial contamination of drinking water. When a violation of a drinking water standard is detected, the public water system is required to report the violation to the state, which in turn reports to the federal government. All health-based violations are compiled in SDWIS/FED. SDWIS/FED was created in 1995 and includes data from various precursor database systems that have violation and inventory data going back to 1976. SDWIS/FED also reports the number of people served by each water system.

Health-Based Drinking Water Standard Violations

Indicator E7 presents statistics on violations of drinking water standards grouped into several categories:

- The "Surface water treatment" category includes violations of requirements in the Surface Water Treatment Rule and Interim Enhanced Surface Water Treatment Rule that specify the type of treatment and maintenance activities that systems must use to prevent microbial contamination of drinking water.

- The "Chemical and radionuclide" category includes violations of the MCLs for organic and inorganic chemicals, such as atrazine, glyphosate, trichloroethylene, tetrachloroethylene, arsenic, cadmium, and mercury, in addition to radionuclide contaminants, such as radium and uranium.
- The "Lead and copper" category includes violations of treatment technique requirements for systems to control the corrosiveness of their water.[2]
- The "Total coliforms" category covers all violations of the MCL for total coliform bacteria, which is an indicator of the presence of various fecal pathogens, including *E.Coli*.[87,88]
- The "Nitrate/nitrite" category takes account of all violations of the MCLs for nitrates and nitrites.

The "Disinfectants and disinfection byproducts" category covers violations of standards for several disinfectants—chlorine, chloramine, and chlorine dioxide—and disinfectant byproducts—total trihalomethanes, haloacetic acids, chlorite, and bromate.[89]

Monitoring and Reporting Violations

Indicator E8 presents statistics on violations of monitoring and reporting requirements. Monitoring and reporting violations occur when a water system does not monitor, does not report monitoring results, or was late in reporting results.[86] All monitoring and reporting violations are compiled from SDWIS/FED.

Data Presented in the Indicators

Indicator E7 estimates the percentage of children ages 0 to 17 years served by community water systems that did not meet all applicable health-based drinking water standards between 1993 and 2009. The indicator is calculated by identifying all community water systems with violations in SDWIS/FED each year by state, then summing the number of people served by those systems with violations. Census data for the number of children in each state are then used to adjust these estimates of the total population served to estimate the percentage of children served by systems with violations in relation to all children served by community water systems.

Indicator E8 estimates the percentage of children ages 0 to 17 years served by community water systems with violations of drinking water monitoring and reporting requirements. This indicator is based on data reported to SDWIS/FED for violations between 1993 and 2009. Violations of monitoring and reporting requirements for Indicator E8 were grouped into the same categories as in Indicator E7, except for the Nitrate/nitrite category.

For the most part, the indicator represents comparisons with a consistent set of standards over the years 1993–2009, with some exceptions. Revisions to the surface water treatment standard were finalized in 2002.[89] A revised standard for radionuclides went into effect in 2003, and for arsenic (included in the chemical and radionuclide category) in 2006.[90] A new standard for

disinfection byproducts was implemented in 2002 for larger drinking water systems, and in 2004 for smaller systems.[91] The revisions to the surface water treatment standard were significant enough to warrant a break in the trend lines for this category in Indicators E7 and E8 between 2001 and 2002. The break in the "any violation" trend line between 2001 and 2002 is due to both the revision of the surface water standard and the implementation of the new disinfection byproducts standard for large systems beginning in 2002. Revisions to other standards had only minimal impacts on the indicator values. As new and revised drinking water standards take effect, water system compliance with all applicable health-based standards signifies higher levels of public health protection over time.

Violations of health-based standards (as represented in Indicator E7) may be under-reported as a result of monitoring and reporting violations. An EPA audit of drinking water data from 2002–2004 found that only 62% of health-based standards violations were reported to SDWIS.[86] Therefore, the data on systems reporting no violations of health based standards include a number of systems that have not gathered or reported all of the required data needed to make this determination.

Indicators E7 and E8 provide information about the extent to which contaminants in community water systems reach levels that may be of concern for children. However, the indicators do not provide a direct measure of children's exposure to drinking water contaminants and do not give an indication about how drinking water violations are related to health risks. A violation of a health-based standard represents a potential concern for children's health, but the importance of any violation depends on the particular contaminant, the magnitude and duration of the violation, and the extent of the violation within a system. Indicator E7 does not reflect the extent to which a standard has been exceeded or the extent to which a water system's distribution system may have been affected by a violation. The indicator does not take into account the duration of a violation within any calendar year. However, a violation that continues over an extended period of time is included in the indicator for each calendar year in which it occurs. A large water system with a single violation of short duration may significantly affect the indicator value for a single year.

The ability to examine children's potential exposure to contaminated drinking water is limited by the type of information collected and stored in the SDWIS/FED database. States are not required to report the actual contaminant levels measured to SDWIS/FED; instead, they report when standards are not met. As a result, SDWIS/FED data cannot be used to analyze national or local trends in contaminant concentrations, or to provide comparisons to the current health-based standards across all years shown.[xx] EPA is working with states to develop a new drinking

[xx] EPA requires community water systems to provide annual drinking water quality reports to their customers. These reports summarize the contaminants measured in each system's drinking water over the course of a year, providing much more detail than the information reported to SDWIS. The drinking water quality reports for many systems can be found at: http://water.epa.gov/lawsregs/rulesregs/sdwa/ccr/index.cfm.

water data system that will compile and make available actual measurements of contaminant levels.

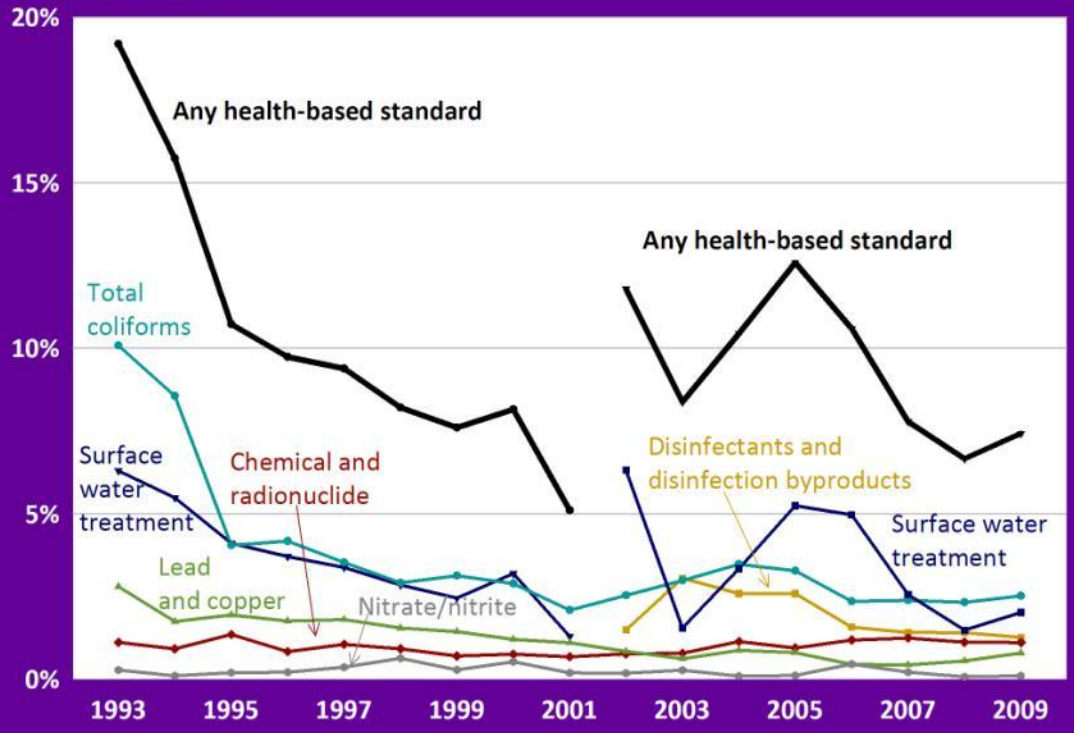
Indicators E7 and E8 are based on drinking water provided to residences served by community water systems. Community water systems are public water systems that serve water to the same residential population year-round.[92] The indicators do not account for all sources of children's drinking water. Some drinking water comes from other types of public water systems, including those that may not serve residences, or may not operate year-round (e.g., schools, factories, office buildings, and hospitals that have their own water systems; gas stations and campgrounds); and bottled water.[xxi] [93-95]

In addition, many homes are not served by community water systems and instead obtain their drinking water from individual residential wells.[93,96] EPA does not have the authority under the Safe Drinking Water Act to regulate wells that serve fewer than 25 persons or 15 service connections. Thus, the SDWIS/FED database does not contain data on non-public water systems, such as privately owned household wells, that are not required to monitor or report the quality of drinking water to EPA.[94,97] In 2000, approximately 15% of the total U.S. population was served by non-public water systems[97] and more than 90,000 new domestic wells are installed every year.[98] Separate data collection activities have found that the contaminants in untreated groundwater are generally at lower levels than the MCL; however, more than 20% of wells sampled by the U.S. Geological Survey between 1991 and 2004 contained at least one contaminant at a level of potential health concern.[99] Approximately 4% of the 2,167 sampled wells exceeded the nitrate MCL, and 7% exceeded the arsenic MCL.[99] Nitrate concentrations above the MCL were more frequently detected in agricultural regions than any other land-use setting.[99] Groundwater-sourced wells in rural and agricultural regions may be at an increased risk for nitrate and nitrite contamination due to local fertilizer use and animal waste runoff.[100]

[xxi] Bottled water is regulated by the Food and Drug Administration.

Indicator E7

Estimated percentage of children ages 0 to 17 years served by community water systems that did not meet all applicable health-based drinking water standards, 1993-2009



Data: U.S. Environmental Protection Agency, Office of Water, Safe Drinking Water Information System, Federal Version

Note: Breaks in lines for "Any health-based standard" and "Surface water treatment" reflect substantial regulatory changes implemented in 2002.

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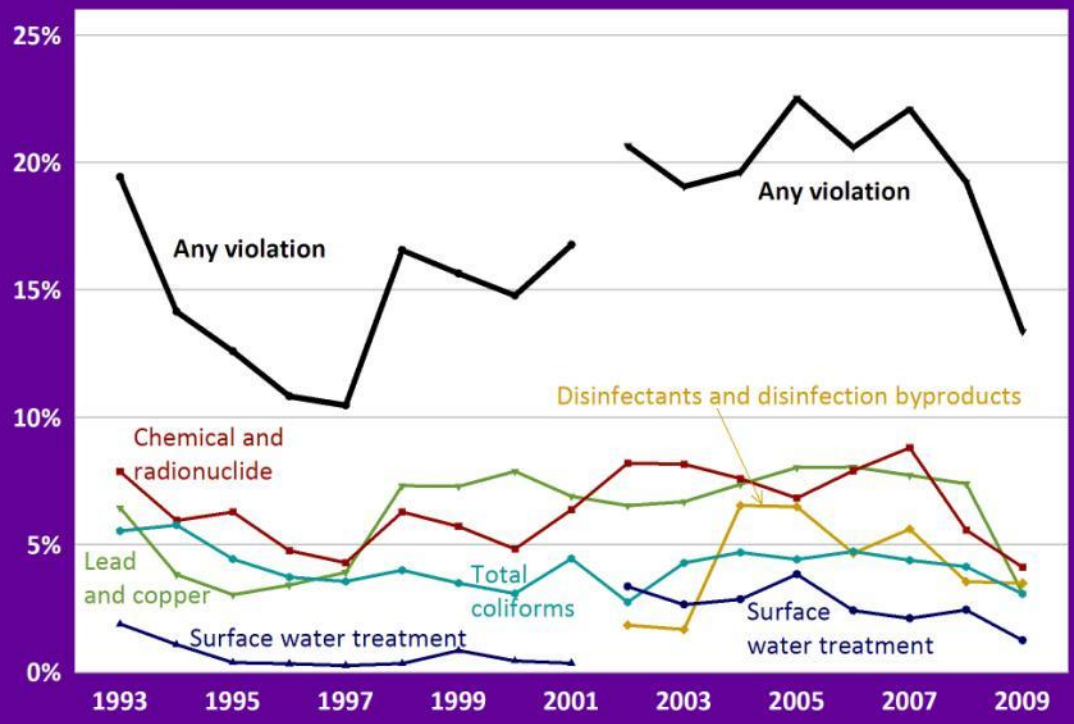
Data characterization

- Data for this indicator are obtained from EPA's database to which states are required to report public water system violations of national drinking water standards.
- All violations of health-based standards are supposed to be reported to the database; however, it is known that not all violations are reported and the magnitude of underreporting is not known.
- Some drinking water standards have been changed over time to increase the level of public health protection; therefore, as noted on the figure, some types of violations in more recent years are not strictly comparable to violations in earlier years.
- Non-public drinking water systems, such as private wells, are not represented in the database. In 2000, about 15% of the U.S. population was served by non-public water systems.

- The estimated percentage of children served by community drinking water systems that did not meet all applicable health-based standards declined from 19% in 1993 to about 5% in 2001. Since 2002, this percentage has fluctuated between 7% and 13%, and was 7% in 2009.
- The estimated percentage of children served by community drinking water systems that did not meet surface water treatment standards varied substantially from 2002–2007, following the adoption of new regulatory requirements. The percentage was more consistent from 2007–2009, and was 2% in 2009.
- Total coliforms indicate the potential presence of harmful bacteria associated with infectious illnesses. The estimated percentage of children served by community drinking water systems that did not meet the health-based standard for total coliforms was about 10% in 1993 and about 3% in 2009.
- A new standard for disinfection byproducts was adopted in 2001. The estimated percentage of children served by community water systems that had violations of the disinfection byproducts standard has declined steadily from 3% in 2003 to about 1% in 2009.

Indicator E8

Estimated percentage of children ages 0 to 17 years served by community water systems with violations of drinking water monitoring and reporting requirements, 1993-2009



Data: U.S. Environmental Protection Agency, Office of Water, Safe Drinking Water Information System, Federal Version

Note: Breaks in lines for "Any violation" and "Surface water treatment" reflect substantial regulatory changes implemented in 2002.

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Data characterization

- Data for this indicator are obtained from EPA's database to which states are required to report public water system violations of national drinking water standards.
- Not all violations of monitoring and reporting requirements are reported to the database, and the magnitude of underreporting is not known.
- Some drinking water standards have been changed over time to increase the level of public health protection; therefore, as noted on the figure, some types of violations in more recent years are not strictly comparable to violations in earlier years.
- Non-public drinking water systems, such as private wells, are not represented in the database. In 2000, about 15% of the U.S. population was served by non-public water systems.

- Between 1993 and 2009, the estimated percentage of children served by community water systems that had at least one monitoring and reporting violation fluctuated between about 11% and 23%, and was 13% in 2009.
- In 1993, approximately 6% of children served by community water systems lived in an area with significant monitoring and reporting violations for lead and copper. This figure dropped to about 3% in 2009.
- The estimated percentage of children served by community water systems with a chemical and radionuclide monitoring violation has varied between 4 and 9%, and was 4% in 2009.

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Chemicals in Food

Children's diets are an important pathway for exposure to some environmental chemicals and other contaminants. Children may be at a greater risk for exposures to contaminants because they consume more food relative to their body weight than do adults. Additionally, children's dietary patterns are often less varied than those of adults, suggesting that there are greater opportunities for continuous exposure to a foodborne contaminant than in adults.[1]

Food contamination can come from multiple sources, including antibiotics and hormones in meat and dairy products, as well as microbial contamination that can lead to illness. An estimated 48 million Americans suffer from foodborne illnesses each year,[2] and children under age five have the highest incidence of most of these infections.[3] Microbial contamination of food is monitored and regulated by a number of federal agencies, including the Department of Agriculture and the Food and Drug Administration.[xxii] In addition, a wide variety of chemicals from man-made sources may be found in or on foods, typically at low levels. Chemicals in foods may come from application of pesticides to crops, from transport of industrial chemicals in the environment, or from chemicals used in food packaging products. A number of persistent environmental contaminants tend to accumulate in all types of animals, and are frequently found in meat, poultry, fish, and dairy products. Other chemicals, such as perchlorate and a variety of pesticides, are often found in fruits, vegetables, and other agricultural commodities. Some chemicals in food, such as mercury and perchlorate, have naturally occurring as well as man-made sources. The health risks from chemicals in food are dependent on both the actual level of a chemical in the food as well as the amount of the food consumed by individuals.

Following this text, an indicator is presented for organophosphate pesticides in selected foods. Many chemicals of concern in food lack sufficient data to generate reliable, nationally representative indicators, particularly for children. Selected chemicals of concern for children's health that are frequently found in foods are summarized below. Further details can be found in the Biomonitoring section of this report for several of these chemicals, including methylmercury, polychlorinated biphenyls (PCBs), polybrominated diphenyl ethers (PBDEs), phthalates, perfluorochemicals (PFCs), and perchlorate.

Methylmercury

Mercury is a naturally occurring element that is released to the environment from a variety of sources, including the combustion of coal, the use of mercury in industrial processes, and from breakage of products such as mercury thermometers and fluorescent lighting, as well as from

[xxii] More information on microbial contaminants in food is available at http://www.fda.gov/Food/ResourcesForYou/Consumers/ucm103263.htm, http://fsrio.nal.usda.gov/pathogen-detection-and-monitoring, and http://www.fsis.usda.gov/fact_sheets/Foodborne_Illness_&_Disease_Fact_Sheets/index.asp.

natural sources such as volcanoes. Mercury may enter water bodies through direct release or through emissions to the atmosphere that are subsequently deposited to surface waters. Bacteria in water bodies convert the deposited mercury into methylmercury.[4] Methylmercury can be absorbed by small aquatic organisms that then are consumed by predators, including fish.[5] As each organism builds up methylmercury in its own tissues, and as smaller fish are eaten by larger fish, concentrations of methylmercury can accumulate, particularly in large fish with longer lifespans[6-8] such as sharks and swordfish.[9]

EPA has determined that methylmercury is known to have neurotoxic and developmental effects in humans.[10] This conclusion is based on severe adverse effects observed in exposed populations in two high-dose mercury poisoning events in Japan and Iraq. Some other studies of populations with prenatal exposure to methylmercury through regular consumption of fish have reported more subtle adverse effects on childhood neurological development.[11-15] Although ingestion of methylmercury in fish may be harmful, other compounds naturally present in many fish (such as high quality protein and other essential nutrients) are extremely beneficial.

In particular, fish are an excellent source of omega-3 fatty acids, which are nutrients that contribute to the healthy development of infants and children.[16] Pregnant women are advised to seek dietary sources of these fatty acids, including many species of fish. However, the levels of both methylmercury and omega-3 fatty acids can vary considerably by fish species. Thus, the type of fish, as well as portion sizes and frequency of consumption are all important considerations for health benefits of fish and the extent of methylmercury exposure.[16] For this reason, EPA and the U.S. Food and Drug Administration (FDA) provide advisory information on fish consumption to females who are pregnant, breastfeeding, or of childbearing age, and to young children. The advisory encourages consumption of up to 12 ounces per week of a variety of fish and shellfish that are lower in mercury, or, in the absence of a local advisory, consumption of up to 6 ounces per week of fish caught from local waters and no other fish that week. EPA and FDA also recommend that these categories of women and young children avoid consuming shark, swordfish, tile fish, or king mackerel, because these species may contain high levels of methylmercury.[17] Fish that are high in omega-3 fatty acids and low in mercury are expected to offer the greatest health benefits.[9,16,18] EPA and FDA are currently working to update the fish consumption advisory to incorporate the most current science regarding the health benefits of fish consumption and the risks from methylmercury in fish. In 2011, the Departments of Agriculture and Health and Human Services jointly released the 2010 Dietary Guidelines for Americans, which recommended that pregnant or breastfeeding women should consume 8–12 ounces of seafood per week, but avoid consumption of the same high-mercury-containing fish identified in EPA and FDA's advisory.[19] More information regarding current fish advisories, and links to lists of fish and shellfish typically containing lower levels of mercury, can be found at http://water.epa.gov/scitech/swguidance/fishshellfish/fishadvisories/general.cfm#tabs-4. Tribal and state-specific fish advisories can be found at http://fishadvisoryonline.epa.gov/General.aspx.

Polychlorinated biphenyls

Polychlorinated biphenyls (PCBs) are a group of persistent chemicals used in electrical transformers and capacitors for insulating purposes, in gas pipeline systems as a lubricant, and in caulks and other building materials. The manufacture, sale, and use of PCBs were generally banned by law in 1979, although EPA regulations have authorized their continued use in certain existing electrical equipment. Due to their persistent nature, large reservoirs of previously released PCBs remain in the environment. PCBs accumulate in fat tissue, so they are commonly found in foods derived from animals. Consumption of fish is a common source of PCB exposure, but other foods with lower PCB levels that are consumed more frequently, including meat, dairy, and poultry products, also contribute to PCB exposure.[20,21] A study by the U.S. Department of Agriculture found that levels of certain PCBs in beef and chicken declined between 2002 and 2008, while levels in turkey and pork remained relatively constant during the same years.[22] Exposure to PCBs remains widespread;[23,24] however, declining environmental levels of PCBs suggest that children today are exposed to lower levels of PCBs compared with children in previous generations.[20,25-28]

Prenatal exposure to PCBs has been associated with adverse effects on children's neurological development and impaired immune response, primarily through studies of populations that consume fish regularly.[29-31] Although there is some inconsistency in the epidemiological literature, several reviews of the literature have found that the overall evidence supports a concern for effects of PCBs on children's neurological development.[29,30,32-34] The Agency for Toxic Substances and Disease Registry has determined that "Substantial data suggest that PCBs play a role in neurobehavioral alterations observed in newborns and young children of women with PCB burdens near background levels."[20] Some studies have also detected associations between childhood exposure and adverse health effects.[30,35-37] In addition to PCBs, many other organochlorine chemicals, including dioxins, dibenzofurans, and organochlorine pesticides, are persistent and bioaccumulative and are frequently found in foods derived from animals.[38]

Polybrominated diphenyl ethers

Polybrominated diphenyl ethers (PBDEs) are a class of flame retardants used in many applications, including furniture foam, small appliances, and electronic products. PBDEs are intended to slow the ignition and rate of fire growth. Of three forms of PBDEs once used in the United States (pentaBDE, octaBDE, and decaBDE), only the decaBDE form, used primarily in televisions, personal computers, and other electrical appliances, is still in production. Manufacturers of decaBDE have agreed to phase out all uses of the chemical by the end of 2013.[39] However, products manufactured prior to the elimination of the pentaBDE and octaBDE forms in 2004, and products manufactured prior to the phaseout of decaBDE in 2013, can remain in use and contribute to the presence of PBDEs in the environment.

Like PCBs, PBDEs are persistent in the environment, accumulate in fat tissue, and have been found in a variety of foods, including fish, meat, poultry, and dairy products as well as breast milk.[40-48] Exposure studies have concluded that the presence of PBDEs in house dust and in

foods are both important contributors to PBDE exposures for people of all ages, and that exposures from house dust are generally greater than those from food.[46,47,49-54] PBDE toxicity to the developing nervous system as well as endocrine disruption have been identified as areas of potential concern.[40,55-59]

Bisphenol A

Bisphenol A (BPA) is an industrial chemical used in the production of epoxy resins used as inner liners of metallic food and drink containers to prevent corrosion. BPA is also used in the production of polycarbonate plastics that may be used in food and drink containers. The primary route of human exposure to BPA is through diet, when BPA migrates from food and drink containers, particularly when a container is heated.[60-62]

Much of the scientific interest in BPA is related to published research suggesting that BPA may be an endocrine disrupting chemical.[63,64] Endocrine disruptors act by interfering with the biosynthesis, secretion, action, or metabolism of naturally occurring hormones.[63-65] BPA has demonstrated developmental effects in laboratory animals at high doses, though the effects of lower doses similar to typical human exposure levels are the subject of scientific debate.[61,66-70] Based on a critical review of the existing scientific literature, in 2008 the National Toxicology Program (NTP) determined that there was "some concern" (the midpoint on a five-level scale ranging from "negligible" to "serious")[xxiii] for effects of BPA on the brain, behavior, and prostate gland in fetuses, infants, and children.[61] Although there is uncertainty regarding the effects in humans of BPA at typical exposure levels, several retailers and manufacturers have begun phasing out baby products such as bottles and sippy cups that contain BPA. Several states have also introduced legislation to ban or limit BPA in food containers and consumer products. Additional studies by both government and non-government entities are being conducted to provide additional information and address uncertainties about the safety of BPA.

Phthalates

Phthalates are a class of chemicals commonly used to increase the flexibility of plastics in a wide array of consumer products, and have been used in food packaging.[71-74] Some phthalates have been found at higher levels in fatty foods such as dairy products, fish, seafood, and oils, which are most likely to absorb phthalates.[74] Phthalates in a mother's body can enter her breast milk. Ingestion of that breast milk and infant formula containing phthalates may also contribute to infant phthalate exposure.[75] Certain phthalates are suspected endocrine disruptors, and have shown a number of reproductive and developmental effects in laboratory animal studies[76-85] as well as some reported associations in human epidemiological studies.[86-89]

[xxiii] More information on NTP concern levels is available at http://www.niehs.nih.gov/news/media/questions/sya-bpa.cfm.

Perfluorochemicals

Perfluorochemicals (PFCs) are a group of chemicals used in a variety of consumer products, including food packaging, and in the production on nonstick coatings on cookware.[90,91] Long-chain PFCs, including perfluorooctane sulfonic acid (PFOS) and perfluorooctanoic acid (PFOA), have already been or will be phased out by the chemical industry by 2015, although the persistence of these chemicals means that they will remain in the environment for several years despite reductions in emissions. While the routes of human exposure to PFCs are not fully understood, two recent studies have identified food consumption as the primary exposure pathway.[92,93] PFC-treated food-contact packaging, such as microwave popcorn bags, may be a source of PFC exposure.[94,95] Heating these materials may cause PFCs to migrate into food, or into the air where they may be inhaled.[xxiv] Meats may also be contaminated with PFCs due to exposure of source animals to air, water, and feed contaminated with PFCs.[95-97] PFCs have also been detected in some plant-based foods.[93] Studies in laboratory animals have demonstrated reproductive and developmental toxicity of PFCs.[98,99] Some human health studies have reported associations between prenatal exposure to PFCs and a number of adverse birth outcomes,[100-103] while other studies have not.[104,105]

Perchlorate

Perchlorate is a naturally occurring and man-made chemical that has been detected in surface water and groundwater in the United States.[106-109] Perchlorate is used in the manufacture of fireworks, explosives, flares, and rocket propellant.[107,109] Perchlorate has been detected in human breast milk, dairy products, as well as in leafy vegetables and other produce.[108,110-115] Infant formulas have been found to contain perchlorate, and the perchlorate content of the formula is increased if it is prepared with perchlorate-contaminated water.[116-118]

Exposure to high doses of perchlorate has been shown to inhibit iodide uptake into the thyroid gland, thus possibly disrupting the function of the thyroid and potentially leading to a reduction in the production of thyroid hormone.[107,119,120] Thyroid hormones are particularly important for growth and development of the central nervous system in fetuses and infants.[121] Due to the sensitivities of the developing fetus, perchlorate exposures among pregnant women, especially those with preexisting thyroid disorders or iodide deficiency, carry the potential for risk of adverse health effects.

[xxiv] The U.S. Food and Drug Administration recently worked with several manufacturers to remove grease-proofing agents containing C8 perfluorinated compounds from the marketplace. These manufacturers volunteered to stop distributing products containing these compounds in interstate commerce for food-contact purposes as of October 1, 2011. For more information, see http://www.fda.gov/Food/FoodIngredientsPackaging/FoodContactSubstancesFCS/ucm308462.htm.

Organophosphate Pesticides

Agricultural crops are frequently treated with pesticides to control insects and other pests that may affect crop growth. Some of the most prevalent classes of pesticides used in growing food crops are the carbamates, pyrethroids, and the organophosphates. After crops are harvested, they may retain residues of these pesticides. Apples, corn, oranges, rice, and wheat are among the agricultural commodities consumed in large amounts by children.

Organophosphates are one class of pesticides that are of concern for children's health. Examples of organophosphate pesticides include chlorpyrifos, azinphos methyl, methyl parathion, and phosmet. These pesticides are frequently applied to many of the foods important in children's diets, and certain organophosphate pesticide residues can be detected in small quantities on these foods. Organophosphates can interfere with the proper function of the nervous system when exposure is sufficiently high.[1,122] Childhood is a period of increased vulnerability, because many children may have low capacity to detoxify organophosphate pesticides through age 7 years.[123] Recent studies have reported an association between prenatal organophosphate exposure and childhood attention deficit/hyperactivity disorder (ADHD) in U.S. communities with relatively high exposures to organophosphate pesticides,[124] as well as with exposures found within the general US population.[125] Other recent studies have reported associations between prenatal organophosphate pesticide exposures and a variety of neurodevelopmental deficits in childhood, including reduced IQ, perceptual reasoning, and memory.[126-128] Since 1999, EPA has imposed restrictions on the use of the organophosphate pesticides azinphos methyl, chlorpyrifos, and methyl parathion on certain food crops and around the home, due largely to concerns about potential exposures of children.[129-131]

The 1996 Food Quality Protection Act required EPA to identify and assess the extent of dietary pesticide exposure in the United States, and to determine whether there was a "reasonable certainty of no harm" to vulnerable populations including infants and children.[132] The U.S. Department of Agriculture's Pesticide Data Program (PDP) provides data annually on pesticide residues in food, with a specific focus on foods often consumed by children.[133] Other researchers have supplemented the PDP with their own analyses. A recent study measured pesticide residues in 24-hour duplicate food samples of fruits, vegetables, and juices served to children, and found that 14% of the samples contained at least one organophosphate pesticide.[134] Additional pesticide residue data are available from FDA's pesticide residue monitoring program.[135] A number of pesticide residues, along with a variety of other chemicals in food, are also measured in FDA's Total Diet Study.[136] When pesticide residue data are combined with dietary consumption surveys, it can be possible to estimate pesticide exposure from dietary intake.

Indicator E9 presents the percentage of samples of two fruits and two vegetables analyzed by the USDA PDP that have detectable residues of organophosphate pesticides. This indicator allows for a general comparison of the frequency of organophosphate detection over time for four foods typically consumed by children, although data are not available on each fruit or vegetable for every year.

Indicator E9: Percentage of sampled apples, carrots, grapes, and tomatoes with detectable residues of organophosphate pesticides, 1998–2009

About the Indicator: Indicator E9 presents the percentage of sampled apples, carrots, grapes, and tomatoes that were found to contain detectable residues of organophosphate pesticides from 1998–2009. These foods were selected because they are frequent components of children's diets, and because data for these foods were available for multiple years. The data are from an analysis of pesticide residues in foods conducted annually by the U.S. Department of Agriculture.

Pesticide Data Program

The U.S. Department of Agriculture (USDA) collects data on pesticide residues in food annually. USDA's Pesticide Data Program (PDP), initiated in 1991, focuses on measuring pesticide residues in foods that are important parts of children's diets, including apples, apple juice, bananas, carrots, grapes, green beans, orange juice, peaches, pears, potatoes, and tomatoes.

Samples are collected from food distribution centers in 10 states across the country.[137] The PDP has a statistical design in which food samples are randomly selected from the national food distribution system and reflect what is typically available to the consumer, including both domestic and imported foods.[137] Different foods are sampled each year. In its history, the PDP has tested for more than 440 different pesticides.[133] In 2009, the PDP analyzed fruit and vegetables for 309 pesticides and related chemicals. Prior to analysis, the PDP processes samples by following the preparations an average individual would use before consuming an item. This includes washing fruits and vegetables, as well as removing inedible portions of a food item. For example, tomatoes and grapes are washed with the stems and other materials removed, while apples are washed and the stems and cores are removed.

Data Presented in the Indicator

Indicator E9 displays the percentage of apple, grape, carrot, and tomato samples with detectable organophosphate pesticide residues reported by the PDP from 1998–2009. These four foods were selected as those that were sampled by the PDP in at least five years from 1998–2009 and are among the 20 most-consumed foods identified in an analysis by EPA.[138] Other foods not shown here may have either greater or lesser frequencies of organophosphate pesticide residue detection than the four foods presented in this indicator.

The 43 organophosphates that were sampled in every one of the years 1998–2009 are included in calculation of the indicator; 53 other organophosphates that were added to or dropped from the program in these years are excluded so that the chart represents a consistent set of pesticides for all years shown. Some aspects of trends in organophosphate residues could be missed by the indicator if any organophosphates other than the 43 considered in the indicator had substantial changes in use on the four selected foods during the years 1998–2009. For

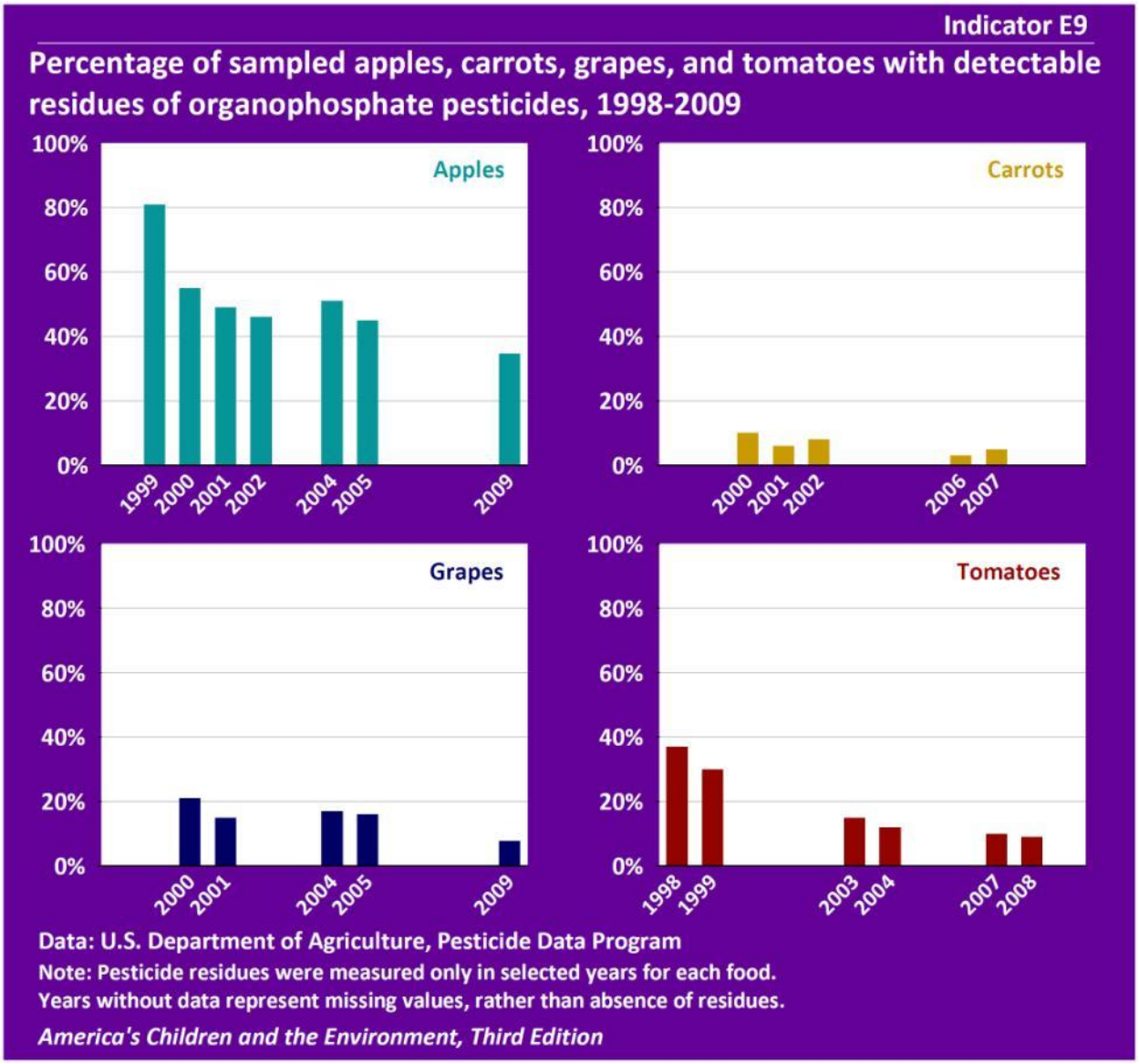
example, a decrease in the percentage of detections of organophosphate residues may reflect an actual decrease in the use of organophosphate pesticides, but can definitively represent only a decrease in the residues of the 43 OPs included in the indicator; it does not account for potential substitution with other organophosphates or other types of pesticides.

The indicator also defines "detectable" based on the ability to measure residues in the PDP in 1998, so that introduction of more sensitive measurement techniques over time does not affect the indicator and allows for direct comparison of data collected in previous years with those collected today. This means that some produce samples analyzed in recent years with improved detection technology would, for purposes of indicator calculation, be considered to have non-detectable organophosphate residues based on comparison with the older, higher limit of detection.[xxv]

The fruits and vegetables shown in this indicator were each sampled in five to seven years between 1998 and 2009. Gaps in the percentage of residue detections from year to year thus represent missing information, rather than an absence of organophosphate residues.

This indicator is a surrogate for children's exposure to pesticides in foods: If the frequency of detectable levels of pesticides in foods decreases, it is likely that exposures will decrease. However, the indicator does not account for many additional factors that affect the risk to children. For example, some organophosphates pose greater risks to children than others do, and residues on some foods may pose greater risks than residues on other foods due to differences in amounts consumed. The indicator also does not distinguish between residue levels that are barely detectable and those that are much higher, which would pose a greater concern for children's health. Finally, exposures to organophosphate pesticides may also occur by pathways other than the diet, such as ingestion of pesticides present in house dust and drinking water.

[xxv] An alternate analysis of the data that considered all detectable residues, without holding the limit of detection constant at 1998 levels, resulted in percentages of food samples with detectable organophosphate pesticide residues very similar to those shown in the indicator.



Data characterization

- Data for this indicator are obtained from a U.S. Department of Agriculture program that measures pesticide residues in food samples collected from 10 states.
- Food samples are randomly selected from the national food distribution system and reflect what is typically available to the consumer.
- The types of foods sampled change over time; so, for example, data for pesticide residues on apples are not available every year.
- The indicator is calculated using the measurement sensitivity as of 1998 for each year shown; more sensitive measurement techniques have been incorporated over time.

- In 1999, 81% of sampled apples had detectable organophosphate pesticide residues. In 2009, 35% had detectable residues.

- In 2000, 10% of sampled carrots had detectable organophosphate pesticide residues. In 2007, 5% had detectable residues.
- In 2000, 21% of sampled grapes had detectable organophosphate pesticide residues. In 2009, 8% had detectable residues.
- In 1998, 37% of sampled tomatoes had detectable organophosphate pesticide residues. In 2008, 9% had detectable residues.

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Contaminated Lands

Accidents, spills, leaks, and improper disposal and handling of hazardous materials and wastes have resulted in tens of thousands of contaminated sites across the United States. The nature of the contaminants and the hazards they present vary greatly from site to site. These contaminants include industrial solvents, petroleum products, metals, residuals from manufacturing processes, pesticides, and radiological materials, as well as certain naturally occurring substances such as asbestos. Contaminated lands can threaten human health and the environment, in addition to hampering economic growth and the vitality of local communities.

The presence of contaminated soils in a particular location may or may not have health consequences. Soils, unlike air and water, are not intentionally inhaled, absorbed, or ingested. Contaminants diffuse more slowly through soil than through air or water, so contaminants are rarely distributed uniformly across a contaminated site. Soils are a concern if children are playing, attending school, or residing on or near to contaminated land. People and pets may track contaminated soils and dusts into homes where infants and toddlers are playing. Some contaminants may harm or penetrate the skin, and by touching or playing in soil children may come into direct contact with them. Children may ingest soils through hand-to-mouth play or by eating without first washing their hands after having touched contaminated soil. Soil dust may be carried on the wind and inhaled into the lungs, where it can be very damaging. The optimal approach to minimizing risks to children from contaminated soils is to prevent these exposures.

In addition, contaminated land may contribute to pollution of ground water, surface water, ambient air, and foods, creating additional potential human exposure routes. For example, consumption of fish caught at or near a contaminated site may increase risk of exposure to contaminants from the site. The same is true of drinking water from contaminated ground- or surface water sources. When drinking water sources are affected at EPA-tracked contaminated sites, an alternate water supply may need to be provided, in some cases permanently.

Cleanup of contaminated lands may be conducted by EPA, other federal agencies, states, tribes, municipalities, or the party responsible for the contamination. As of September 2011, EPA's programs for assessing and cleaning up contaminated lands track roughly 22 million acres of land across the United States, or nearly 1% of the entire U.S. land mass.[1] EPA and its partners conduct work on contaminated lands through federally mandated programs such as the Superfund and Corrective Action programs. The Superfund program, implemented under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), aims to clean up some of the most hazardous and highly polluted inactive commercial, industrial, and residential properties in the country. The Corrective Action program, implemented under the Resource Conservation and Recovery Act (RCRA), aims to control and clean up releases at operating hazardous waste treatment, storage, and disposal facilities. EPA is also responsible for other programs that focus on management of contaminated lands, including Brownfields, underground storage tanks, and RCRA waste management and minimization programs.

EPA prioritizes sites for cleanup using information from initial investigations regarding possible threats to human health or the environment. EPA's primary concern is to protect people from the most contaminated lands and to clean up these sites to a standard that is protective, and that state, local, or tribal governments and communities deem appropriate based on the future uses of the individual site. EPA and partner agencies work to contain possible routes for exposure as soon as possible.[2,3]

When a potential pathway for exposure is identified, a process is normally initiated for the pathway to be minimized or eliminated. For Superfund sites and for hazardous waste facilities requiring Corrective Action, EPA or authorized state regulators assess contaminated media, exposure pathways, risks from complete pathways, and the significance of any risks. If no significant human health risks are identified, a determination is made that the site has all human health protective measures in place. If significant human health risks are or may be present, regulators choose site-specific controls (e.g., fencing, caps, containment walls) and cleanup activities (e.g., excavation, groundwater treatment) necessary to reduce the risks.

If additional contamination or previously unrecognized pathways of exposure are identified, a site that is designated as having all human health protective measures in place may lose that designation until pathways of exposure are controlled.

When a site is designated as having all human health protective measures in place, known pathways of exposure have been controlled, although additional cleanup work may remain. These sites pose a reduced risk to children compared with most sites that have not yet been designated as having all human health protective measures in place. However, there can be a number of reasons why a site has not yet achieved that designation. For example, some sites have not yet been adequately assessed, and it is thus unknown whether these sites pose significant risk to human health.

This approach to managing potential exposures is based on identified presence of contaminants and potential exposure pathways because there is often an absence of information identifying actual children's exposures; however, there are notable exceptions where EPA and other federal and state agencies have addressed documented exposures.[4-10]

Children who have been exposed to contaminants do not all experience the same health outcomes. The magnitude and duration of an exposure, the pathway of exposure (ingestion, inhalation, dermal), the stage of development at which a child is exposed, and differences in genetic susceptibility all influence the variation in outcome from exposure. Even after exposure characteristics and genetic factors have been taken into consideration, variation remains in risks experienced by different individuals and different communities as a consequence of exposures to contaminants. This variation may in part be explained through socio–cultural and socioeconomic factors that have been associated with physical and psychological health, including family income, unemployment, nutrition, education, housing and infrastructure, race, gender, class, access to health services, social cohesion, participation in local decision-making, exercise, and health-related behaviors (e.g., smoking, drug abuse).[11-22]

Of the many sociological determinants of health, the relationships between race/ethnicity and health status and between lower levels of income and less optimal health are among the most documented.[23-26] Because these factors are related to many of the other sociological determinants, they are frequently used as proxies for a larger set of factors. For these reasons, the following indicators of children living in proximity to contaminated lands focus on differences by race/ethnicity and family income level.

Indicator E10: Percentage of children ages 0-17 years living within one mile of Superfund and Corrective Action sites that may not have all human health protective measures in place, 2009

Indicator E11: Distribution by race/ethnicity and family income of children living near selected contaminated lands in 2009, compared with the distribution by race/ethnicity and income of children in the general U.S. population

About the Indicators: Indicators E10 and E11 present information about children living within one mile of Superfund sites or RCRA Corrective Action sites that may not have had all human health protective measures in place as of October 1, 2009. Site boundaries were estimated and a computer mapping tool was used to identify all land areas within one mile of each of these sites. Data from the 2000 U.S. Census were then used to estimate the population of children living within these areas. Indicator E10 provides information about U.S. children living within one mile of these selected sites, including the percentage of children in proximity by race, ethnicity, and family income. Indicator E11 compares the race/ethnicity profile of children living within one mile of these selected sites with the profile for all children living in the United States.

Corrective Action and Superfund Sites

EPA's Office of Solid Waste and Emergency Response manages the RCRA Corrective Action Program and the Superfund Program, and maintains inventories of sites in each program. The Comprehensive Environmental Response, Compensation, and Liability Information System (CERCLIS) database provides information on Superfund sites, and the Resource Conservation and Recovery Act Information (RCRAInfo) database provides information on RCRA Corrective Action sites. As of October 1, 2009 there were 1,653 Corrective Action and Superfund sites, totaling more than 10 million acres, that may not have had all human health protective measures in place.[3] Of the 3,746 Corrective Action sites at that time, 1,297 fell into this category. Of the 1,727 Superfund sites (which includes both sites that are on the National Priorities List and sites that are not on the NPL but for which the Superfund program has some responsibilities), a total of 356 fell into this category. The location and extent of each site are characterized by the latitude and longitude of a single point within that site, and the area (total acres) of the site, obtained from the official documentation for each site.[xxvi] A map displaying the distribution of these sites across the country and their prevalence in urbanized areas is available in the Methods document for this topic (available at www.epa.gov/ace).

[xxvi] Actual boundaries of the sites are available in digital form for only a few sites.

Some of the largest sites that EPA oversees are federal facilities. Among the sites that may not have had all human health protective measures in place in 2009, 47 Corrective Action sites and 62 Superfund sites are federal facilities.

Estimating Site Areas and Children's Proximity

For purposes of indicator calculation, the actual land area within each site was approximated using the latitude/longitude and acreage information. A circle whose area equaled the site's acreage was drawn around each site's latitude/longitude identification point. It is important to note that these areas are not the actual site boundaries, and are not expected to reflect the actual area of contamination. Contamination will likely be determined by factors such as the release of waste, the contours of the land, and groundwater flow. Sites also have hotspots (areas with high levels of contamination) and areas that have been remediated or were never contaminated. The site boundaries are therefore likely to overestimate the area of a site that is contaminated. Nonetheless, approximating the area of a site with a circle is a reasonable assumption that provides the best available information for this analysis.

To identify land areas in proximity to the selected contaminated lands, a one-mile buffer was drawn around the circle representing each site. Data on total child population, and population by race and ethnicity, were collected from the 2000 Census for children living in Census blocks whose center point was within the one-mile buffer boundary. Information on family income levels (percentage above and below poverty level, by race and ethnicity) was extrapolated for these blocks from Census block group data. Data from the 2000 census were used in order to obtain necessary population race/ethnicity and income statistics at the local level; this information is not available in the 2009 census estimates.[xxvii]

Data Presented in the Indicators

Each indicator presents a characterization of the population of children living within one mile of Superfund or RCRA Corrective Action sites that may not have had all human health protective measures in place as of October 1, 2009. Indicator E10 shows the percentage of children living within one mile of a site, by race/ethnicity and family income. Indicator E11 shows the proportion of children of each race and ethnicity among those living in proximity to the selected sites, compared with the race/ethnicity proportions among all children in the United States. This comparison is also made for children living in homes with incomes below poverty level. Tables of values for these indicators at the state level are available in the Appendix to this document.

[xxvii] A greater percentage of children were living in poverty in 2009 than in 2000; therefore, these calculations will understate the proportion of children below poverty living in proximity to the selected contaminated lands in 2009.

Data for seven race/ethnicity groups are presented in the indicators: White, Black, Asian, American Indian or Alaska Native (AIAN), Native Hawaiian or Other Pacific Islander (NHOPI), All Other Races, and Hispanic. The "All Other Races" category includes all other races not specified, together with those individuals who report more than one race. Children of Hispanic ethnicity may be of any race. Data presented by race do not include any designation of ethnicity; for example, the indicator value labeled "Black" includes both Hispanic and non-Hispanic Black children, and children who are Black and Hispanic are included in the indicator values for both "Black" and "Hispanic" children. Three family income categories are presented in the indicators: all incomes, below the poverty level, and greater than or equal to the poverty level.

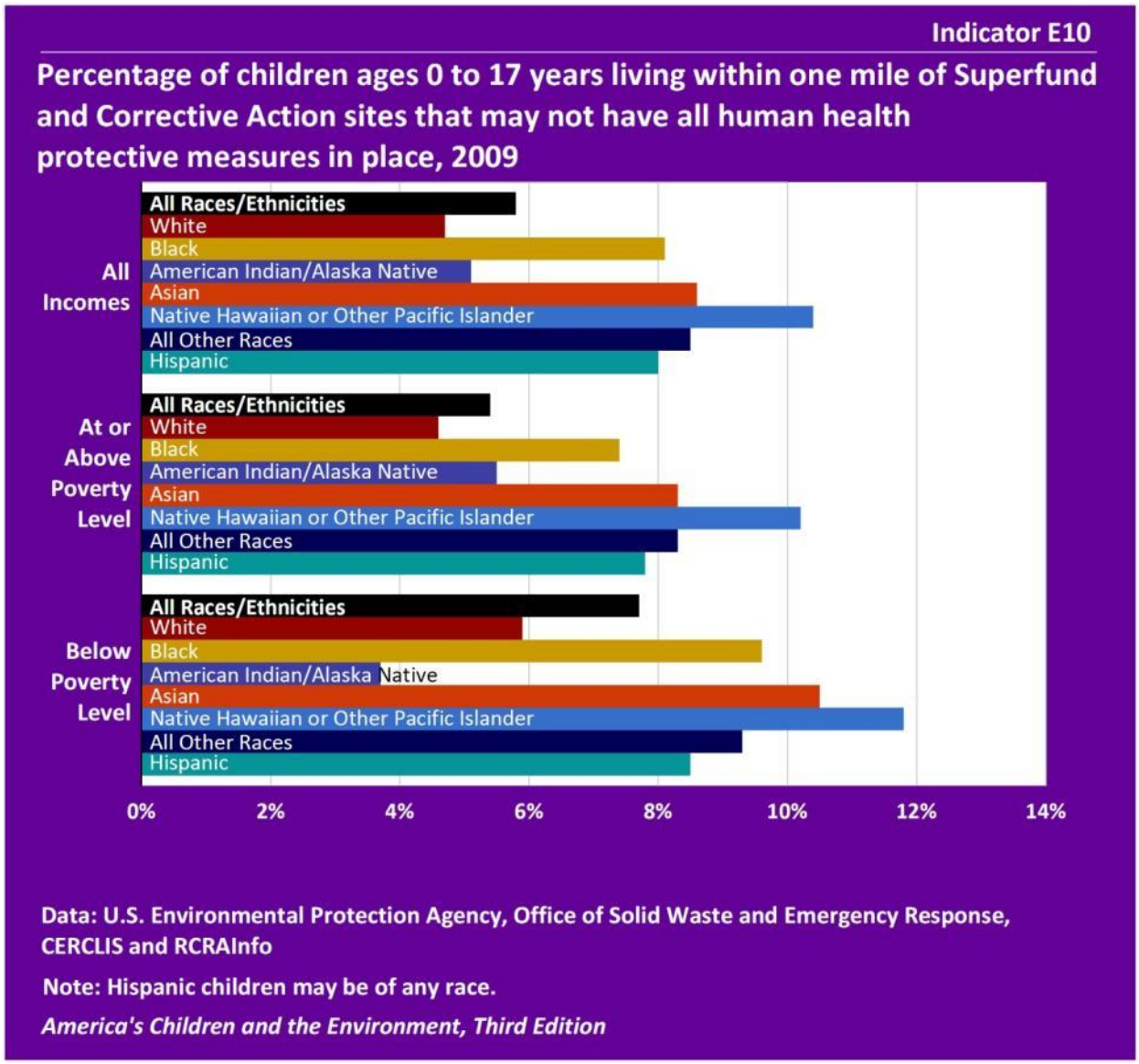
Designation of sites that may not have all human health protective measures in place were made for the first time in 2009; trend data are not reported because these designations were not analyzed for purposes of this report in earlier years.[xxviii]

For purposes of these indicators, proximity to a site is used as a surrogate for potential exposure to contaminants found at these sites. The indicators do not imply any specific relationship between childhood illness and a child's proximity to a Superfund or Corrective Action site. Information on amounts of environmental contamination, which would be a source of exposure to children, is generally available for these sites, but information on the extent to which children are actually exposed is not generally available. Because of the ways in which children can be exposed to land contaminants and the potential for certain contaminants to move into groundwater or to vaporize through soil, the proximity to contaminated sites may increase the potential for exposure and the possible health consequences, but proximity to a site does not mean that there will always be exposure. Nor does proximity to a site represent risks of adverse health effects. The risk of exposure posed to children varies significantly across all the different types of contaminated sites and the different activities of children on or near the sites. Many sites do not pose risks outside of property boundaries.

These indicators present a high-end approximation of children at risk from the Corrective Action and Superfund sites that may not have all human health protective measures in place, but do not include children near the much larger universe of Brownfield sites, leaking underground storage tanks, and sites addressed solely by state, tribal, and local authorities or private companies. While the indicators include those RCRA Corrective Action sites assumed to have the most potential for contamination, these sites represent only a subset of waste treatment, storage, or disposal facilities currently regulated by EPA. The indicators also do not capture the proportion of children living near contaminated sites that are yet to be identified. Access to uncontrolled contamination remains the greatest risk of potential exposure, and risks are most likely to have been greatest prior to intervention by EPA and partner agencies. The

[xxviii] These data cannot be compared to Indicator E9 from previous editions of *America's Children and the Environment*. Previous versions considered only Superfund sites; represented each site as a single point, rather than an area; and did not consider the status of human health protective measures put in place at the sites.

ultimate cleanup of these sites best assures reduced health risks for children by eliminating the possibility of exposure and promotes the health of their communities since cleanup opens the way for sustainable redevelopment and revitalization opportunities.



Data characterization

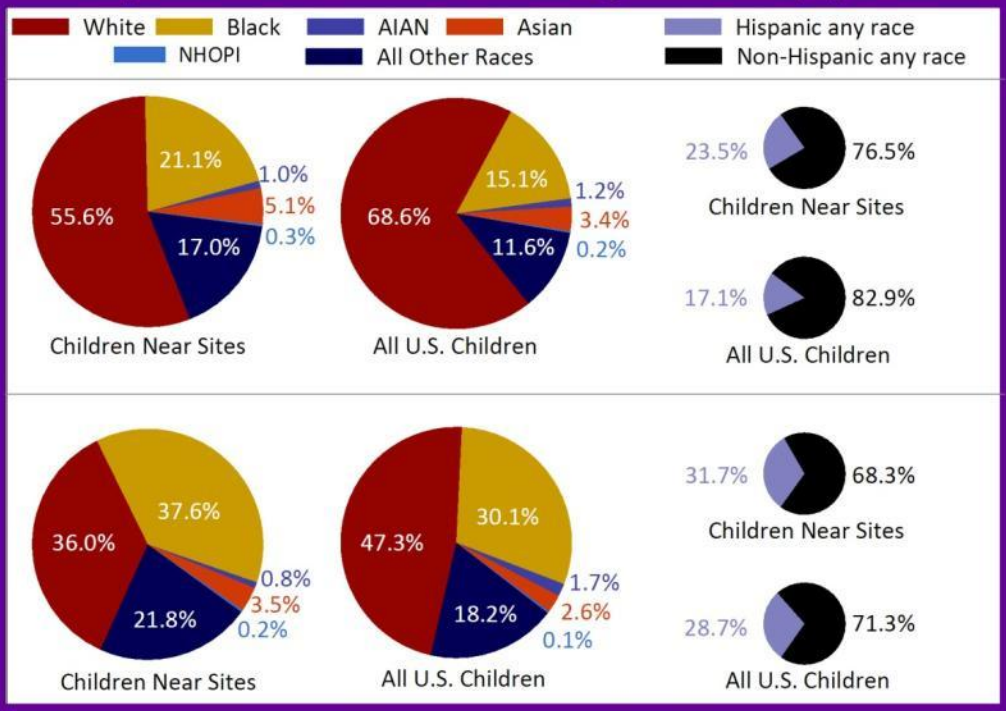
- Data on Superfund and RCRA Corrective Action sites are reported by EPA regional offices and states, and compiled in EPA's databases of information on contaminated sites.
- Information for each site includes the site name, state in which the site is located, latitude, longitude, estimated acreage, and site status.
- Areas of known or suspected contamination may be less than the total acreage at each site.

- Approximately 6% of all children in the United States lived within one mile of a Corrective Action or Superfund site that may not have had all human health protective measures in place as of 2009.

- About 8% of Black children, 9% of Asian children, 9% of children of All Other Races, and 10% of Native Hawaiian and Other Pacific Islander (NHOPI) children lived in proximity to the designated sites. About 8% of Hispanic children, who may be of any race, lived in proximity to the sites. In contrast, about 5% of White children and 5% of American Indian/Alaska Native children lived in proximity to the designated sites.
- About 8% of all children in the United States in families with incomes below the poverty level lived within one mile of the designated sites, compared with about 5% of children above the poverty level. The proportion of children below the poverty level in proximity to the designated sites was generally greater than the proportion for those above poverty level for each race and ethnicity; the only exception to this pattern was for American Indian and Alaskan Native (AIAN) children.

Indicator E11

Distribution by race/ethnicity and family income of children living near selected contaminated lands* in 2009, compared with the distribution by race/ethnicity and income of children in the general U.S. population



Data: U.S. EPA, Office of Solid Waste and Emergency Response, CERCLIS and RCRAInfo

Note: AIAN = American Indian/Alaska Native. NHOPI = Native Hawaiian or Other Pacific Islander. Hispanic children may be of any race.

* Within one mile of Superfund and Corrective Action sites that may not have all human health protective measures in place.

America's Children and the Environment, Third Edition

Data characterization

- Data on Superfund and RCRA Corrective Action sites are reported by EPA regional offices and states, and compiled in EPA's databases of information on contaminated sites.
- Information for each site includes the site name, state in which the site is located, latitude, longitude, estimated acreage, and site status.
- Areas of known or suspected contamination may be less than the total acreage at each site.

- Approximately 21% of all children living within one mile of a Corrective Action or Superfund site that may not have had all human health protective measures in place were Black, while 15% of children in the United States as a whole are Black. Black children account for about 30% of all U.S. children in homes below poverty level; among children below poverty level living within one mile of a designated site, about 38% were Black.

- The percentages of Asian children, Hispanic children, and children of "All Other Races" among children living close to the designated sites were also greater than the percentages of these children in the entire U.S. population, considering all incomes and considering only those in homes with incomes below poverty level.

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Climate Change

Climate change refers to any significant change in climate variables including temperature, precipitation, or wind that lasts for decades or longer. It may include changes in variability of average weather conditions or extreme weather conditions. Both human activities and natural factors contribute to climate change. Human activities, such as burning fossil fuels; cutting down forests; and developing land for farms, cities, and roads, release heat-trapping greenhouse gases into the atmosphere. Natural causes, such as changes in the Earth's orbit, the sun's intensity, the circulation of the ocean and the atmosphere, and volcanic activity, contribute to climate change in a variety of ways.[1]

Climate change may increase children's exposure to extreme temperatures, polluted air and water, extreme weather events, wildfires, infectious disease, allergens, pesticides, and other chemicals. These exposures may affect children's health in a number of direct and indirect ways. It is important to note that climate change will likely result in a mix of both positive and negative health impacts. For example, warmer summers may increase the number of heat-related injuries and deaths, while warmer winters may result in fewer cases of cold-related injuries and deaths.[2] The effects of climate change will also vary from one location to another and will likely change over time as climate change continues.[2,3] Furthermore, the human health risks from climate change may be affected strongly by changes in health care advances and accessibility, public health infrastructure, and technology.[2,4-6]

Direct effects of extreme temperatures are one area of concern, as climate change is expected to increase the number and intensity of hot days, hot nights, and heat waves in the United States.[5,7,8] Heat exposure can result in heat rashes, heat stroke, heat exhaustion, and even death; children may be especially at risk because they often spend more time outside than adults do.[2,9] Children's bodies are less effective at adapting to heat compared with those of adults.[10] Also, children may not feel the need to drink as urgently, which can lead to severe dehydration and electrolyte imbalance.[10,11] Humidity can further exacerbate heat stress in children.[10,11] Infants may be especially vulnerable to heat events in part because they depend on adults for care and are unable to communicate thirst and discomfort.[6,12,13] Caregivers can help protect children from heat-related health effects.[14]

Many factors can modify the impact of heat exposure, including geographic location, income level, and the built environment.[15] Studies have shown that the temperature at which mortality and morbidity (e.g., respiratory hospital admissions) can occur from heat exposure varies based on location.[16-18] Extreme heat exposure may have a greater impact on populations living in regions that experience high temperatures less frequently, such as the Northwest and Midwest United States. In warmer climates such as those in the South and Southwest United States, the population may be acclimated to heat and area infrastructure is better designed to accommodate high temperatures.[13,19] A higher income allows families to adapt more easily to meet the challenges of climate change compared with lower-income families, because they can

afford the use of air conditioners and other cooling methods to create a more ideal and comfortable environment.[3]

The urban built environment can both exacerbate and alleviate the effects of heat. For example, high concentrations of buildings in urban areas cause what is known as the urban heat island effect: generating as well as absorbing and releasing heat, resulting in urban centers that are several degrees warmer than surrounding areas. Expanding the area of parks and green spaces and increasing the density of trees in and around cities can help to reduce this effect.[6]

Warmer winters may have the effect of decreasing the number of cold-related deaths and injuries.[2,15] It is difficult to estimate the net changes in mortality due to climate change; however, a recent assessment by the United States Global Change Research Program concluded that increases in heat-related mortality due to climate change are unlikely to be compensated by decreases in cold-related mortality.[8]

High temperatures, heat waves, and associated stagnant air masses can increase levels of air pollution, specifically ground level ozone, fine particulate matter ($PM_{2.5}$), nitrogen oxides, and sulfur oxides.[2,6,8,9] These air pollutants can be harmful for children: they may contribute to the development of new cases of asthma, aggravate preexisting cases of asthma, cause decrements to lung function, increase respiratory symptoms such as coughing and wheezing, and increase hospital admissions and emergency room visits for respiratory diseases.[20-35] Because children may spend a lot of time outdoors, often while exerting themselves for sports or play, they can be especially vulnerable to the impacts of poor air quality.[8]

Climate change is likely to change the timing, frequency, and intensity of extreme weather events, including heat waves, hurricanes, heavy rainfall, droughts, high coastal waters, and storm surges.[5,36] These events can cause traumatic injury and death, as well as emotional trauma. Extreme weather events are also associated with increased risk of food- and water-borne illnesses as sanitation, hygiene, and safe food and water supplies are often compromised after these types of events.[2] One study found that periods of heavy rainfall were associated with increased emergency room visits for gastrointestinal illness among children.[37] Heavy rainfall may result in flooding, which can lead to contamination of water with dangerous chemicals, heavy metals, or other hazardous substances from storage containers or from preexisting chemical contamination already in the environment.[2,36] Elevated temperatures and low precipitation are also projected to increase the size and severity of wildfires. This can lead to increased eye and respiratory illnesses and injuries, which include burns and smoke inhalation.[2] Extreme weather events can be especially dangerous for children because they are dependent on adults for care and protection.[7]

A number of infectious diseases may be affected by climate change. The combined effects of increased temperature and precipitation are projected to cause increases in some water-, food-, and vector-borne illnesses. In general, increased temperature results in higher replication, transmission, persistence, habitat range, and survival of bacterial pathogens (the effect on viral pathogens is less clear), and produces a greater number of water- and food-borne parasitic

infections.[5,6,8] Climate change is also expected to expand or shift the habitat and range of disease-carrying organisms, such as mosquitoes, ticks, and rodents.[5] Changes in the geographic distribution of disease-carrying organisms may alter the spread of vector-borne diseases such as Lyme disease, West Nile virus and Dengue fever.[5] Children may be at greater risk for these types of infectious diseases as they spend more time outdoors compared with adults, where they might contact disease-carrying organisms, and they have less-developed immune systems.[14]

Climate change, including changes in carbon dioxide (CO_2) concentrations and temperature, may affect the growth and distribution of allergen-producing vegetation such as weeds, grasses, and trees. Climate change has already caused an earlier onset of the U.S. spring pollen season and a lengthened ragweed season.[15,38] The aeroallergens (e.g., pollen) themselves might be changed in terms of production, distribution, dispersion, and allergic potency.[2,6,15] Exposure to weed and grass pollen has been associated with exacerbation of children's asthma, emergency room visits, and hospitalizations.[39-41]

Through various indirect pathways, climate change may lead to increasing levels and/or frequencies of childhood exposure to harmful contaminants.[6,14] Changes in temperature, rainfall, and crop practices related to climate change are likely to affect exposure to pathogens, pesticides, and other chemicals in a number of ways. Broader geographic distribution of pests and increased growth of invasive weeds will likely lead to greater use of pesticides.[6,8] Increased precipitation and increased variability in precipitation are likely to increase pathogen and contaminant levels in lakes and other surface waters.[2,42] The distribution of chemicals in the environment is likely to change: for example, an increase in ice melts caused by a warming climate may release some past emissions of globally transported chemicals, such as PCBs and mercury, that have been trapped in polar ice.[43,44] Increasing concentrations of these chemicals in the atmosphere, and subsequent deposition to land and water, have the potential to increase concentrations of these chemicals in fish and other foods derived from animals. Warmer water temperatures may also increase the release of chemical contaminants from sediments, increasing their uptake in fish.[2] Climate change may result in children spending more time indoors. Buildings that are tightly sealed in response to adverse weather conditions may result in increased exposure to contaminants from poor ventilation and higher concentrations of indoor pollutants such as radon, environmental tobacco smoke, and formaldehyde.[45]

Children are expected to be especially sensitive to the effects of climate change for a number of reasons. Young children and infants are particularly vulnerable to heat-related illness and death.[6] Compared with adults, children have higher breathing rates, spend more time outside, and have less developed respiratory tracts—all making children more sensitive to air pollutants. Additionally, children have immature immune systems, meaning that they can experience more serious impacts from infectious diseases.[8] The greatest impacts are likely to fall on children in poor families, who lack the resources, such as adequate shelter and access to air conditioning, to cope with climate change.[8]

EPA is currently developing a new children's environmental health indicator for climate change. The new indicator will focus on the frequency of extreme heat events over time. EPA intends to complete development of this new indicator in 2014, and it will be made available at www.epa.gov/ace when completed.

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Introduction

What is biomonitoring?

In the field of human exposure assessment, biomonitoring refers to the measurement of chemicals in human body fluids and tissues, such as blood, urine, breast milk, saliva, and hair. Measurements of the levels of pollutants in children's bodies provide direct information about their exposures to environmental contaminants. Measurements in women who may become pregnant, currently are pregnant, or currently are breastfeeding provide information about exposures that may affect conception, the fetus, or the developing child.

Biomonitoring measurements provide an estimate of the amount of a chemical absorbed into the body from all pathways of exposure (for example, ingestion of drinking water, inhalation of air), and thus give a cumulative estimate of the chemical burden that a person carries in their body, sometimes referred to as a body burden. Biomonitoring can characterize differences in exposure among groups within a population, and can characterize changes in population exposure over time. Biomonitoring is an increasingly important element of epidemiological research when evaluating whether chemical exposures are associated with adverse health effects in humans.

What environmental chemicals are included in the Biomonitoring indicators for *America's Children and the Environment, Third Edition (ACE3)*?

Biomonitoring topics were selected for ACE3 based on: (1) research that indicates an association between exposure and children's health or suggests a potential association between exposure and children's health; (2) significant public interest; and (3) the nature of the biomonitoring data available (for example, range of ages for which data are available and frequency of detection). EPA obtained input from its Children's Health Protection Advisory Committee to assist in selecting topics from among the many chemicals with biomonitoring data available. The ACE3 Biomonitoring indicators address the following topics:

- Lead
- Mercury
- Cotinine (a marker for environmental tobacco smoke exposure)
- Perfluorochemicals (PFCs)
- Polychlorinated biphenyls (PCBs)
- Polybrominated diphenyl ethers (PBDEs)
- Phthalates
- Bisphenol A (BPA)
- Perchlorate

For many of the chemicals addressed in this section, scientific findings have reported associations between children's health and the mother's exposure during pregnancy. For this reason, indicators for several of these topics present data for women of child-bearing age—defined here as ages 16 to 49 years.

What data sources were used to develop the Biomonitoring indicators?

Biomonitoring data are generated by collecting samples of blood, urine or other biological specimens from a group of individuals, then measuring the concentrations of selected chemicals in those specimens. There are many scientific research efforts that collect biomonitoring data in the United States, but only the National Health and Nutrition Examination Survey (NHANES) conducted by the National Center for Health Statistics (NCHS) measures chemicals in the blood and urine[1] of a nationally representative sample of the U.S. population. NHANES was therefore identified as the most suitable data source for all Biomonitoring indicators presented in ACE3. Summary statistics for more than 200 chemicals measured in NHANES are reported in the *Fourth National Report on Human Exposure to Environmental Chemicals*,[1] and data files containing individual measurements for each chemical are available from the NHANES website.[2]

Because NHANES is an ongoing, continuous survey that provides data over a number of years using a consistent sample design and consistent methods of measurement, the biomonitoring levels can be compared over time and across demographic groups. However, because of the highly clustered sample design of the survey, multiple NHANES cycles should be combined to yield sample sizes necessary for certain types of statistical analysis. NHANES is not designed to provide detailed estimates for populations that are highly exposed to particular environmental chemicals. In addition, military personnel and people who reside in institutions are excluded from NHANES.

For most of the environmental chemicals currently measured in NHANES, data are available starting in 1999 or more recently; measurement of lead and cotinine began earlier. Availability of NHANES biomonitoring data for children varies by chemical and type of sample, and, in general, biomonitoring data for young children are quite limited. For environmental chemicals measured in urine, NHANES collects data from survey participants ages 6 years and older. For most environmental chemicals measured in blood, NHANES collects data from survey participants ages 12 years and older. Exceptions apply to three chemicals presented in this section: measurements of lead and mercury in blood are conducted for all survey participants ages 1 year and older, and measurements of cotinine in blood are conducted for all participants ages 3 years and older. NHANES does not measure chemicals in breast milk, an important route of exposure for infants, or in target organs where chemicals affect the body.

What can we learn from biomonitoring indicators?

Biomonitoring indicators in ACE3 provide summaries of biomonitoring measurements in blood or urine specimens obtained from a nationally representative target population—either

children within a specified age range, or women of child-bearing age. For chemicals that are persistent in the human body, biomonitoring measurements may be reflective of exposures that have occurred over several months or years. For chemicals that are cleared from the body more rapidly, a biomonitoring measurement may typically reflect exposures that have occurred within the previous few days.

The Biomonitoring indicators prepared for ACE3 focus primarily on presenting biomonitoring data collected over multiple years to evaluate whether there are any changes over time. The biomonitoring indicator values are also compared across various race/ethnicity, income, or age groups.

When health benchmarks are available, biomonitoring data may provide insights about the percentage of a population at risk for adverse health effects; however, in most cases information on health risks associated with levels of chemicals in blood or urine typical for the general population is limited. For some chemicals, such as lead and cotinine, there is an extensive body of literature demonstrating that adverse effects can occur in children with levels of exposure commonly experienced in the general population. However, biomonitoring by itself does not reveal whether any adverse effects have occurred in an individual or in the population.

Biomonitoring indicators present data for one chemical at a time, but biomonitoring studies have found that individuals have multiple chemicals in their bodies.[3-5] While the evidence is still developing for the links between exposures to environmental chemicals and disease, a wide variety of chemicals may act together to produce common adverse outcomes.[6] Thus, even small biological alterations caused by exposure to a single chemical in isolation may have important effects when combined with exposure to other chemicals. The ACE3 Biomonitoring indicators do not reflect this context of simultaneous or sequential exposure to multiple chemicals.

An important limitation of biomonitoring is that, by itself, it provides few clues as to the source(s) of exposure. Data on environmental sources of the chemical are necessary to separate contributions from air, water, food, and/or contaminated soil or dust.

What information is provided for each Biomonitoring topic?

For each topic, an introduction section explains the potential relevance of the chemical to children's health, including a discussion of typical exposure pathways and scientific findings concerning possible adverse health effects.

The introduction section is followed by a description of the indicators, including a summary of the data available from NHANES for the specific chemical or chemical group and information on how each indicator was calculated. One or two indicators, each presented as a graphical representation of the available data, are included for each topic. Where data are available for a sufficient number of years (at least three NHANES two-year cycles), the indicator presents a

time series. When time series data are not available, the indicator shows a comparison of the most current biomonitoring data by race/ethnicity and income level.

All indicator figures present median (50th percentile) values; some time series figures also provide 95th percentile values. The median is the value in the middle of the chemical's distribution: half of the measured population has levels of the chemical in their urine or blood that are greater than the median, and half has levels below the median. The 95th percentile is a value representing the upper range of levels: 5% of the specified group has levels of the chemical in their urine or blood that are greater than the 95th percentile. This value therefore can be thought of as representing a high level relative to the rest of the population, but not a maximum level.[i]

Beneath each figure is a description of the data source and explanatory bullet points highlighting key findings from the data presented in the figure, along with key data from any supplemental data tables. References are provided for each topic at the end of the report.[ii]

Data tables are provided in Appendix A. The tables include all indicator values depicted in the indicator figures, along with additional data of interest not shown in the figures. Metadata describing the data sources are provided in Appendix B. Documents providing details of how the indicators were calculated are available on the ACE website (www.epa.gov/ace).[iii]

Many of the topics presented in the biomonitoring indicators are addressed in Healthy People 2020, which provides science-based, 10-year national objectives for improving the health of all Americans. Appendix C provides examples of the alignment of the biomonitoring topics presented in ACE3 with objectives in Healthy People 2020.

What race/ethnicity groups are used in reporting indicator values?

For each topic in the Biomonitoring section, indicator values are provided for defined race/ethnicity groups—either in the indicator figures or in the data tables—for the following races/ethnicities:

- White non-Hispanic
- Black non-Hispanic

[i] Frequently, a small portion of the population may appear to have much higher levels of an environmental chemical compared with everyone else. In these cases, percentiles in the lower portion of the distribution (below the median) are generally less variable than those well above the median. In NHANES, estimates above the 95th percentile are generally very uncertain due to the sample size, the survey design, and (for many measurements) the substantially skewed distributions.

[ii] In the current copy, references are provided at the end of each topic section. In the final printed report, references will appear, separated by topic, at the end of the report.

[iii] The Methods documents will be posted to the ACE website when the final ACE3 report is posted in early 2013.

- Mexican-American
- All Other Races/Ethnicities

Values are provided for "Mexican-American" ethnicity rather than "Hispanic" ethnicity because in all years up to 2006, NHANES was designed to provide statistically reliable estimates for Mexican-Americans rather than all Hispanics.[iv]

The "All Other Races/Ethnicities" category includes all other races and ethnicities not specified, together with those individuals who report more than one race. The limits of the sample design and sample size often preclude statistically reliable estimates for smaller race/ethnicity groups.[v]

What income groups are used in reporting indicator values?

The ACE3 Biomonitoring indicators present values for income groups defined on the basis of the federal poverty level. Poverty level is defined by the federal government, and is based on income thresholds that vary by year, family size and composition. In 2010, for example, the poverty threshold was $22,113 for a household with two adults and two related children.[7] The biomonitoring indicators (in figures and/or data tables) provide data separately for individuals in families with incomes below poverty level, and those in families with incomes at or above poverty level.

How were the indicators calculated and presented?

Data files: All indicators were calculated from publicly available data files obtained from the NHANES website. Files include values for the biomonitoring measurement, and information on the sampled individual's age, sex, race/ethnicity, and income level (that is, the family income divided by the poverty level). Each individual observation also has a sample weight that is used in calculating population statistics; the weight equals the number of people in the U.S. population represented by the particular observation.

Population age groups: Indicators of biomonitoring data in children used all data available for children ages 17 years and younger, except for lead where the indicator focuses on children ages 5 years and younger. Indicators of biomonitoring data in women of child-bearing age used all available data for women ages 16 to 49 years. As noted above, indicators for women of child-bearing age are included in ACE3 when there are concerns for children's health associated with the mother's exposure during pregnancy. Adjustments were applied in calculating the population distribution of women ages 16 to 49 years to incorporate birth rates specific to age and race/ethnicity.[8] These adjustments give greater weight to women of ages more likely to

[iv] NHANES now oversamples Hispanics instead of Mexican-Americans, beginning with NHANES 2007–2008. Please see http://www.cdc.gov/nchs/nhanes/nhanes2007-2008/sampling_0708.htm/ .

[v] Separate estimates for Asians may be feasible for some biomonitoring measures in the future, as NHANES started oversampling Asians in the 2011-2012 cycle.

give birth, and reduce the contribution to the calculated indicator values of women of ages less likely to give birth (e.g., those ages 40 to 49 years). Without the birth rate adjustment, the indicator values would be calculated as if all women ages 16 to 49 years are equally likely to give birth.[vi]

Calculation of 50th and 95th percentiles over specified time periods: For all ACE3 Biomonitoring indicators, the 50th and 95th percentile values were selected as the indicator statistics to represent the central tendency and upper end of the exposure distribution. Where data are available for at least three 2-year NHANES survey periods, the indicator presentation focuses on how the measured values have changed over time. If data are available for only one or two NHANES survey periods, the indicator presentation focuses on demographic comparisons.

The 50th and 95th percentiles were also calculated for different population groups (defined by race/ethnicity or income) for all chemicals considered in the indicators. A single two-year NHANES cycle frequently will not include enough sampled individuals to provide statistically reliable estimates for all population groups of interest. Four-year data sets were used to ensure that there were a sufficient number of observations for each population group, using the two most current two-year NHANES cycles reported for each chemical. All calculations incorporated the NHANES sample weights.

Statistical considerations in presenting and characterizing the indicators: Statistical analysis has been applied to the ACE3 Biomonitoring indicators to evaluate trends over time in indicator values (for example, median concentration of lead in blood), or differences in indicator values between demographic groups.[vii] These analyses use a 5% significance level, meaning that a conclusion of statistical significance is made only when there is no more than a 5% probability that the observed trend or difference occurred by chance ($p \leq 0.05$).

[vi] The adjustment involves calculating age- and race/ethnicity-specific birth rates. Birth rates (i.e., average number of births per woman annually) are derived for each single year of age (age 16, age 17, etc.) separately for each race/ethnicity group (White non-Hispanic, Black non-Hispanic, Mexican-American, and "All Other Races/Ethnicities"). The standard NHANES sample weight for each observation is then multiplied by the calculated birth rate corresponding to the age and race/ethnicity of the sampled woman. This produces a birth rate-adjusted weight that is applied in the same manner as standard NHANES sample weights. There may be multiple ways to implement an adjustment to the data that accounts for birth rates by age. The National Center for Health Statistics has not fully evaluated the method used in ACE, or any other method intended to accomplish the same purpose, and has not used any such method in its publications. NCHS and EPA are working together to further evaluate the birth rate adjustment method used in ACE and alternative methods.

[vii] The approach used in ACE3 focuses on identifying statistical trends and differences in the 50th and 95th percentiles of the NHANES biomonitoring data. Other approaches to analyzing trends in the NHANES biomonitoring data may focus on different summary statistics, such as the geometric mean or the percentage of the population exceeding some designated level. Assessment of trends in other summary statistics (such as the geometric mean) will not necessarily lead to the same conclusions as assessments of trends in the 50th and 95th percentiles.

The statistical analysis of trends over time for an ACE3 Biomonitoring indicator is dependent on how the indicator values vary over time, the number of NHANES survey cycles with data included in the analysis, the number and variability of measurements in each survey cycle, and various aspects of the survey design. The evaluation of trends over time incorporates data from each survey cycle within the time period reported (for example, 2001–2002, 2003–2004, 2005–2006, 2007–2008, and 2009–2010). A finding of statistical significance for differences in indicator values between demographic groups depends on the magnitude of the difference, the number and variability of measurements in each group, and various aspects of the survey design. For example, if two groups from the U.S. population have different median levels of a chemical in blood or urine, the statistical test is more likely to detect a difference when samples have been obtained from a larger number of people in those groups. Similarly, if there is low variability in measured levels of the chemical within each group, then a difference between groups is more likely to be detected. It should be noted that when statistical testing is conducted for differences among multiple demographic groups (for example, considering both race/ethnicity and income level), or for multiple chemicals, the large number of comparisons involved increases the probability that some differences identified as statistically significant may actually have occurred by chance.

A finding of statistical significance is useful for determining that an observed trend or difference was unlikely to have occurred by chance. However, a determination of statistical significance by itself does not convey information about the magnitude of the difference in chemical concentrations or the potential difference in the risk of associated health outcomes. Furthermore, a lack of statistical significance means only that occurrence by chance cannot be ruled out. Thus, a conclusion about statistical significance is only part of the information that should be considered when determining the public health implications of trends or differences in indicator values.

In some cases, calculated indicator values have substantial uncertainty. Uncertainty in these estimates is assessed by looking at the relative standard error (RSE), a measure of how large the variability of the estimate is in relation to the estimate (RSE = standard error divided by the estimate).[viii] The estimate should be interpreted with caution if the RSE is at least 30% but is less than 40%; a notation is provided for such estimates in the indicator figures and tables. If the RSE is greater than 40%, the estimate is considered to have very large uncertainty and is not reported.[ix]

[viii] Standard errors for all biomonitoring indicator values are provided in a file available on the ACE website (www.epa.gov/ace). (This file will be posted to the ACE website when the final ACE3 report is posted in early 2013.)

[ix] The RSE itself may also be uncertain for some estimates, particularly for values based on small samples, such as values stratified by race/ethnicity or income and chemicals measured in a subsample of NHANES participants (rather than all NHANES participants). Degrees of freedom is a statistical measure that provides an indication of this uncertainty. Estimates with between 7 and 11 degrees of freedom have a notation stating that they should be

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interpreted with caution. Estimates with fewer than 7 degrees of freedom were considered unreliable and are not reported.

Lead

Lead is a naturally occurring metal used in the production of fuels, paints, ceramic products, batteries, solder, and a variety of consumer products. The use of leaded gasoline and lead-based paint was eliminated or restricted in the United States beginning in the 1970s, resulting in substantial reductions in exposure to lead. However, children continue to be exposed to lead due to the widespread distribution of lead in the environment. For example, children are exposed to lead through the presence of lead-based paint in many older homes, the presence of lead in drinking water distribution systems, and current use of lead in the manufacture of some products.

In the United States, the major current source of early childhood lead exposure is lead-contaminated house dust.[1,2] Exposure to lead in house dust tends to be highest for young children, due to their frequent and extensive contact with floors, carpets, window areas, and other surfaces where dust gathers, as well as their frequent hand-to-mouth activity. A major contributor to lead in house dust is deteriorated or disrupted lead-based paint.[3-5] Housing units constructed before 1950 are most likely to contain lead-based paint, but any housing unit constructed before 1978 may also contain lead-based paint.[6] As of 2000, approximately 15.5 million housing units in the United States had one or more lead dust hazards on either floors or windowsills.[7] New lead dust hazards occur when lead in house paint is released during home renovation and remodeling activities.[8,9]

Two other contributors to lead in house dust are lead-contaminated soil and airborne lead.[10-13] Known sources of lead in soil include historical airborne emissions of leaded gasoline, emissions from industrial sources such as smelters, and lead-based paint.[14,15] Current sources of lead in ambient air in the United States include smelters, ore mining and processing, lead acid battery manufacturing, and coal combustion activities such as electricity generation.[15]

Lead-contaminated house dust is not the only source of childhood lead exposure. Direct contact with lead-contaminated soil,[13] ingestion of lead-based paint chips,[16] and inhalation of lead in ambient air also contribute to childhood lead exposure. Drinking water is an additional known source of lead exposure among children in the United States, particularly from corrosion of pipes and other elements of the drinking water distribution systems.[5,17,18] Exposure to lead via drinking water may be particularly high among very young children who consume baby formula prepared with drinking water that is contaminated by leaching lead pipes.[17] Although childhood exposure to lead in the United States typically occurs through contact with contaminated environmental media; children may also be exposed through lead-contaminated toys;[5,19] jewelry;[20] tobacco smoke;[21] imported candies, spices, and condiments;[5,22] and imported folk remedies.[23,24]

Compared with adults, children's bodies typically absorb a much greater fraction of a given amount of ingested lead. Once absorbed, most of the lead is stored in bones, where it can stay

many years, while other lead goes into the blood and can be eliminated more quickly. Elimination of lead from the body usually occurs through urine or feces.[25]

Childhood blood lead levels in the United States differ across groups in the population, such as those defined by socioeconomic status and race/ethnicity.[26] Children living in poverty and Black non-Hispanic children tend to have higher blood lead levels[27] and higher levels of lead-contaminated dust in the home[6] than do other children. Blood lead levels tend to be higher for children living in older housing, most likely because older housing units are more likely to contain lead-based paint.[6,28] Blood lead levels may vary by nutritional status: conditions such as iron deficiency have been associated with higher blood lead levels in children.[15] In addition, some children who have immigrated to the United States may have been exposed to lead in their previous countries of residence. Foreign birth place and recent foreign residence have both been positively associated with the risk of elevated blood lead levels among immigrant children in the United States.[27,29]

Childhood blood lead levels in the United States have declined substantially since the 1970s. The decline in blood lead levels is due largely to the phasing out of lead in gasoline between 1973 and 1995,[30] and to the reduction in the number of homes with lead-based paint hazards.[7] Some decline was also a result of regulations reducing lead levels in drinking water, as well as legislation limiting the amount of lead in paint and restricting the content of lead in solder, faucets, pipes, and plumbing, and the elimination of lead-soldered cans for food use.[5] In the United States, lead content is banned or limited in many products, including food and beverage containers, ceramic ware, toys, Christmas trees, polyvinyl chloride pipes, vinyl mini-blinds, and playground equipment.[5] However, because trace levels of lead may be present in these products, normal use may still result in lead exposure.[5]

The National Toxicology Program (NTP) has concluded that childhood lead exposure is associated with reduced cognitive function.[31] Children with higher blood lead levels generally have lower scores on IQ tests[32-38] and reduced academic achievement.[31] In addition to the effects on IQ and school performance, research on the effects of lead has increasingly been addressing the effects of lead on behavior. The NTP has concluded that childhood lead exposure is associated with attention-related behavioral problems (including inattention, hyperactivity, and diagnosed attention-deficit/hyperactivity disorder) and increased incidence of problem behaviors (including delinquent, criminal, or antisocial behavior).[31] Studies have reported that lead exposure in children may contribute to decreased attention,[38-43] hyperactivity-impulsivity,[44] and increased likelihood of attention-deficit/hyperactivity disorder.[44-52] Other adverse behavioral outcomes that have been associated with childhood lead exposure in some studies include conduct disorders,[53,54] increased risks of juvenile delinquency and antisocial behaviors,[55-57] higher total arrest rates, and arrest rates for violent crimes in early adulthood.[58,59] Socioeconomic status may also modify the effect of lead on these cognitive and behavioral changes, resulting in stronger effects in children with lower socioeconomic status.[60,61]

Mothers who are exposed to lead can transfer lead to the fetus during pregnancy and to the child while breast feeding.[62,63] The NTP has concluded that there is "limited evidence" that prenatal lead exposure is associated with cognitive and behavioral effects in children.[31] The Centers for Disease Control and Prevention (CDC) has recently published guidelines for screening pregnant and lactating mothers for possible lead exposure to better protect the fetus.[64]

Many studies of the effects of lead focus on outcomes in children ages 5 years and younger. This focus reflects scientific thinking that early childhood is when children tend to experience peak exposures to lead, and also when they are most biologically susceptible to the effects of lead. Increased susceptibility to the neurodevelopmental effects of lead in the first three years of life is expected because this period is characterized by major growth and developmental events in the nervous system.[15] However, lead is toxic to individuals of all ages, and children older than 5 years may also be susceptible to the neurodevelopmental effects of lead. Blood lead measurements at various ages in early childhood have been found to be strongly correlated with cognitive deficits,[36] and some analyses have found that effects are more strongly associated with blood lead levels at school age (i.e., 5- to 6-year-old children) compared with levels measured earlier in life.[65,66]

Childhood lead exposures may also have lifelong effects. For instance, high childhood blood lead concentrations are associated with significant region-specific brain volume loss in adults, with greater effects seen in males.[67,68] Childhood blood lead concentrations are also inversely associated with intellectual functioning in young adulthood.[69] In addition, lead stored in bones has the potential to be released into the bloodstream later in life. Such is the case with pregnant women, breastfeeding women, and elderly persons, as blood lead levels are comparatively elevated in these populations.[25,70,71] Finally, childhood exposures to lead may contribute to a variety of neurological disorders and neurobehavioral effects in later life. [25,71-73]

Until recently, CDC defined a blood lead level of 10 micrograms per deciliter ($\mu g/dL$) as "elevated"; this definition was used to identify children for blood lead case management.[72,74] However, no level of lead exposure has been identified that is without risk of deleterious health effects.[15] CDC's Advisory Committee on Childhood Lead Poisoning Prevention (ACCLPP) recommended in January 2012 that the 97.5th percentile of children's blood lead distribution (currently 5 $\mu g/dL$) be defined as "elevated" for purposes of identifying children for follow-up activities such as environmental investigations and ongoing monitoring.[75] CDC has adopted the ACCLPP recommendation.[76] CDC specifically notes that "no level of lead in a child's blood can be specified as safe,"[1] and the NTP has concluded that there is sufficient evidence for adverse health effects in children at blood lead levels less than 5 $\mu g/dL$.[31]

The following two indicators use the best nationally representative data available on blood lead levels over time in children. Indicators B1 and B2 present blood lead concentrations for children ages 1 to 5 years.

Indicator B1: Lead in children ages 1 to 5 years: Median and 95th percentile concentrations in blood, 1976–2010

Indicator B2: Lead in children ages 1 to 5 years: Median concentrations in blood, by race/ethnicity and family income, 2007–2010

About the Indicators: Indicators B1 and B2 present concentrations of lead in blood of U.S. children ages 1 to 5 years. The data are from a national survey that collects blood specimens from a representative sample of the population every two years, and then measures the concentration of lead in the blood. Indicator B1 presents concentrations of lead in blood over time. Indicator B2 shows how blood lead levels differ by race/ethnicity and family income.

NHANES

The National Health and Nutrition Examination Survey (NHANES) provides nationally representative biomonitoring data for lead. NHANES is designed to assess the health and nutritional status of the civilian noninstitutionalized U.S. population and is conducted by the National Center for Health Statistics, part of the Centers for Disease Control and Prevention (CDC). NHANES conducts interviews and physical examinations with approximately 10,000 people in each two-year year survey cycle. CDC's National Center for Environmental Health measures concentrations of environmental chemicals in blood and urine samples collected from NHANES participants. Summaries of the measured values for more than 200 chemicals are provided in the *Fourth National Report on Human Exposure to Environmental Chemicals*.[77]

Lead

Indicators B1 and B2 present levels of lead in children's blood. Blood lead levels are reflective of relatively recent exposure and, to a varying extent across individuals, may also incorporate contributions of long-term lead exposures.[15] All values are reported as micrograms of lead per deciliter of blood (µg/dL).

Concentrations of lead in the blood of children have been measured in NHANES beginning with the 1976–1980 survey cycle (referred to as NHANES II). For 2009–2010, NHANES collected lead biomonitoring data for 8,793 individuals ages 1 year and older, including 836 children ages 1 to 5. Lead was detected in 100% of all individuals sampled. The median blood lead level among all NHANES participants in 2009–2010 was 1.1 µg/dL and the 95th percentile was 3.3 µg/dL.

Data Presented in the Indicators

Indicator B1 presents median and 95th percentile concentrations of lead in blood over time for children ages 1 to 5 years, using NHANES data from 1976–2010.

Indicator B2 presents current median concentrations of lead in blood for children ages 1 to 5 years of different races/ethnicities and levels of family income, using NHANES data from 2007–2008 and 2009–2010.

The data from two NHANES cycles are combined to increase the statistical reliability of the estimates for each race/ethnicity and income group, and to reduce any possible influence of geographic variability that may occur in two-year NHANES data. The current 95th percentiles of blood lead by race/ethnicity and income are presented in the data tables.

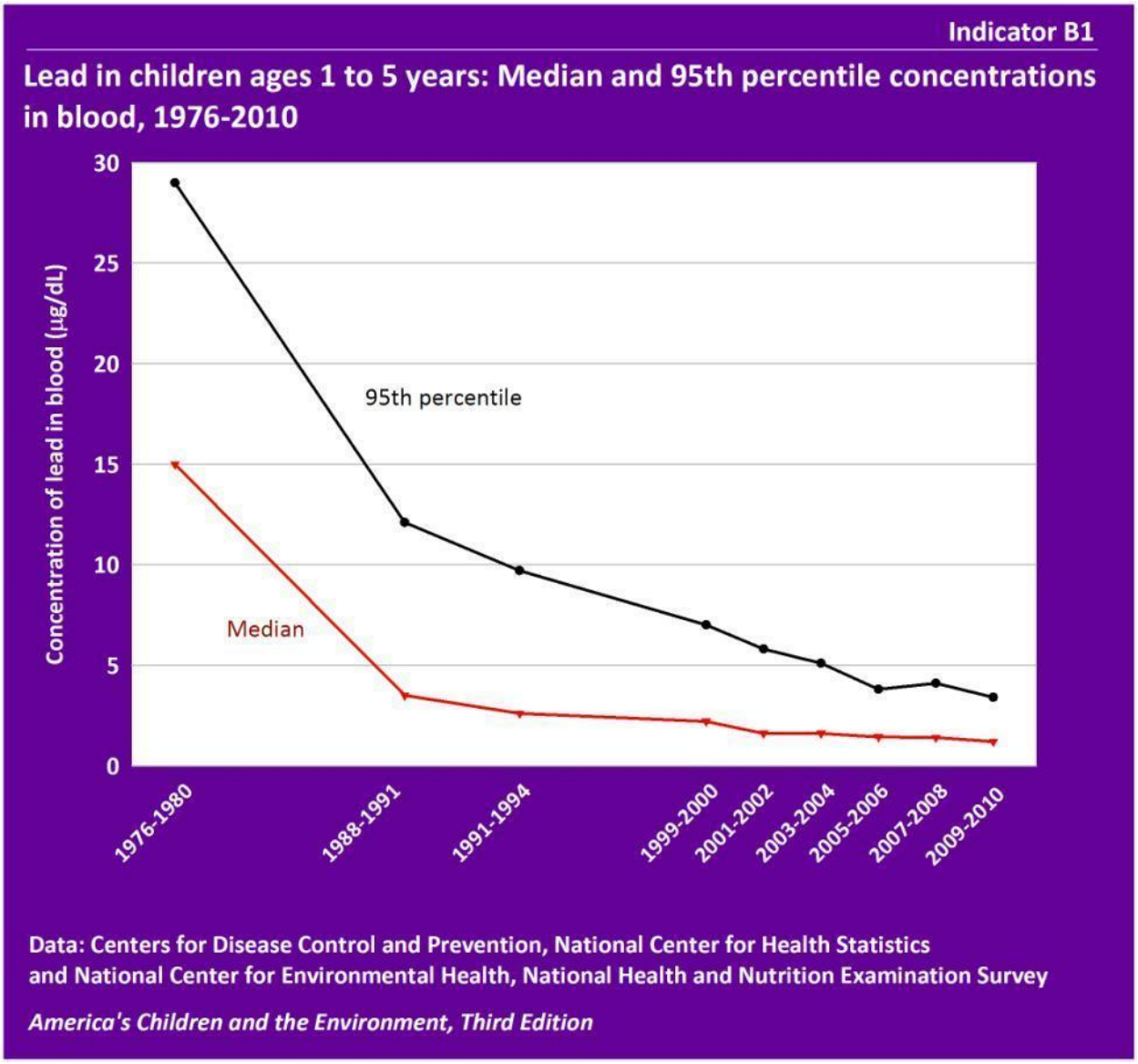
Four race/ethnicity groups are presented in Indicator B2: White non-Hispanic, Black non-Hispanic, Mexican-American, and "All Other Races/Ethnicities." The "All Other Races/Ethnicities" category includes all other races and ethnicities not specified, together with those individuals who report more than one race. The limits of the sample design and sample size often prevent statistically reliable estimates for smaller race/ethnicity groups. The data are also tabulated across three income categories: all incomes, below the poverty level, and greater than or equal to the poverty level.

The sensitivity of measurement techniques has improved over the years spanned by Indicator B1, allowing increased detection of lower blood lead levels. These improvements do not affect the comparability of the median or 95th percentiles over time, since between 92 and 100% of children have had detectable levels of lead in each NHANES cycle.

Additional information on how median and 95th percentile blood lead levels vary among different age groups for children ages 1 to 17 years is presented in a supplementary data table. Another data table provides median blood lead levels for the same race/ethnicity and income groups in 1991–1994, for comparison with the more current data presented in Indicator B2.

The indicators focus on ages 1 to 5 years because this age range has been the focus for research, data collection, and intervention due to the elevated exposures that occur during early childhood and the sensitivity of the developing brain to the effects of lead. Blood lead data for school-age children, whose neurological development is also affected by lead exposure, are included in the data tables for this indicator.

Please see the Introduction to the Biomonitoring section for an explanation of the terms "median" and "95th percentile," and information on the statistical significance testing applied to these indicators.

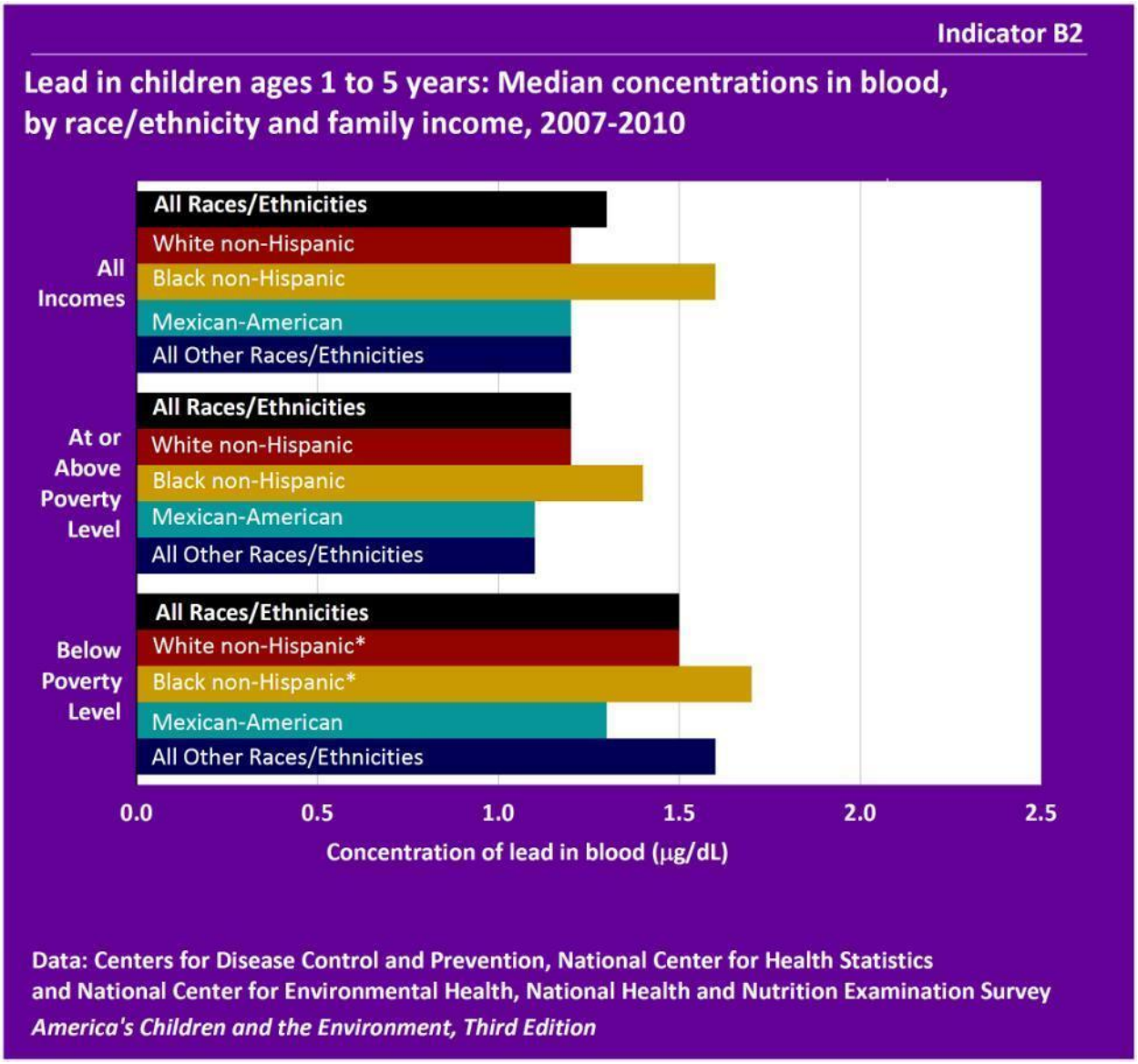


Data characterization

- Data for this indicator are obtained from an ongoing continuous survey conducted by the National Center for Health Statistics.
- Survey data are representative of the U.S. civilian noninstitutionalized population.
- Lead is measured in blood samples obtained from individual survey participants.

- The median concentration of lead in the blood of children between the ages of 1 and 5 years dropped from 15 µg/dL in 1976–1980 to 1.2 µg/dL in 2009–2010, a decrease of 92%.
- The concentration of lead in blood at the 95th percentile in children ages 1 to 5 years dropped from 29 µg/dL in 1976–1980 to 3.4 µg/dL in 2009–2010, a decrease of 88%.

- The largest declines in blood lead levels occurred from the 1970s to the 1990s, following the elimination of lead in gasoline. The data show continuing declines in blood lead levels from 1999–2000 through 2009–2010, when the primary focus of lead reduction efforts has been on lead-based paint in homes.
- These decreasing trends were all statistically significant, including the trend in both the median and 95th percentile over the most recent 12 years (from 1999–2000 to 2009–2010).
- In 2009–2010, median blood lead levels by age group were: 1.2 µg/dL for age 1 year and age 2 years; 1.1 µg/dL for ages 3 to 5 years; 0.8 µg/dL for ages 6 to 10 years; 0.7 µg/dL for ages 11 to 15 years; and 0.7 µg/dL for ages 16 to 17. The 95th percentile blood lead levels were 4.2, 3.5, 2.8, 2.1, 1.7, and 1.4 µg/dL, respectively, for ages 1, 2, 3 to 5, 6 to 10, 11 to 15, and 16 to 17 years. (See Table B1a.)
  - The differences among age groups in median and 95th percentile blood lead levels were statistically significant.



*The estimate should be interpreted with caution because the standard error of the estimate is relatively large: the relative standard error, RSE, is at least 30% but is less than 40% (RSE = standard error divided by the estimate), or the RSE may be underestimated.

Data characterization

- Data for this indicator are obtained from an ongoing continuous survey conducted by the National Center for Health Statistics.
- Survey data are representative of the U.S. civilian noninstitutionalized population.
- Lead is measured in blood samples obtained from individual survey participants.

- The median blood lead level in children ages 1 to 5 years in 2007–2010 was 1.3 µg/dL. The median blood lead level in Black non-Hispanic children ages 1 to 5 years in 2007–2010 was

1.6 µg/dL, higher than the level of 1.2 µg/dL in White non-Hispanic children, Mexican-American children, and children of "All Other Races/Ethnicities."
    - The median blood lead level in Black non-Hispanic children was statistically significantly higher than the median level for each of the remaining race/ethnicity groups.
- The median blood lead level for children living in families with incomes below the poverty level was 1.5 µg/dL, and for children living in families at or above the poverty level it was 1.2 µg/dL, a difference that was statistically significant.
- The 95th percentile blood lead level among all children ages 1 to 5 years was 3.9 µg/dL. The 95th percentile blood lead level in Black non-Hispanic children ages 1 to 5 years in 2007–2010 was 5.8 µg/dL, compared with 3.5 µg/dL for White non-Hispanic children and children of "All Other Races/Ethnicities," and 3.3 µg/dL for Mexican-American children. (See Table B2a.)
- The 95th percentile blood lead level in Black non-Hispanic children was statistically significantly higher than the 95th percentile for each of the remaining race/ethnicity groups.
- Among children ages 1 to 5 years in families with incomes below poverty level, the 95th percentile blood lead was 4.7 µg/dL, and among those in families at or above the poverty level, it was 3.3 µg/dL, a difference that was statistically significant after accounting for differences by age, sex, and race/ethnicity. (See Table B2a.)
- The 95th percentile blood lead levels in children ages 1 to 5 years were higher for those in families with incomes below the poverty level compared with those at or above the poverty level within each race/ethnicity group. Black non-Hispanic children in families with incomes below the poverty level had the highest 95th percentile blood lead level, 6.8 µg/dL, which was 60% higher than for Black non-Hispanic children with families at or above the poverty level. (See Table B2a.)
    - The differences in 95th percentile blood lead levels between income groups were statistically significant for Black non-Hispanic children and children of "All Other Races/Ethnicities." The difference was also statistically significant for Mexican-American children after accounting for differences by age and sex.
- Between 1991–1994 and 2007–2010, median blood lead levels among Black non-Hispanic children ages 1 to 5 years declined 63%: from 4.3 µg/dL to 1.6 µg/dL. Over the same time period, median blood lead levels among Mexican-American children ages 1 to 5 years declined 61%: from 3.1 µg/dL to 1.2 µg/dL, and median blood lead levels among White non-Hispanic children ages 1 to 5 years declined 48%: from 2.3 µg/dL to 1.2 µg/dL. The differences over time were statistically significant for each race/ethnicity. (See Table B2b.)

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Mercury

Mercury is a metal that is liquid at room temperature. There are three major forms of mercury: 1) organic mercury; 2) non-elemental forms of inorganic mercury; and 3) elemental mercury. Organic mercury, predominantly in the form of methylmercury, is found primarily in fish. Non-elemental forms of inorganic mercury are found primarily in batteries, some disinfectants, and some health products and creams. Lastly, elemental mercury is found in thermometers, fluorescent bulbs, dental amalgam fillings, switches in certain automobiles (used for convenience lighting in hoods and trunks, mostly in vehicles manufactured prior to 2003), and other sources.[1,2]

Mercury is released from its natural form in the earth's crust as a result of both human activities and natural processes. Coal-burning power plants are the largest source of mercury emissions in the United States.[3] Other sources of mercury emissions include the combustion of waste and industrial processes that use mercury.[3,4] When released into the atmosphere, either from human activities or from non-human sources, such as volcanoes, mercury can travel long distances on global air currents and can be deposited on land and water far from its original source.[4,5] In addition to these mercury emissions, there is concern that an increase in ice melts caused by a warming climate may release some past mercury emissions that have been trapped in polar ice.[6] Moreover, mercury deposited on the surface in the Arctic vaporizes each spring when the sunlight returns, causing increased concentrations in the atmosphere.[7,8]

Human exposure to elemental and inorganic mercury can occur at work, from accidental mercury releases, through the use of products containing mercury, through ritual and folk medicine uses of mercury, as well as dental restorations with mercury-silver amalgams.[4,9,10] Sources of childhood exposure to elemental and inorganic mercury in the home include the tracking of mercury into the home from the workplace by parents, mercury-containing devices in the home, and very rarely from intentionally heating mercury in the home for the purpose of extracting gold.[11] In schools, the most common sources of exposure are elemental and inorganic mercury stored in science laboratories, and mercury from broken instruments such as thermometers; less common sources are certain mercury-containing gymnasium floors manufactured between 1960 and 1980 found in some schools.[11,12] The adverse health effects of elemental and inorganic mercury exposure in childhood have not been extensively studied. However, inhaling high concentrations of elemental mercury vapor can lead to lung problems, neurobehavioral effects, mood changes, and tremors.[9] Although elemental mercury vapor emissions from dental amalgams are a major source of mercury exposure in the U.S. general population, two prospective clinical trials in children have found no evidence of adverse effects on IQ, memory, attention, or other neurological functions.[13-15]

Thimerosal is an organic mercury-containing preservative that is used in some vaccines to prevent contamination and growth of harmful bacteria in vaccine vials. The presence of thimerosal in many vaccines administered to infants led to concerns about possible effects on

children's neurological development, including a hypothesis that mercury in vaccines could be a contributing factor to the incidence of autism. The Institute of Medicine has rejected the hypothesis of a causal relationship between thimerosal-containing vaccines and autism.[16] In addition, two recent studies have concluded that prenatal and infant exposure to thimerosal-containing vaccines is not related to increased risk of autism.[17,18] Since 2001, thimerosal has not been used in routinely administered childhood vaccines, with the exception of some influenza vaccines.[19]

Methylmercury is another form of organic mercury, which may form when mercury is deposited into water systems such as oceans, rivers, lakes, and wetlands; the mercury is converted by bacteria and other microorganisms into methylmercury. Methylmercury then bioaccumulates up the aquatic food web; fish that live long and feed on other fish (i.e., predatory fish) can accumulate high levels of methylmercury. The concentration of methylmercury in the larger fish at the top of the food chain can reach levels a million times higher than in the water.[20] Consuming fish is the main way that people are exposed to methylmercury. This includes fish commercially distributed in stores and restaurants as well as those that people catch for consumption by their families and communities. Each person's exposure depends on the amount of methylmercury in the fish that they eat and how often they eat fish. These exposure levels are of particular importance for women of child-bearing age because of the potential for prenatal exposure: methylmercury easily crosses the placenta and blood-brain barrier.[15] As such, the prenatal period is considered the most sensitive period of exposure.[15]

EPA has determined that methylmercury is known to have neurotoxic and developmental effects in humans.[4] This determination was based on effects in people prenatally exposed to extremely high levels of methylmercury during accidental mercury poisoning events in Japan and Iraq. Severe adverse health effects observed in the prenatally exposed population included cerebral palsy, intellectual disability (mental retardation), deafness, and blindness.[15,21,22]

Prospective cohort studies have been conducted in island populations where frequent fish consumption leads to methylmercury exposure in pregnant women at levels much lower than in the poisoning incidents but much greater than those typically observed in the United States. These studies are designed to investigate possible associations of prenatal methylmercury exposure with more subtle adverse neurodevelopmental effects than those observed in the poisoning incidents. However, the expected beneficial impacts of prenatal fish consumption on neurodevelopment can make it more difficult to detect such outcomes. Prenatal exposure to mercury in these studies is represented by measurement of total mercury in blood or hair samples obtained from a woman during pregnancy or at delivery. Results from such studies in New Zealand and the Faroe Islands[15,23-28] suggested that increased prenatal mercury exposure due to maternal fish consumption was associated with decrements in attention, language, memory, motor speed, and visual-spatial function (like drawing) during childhood. These associations were not seen in initial results reported from a study in the Seychelles Islands.[29] Further analyses of the Seychelles study population did find associations between prenatal

mercury exposure and some neurodevelopmental deficits, after researchers had accounted for the developmental benefits of fish consumption.[30-32]

More recent studies have been conducted in Massachusetts and New York City, with maternal blood mercury levels within the range of typical levels in the U.S. general population.[33-35] In Massachusetts, total mercury in blood samples collected during the second trimester of pregnancy was associated with reduced cognitive development in testing conducted at age 3 years, after adjusting for the positive effects of fish/seafood consumption during pregnancy.[34] In the New York study, total cord blood mercury was associated with decreased IQ scores in testing conducted at age 4 years, after adjusting for the positive effects of fish/seafood consumption during pregnancy.[33]

Findings of neurodevelopmental effects from early childhood methylmercury exposure are more limited than for prenatal exposure, with several studies reporting mixed findings.[25,36-39] Animal and epidemiological studies suggest that early life exposure to methylmercury (including prenatal exposures) may also affect cardiovascular,[40,41] immune,[15,42,43] and reproductive health.[15]

Although ingestion of methylmercury in fish may be harmful, other compounds naturally present in many fish (such as high quality protein and other essential nutrients) are beneficial. In particular, fish are an excellent source of omega-3 fatty acids, which are nutrients that contribute to the healthy development of infants and children.[44] Pregnant women are advised to seek dietary sources of these fatty acids, including many species of fish. However, the levels of both methylmercury and omega-3 fatty acids can vary considerably by fish species. Thus, the type of fish, as well as portion sizes and frequency of consumption, are all important considerations for health benefits of fish and the extent of methylmercury exposure.

For these reasons, EPA and the U.S. Food and Drug Administration (FDA) issued a fish consumption advisory in 2004 that advises young children and pregnant females to consume up to 12 ounces a week of lower-mercury fish and shellfish, such as shrimp, canned light tuna, salmon, pollock, and catfish, but to avoid any consumption of high-mercury-containing fish, such as shark, swordfish, tile fish, or king mackerel.[45] EPA and FDA are currently working to update the fish consumption advisory to incorporate the most current science regarding the health benefits of fish consumption and the risks from methylmercury in fish. In 2011, the Departments of Agriculture and Health and Human Services jointly released the *2010 Dietary Guidelines for Americans*, which recommended that pregnant or breastfeeding women should consume 8–12 ounces of seafood per week, but avoid consumption of the same high-mercury-containing fish identified in the EPA-FDA advisory.[46] In addition, many state health departments provide advice regarding healthy sources of fish that are lower in mercury. Web links to state advice regarding fish consumption can be found at http://www.epa.gov/waterscience/fish/states.htm (for an example, see Washington state's "Eat Fish, Choose Wisely" available at http://www.doh.wa.gov/ehp/oehas/fish/fishchart.htm).

State advisories may address both store-bought fish and fish caught by individuals in local lakes, rivers, and coastal waters.

Because methylmercury exposure in pregnant women is a concern for children health, studies have measured the level of mercury in women's bodies. Mercury can be measured in blood and is often called "blood mercury." In most cases, total blood mercury is reported, and the measurements do not distinguish methylmercury in blood from the other forms of mercury. In the United States, and in populations where most mercury exposure comes from fish consumption, the majority of total blood mercury is from methylmercury. Among women 16 to 49 years of age in the United States, levels of mercury in blood tend to be highest for Native American, Pacific Islander, Asian American, and multi-racial women.[47-49] A survey of adults in New York City found that blood mercury levels were three times higher than the national levels. Asian Americans in this study had higher blood mercury levels than other race/ethnicity groups.[50] Among women ages 16 to 49 years in the United States, blood mercury levels are higher for those who eat fish more often or in higher quantities.[51,47] Asian American populations have been identified as high consumers of seafood compared with White non-Hispanics or Black non-Hispanics.[50]

For women of all races, blood mercury levels tend to be higher in those women with higher family incomes.[48,50,52] Fish consumption rates are highest among women with relatively high family incomes, and this higher rate of fish consumption leads to increased blood mercury levels.[48,52] Concentrations of total mercury in blood among women also seem to vary with geographic region, and potentially by coastal region. Based on data from 1999–2004, blood mercury levels for women ages 16 to 49 years were higher in the Northeastern region of the United States compared with other regions.[48] Estimated mercury intake from fish consumption also follows this observed pattern. Women living in coastal regions had blood mercury levels higher than those living in noncoastal regions, and among coastal populations, the highest blood mercury levels were reported for the Atlantic and Pacific coastal regions, followed by the Gulf Coast and Great Lakes regions, respectively. Furthermore, subsistence populations (individuals who sustain a portion of their diets by catching and eating fish from local waters), or those who consume fish as a large portion of their diet because of taste preference or in the pursuit of health benefits, may have elevated blood mercury levels, depending on the source and species of fish.[4]

The indicator that follows uses the best nationally representative data currently available on blood mercury levels over time for women of child-bearing age. Indicator B3 presents median and 95th percentile blood mercury levels for women ages 16 to 49 years.

Indicator B3: Mercury in women ages 16 to 49 years: Median and 95th percentile concentrations in blood, 1999–2010

About the Indicator: Indicator B3 presents concentrations of mercury in blood of U.S. women ages 16 to 49 years. The data are from a national survey that collects blood specimens from a representative sample of the population every two years, and then measures the concentration of mercury in the blood. The indicator presents concentrations of mercury in blood over time. The focus on women of child-bearing age is based on concern for potential adverse effects in children born to women who have been exposed to mercury.

NHANES

The National Health and Nutrition Examination Survey (NHANES) provides nationally representative biomonitoring data for mercury. NHANES is designed to assess the health and nutritional status of the civilian noninstitutionalized U.S. population and is conducted by the National Center for Health Statistics, part of the Centers for Disease Control and Prevention (CDC). Interviews and physical examinations are conducted with approximately 10,000 people in each two-year survey cycle. CDC's National Center for Environmental Health measures concentrations of environmental chemicals in blood and urine samples collected from NHANES participants. Summaries of the measured values for more than 200 chemicals are provided in the *Fourth National Report on Human Exposure to Environmental Chemicals*.[53]

Mercury

Indicator B3 presents levels of mercury in blood of women of child-bearing age. Organic, inorganic, and total mercury can be measured in blood.[i] The concentration of total mercury in blood is a marker of exposure to methylmercury in populations where fish consumption is the predominant source of mercury exposure. Previous analysis shows that, in general, methylmercury accounts for a large percentage of total mercury in blood among women of child-bearing age in the United States.[47] Total blood mercury is generally representative of methylmercury exposures in the past few months.[54,55] All values are reported as micrograms of mercury per liter of blood (µg/L).

Concentrations of total blood mercury have been measured in all NHANES participants ages 1 to 5 years and all female participants ages 16 to 49 years beginning with the 1999–2000 survey cycle. Starting with the 2003–2004 survey cycle, NHANES measured blood mercury in all participants ages 1 year and older.[56] Separate measurements of inorganic blood mercury have been reported starting with the 2003–2004 NHANES survey cycle.

[i] NHANES also measures mercury levels in participant's urine samples, which is considered a more robust determinant of body burden of mercury from long-term exposure, particularly for inorganic mercury.

For 2009–2010, NHANES collected mercury biomonitoring data for 8,793 individuals ages 1 year and older, including 1,871 women ages 16 to 49 years. Mercury was detected in 81% of all individuals sampled. The frequency of mercury detection was 83% in women ages 16 to 49 years.[ii] The median blood mercury level among all NHANES participants in 2009–2010 was 0.8 µg/L and the 95th percentile was 5.1 µg/L.

Birth Rate Adjustment

Indicator B3 uses measurements of mercury in blood of women ages 16 to 49 years to represent the distribution of mercury exposures to women who are pregnant or may become pregnant. However, blood mercury levels increase with age,[56] and women of different ages have a different likelihood of giving birth. For example, in 2003–2004, women aged 27 years had a 12% annual probability of giving birth, and women aged 37 years had a 4% annual probability of giving birth.[57] A birth rate-adjusted distribution of women's mercury levels is used in calculating this indicator,[iii] meaning that the data are weighted using the age-specific probability of a woman giving birth.[58]

Data Presented in the Indicators

Indicator B3 presents median and 95th percentile concentrations of mercury in blood over time for women ages 16 to 49 years, using NHANES data from 1999–2010.

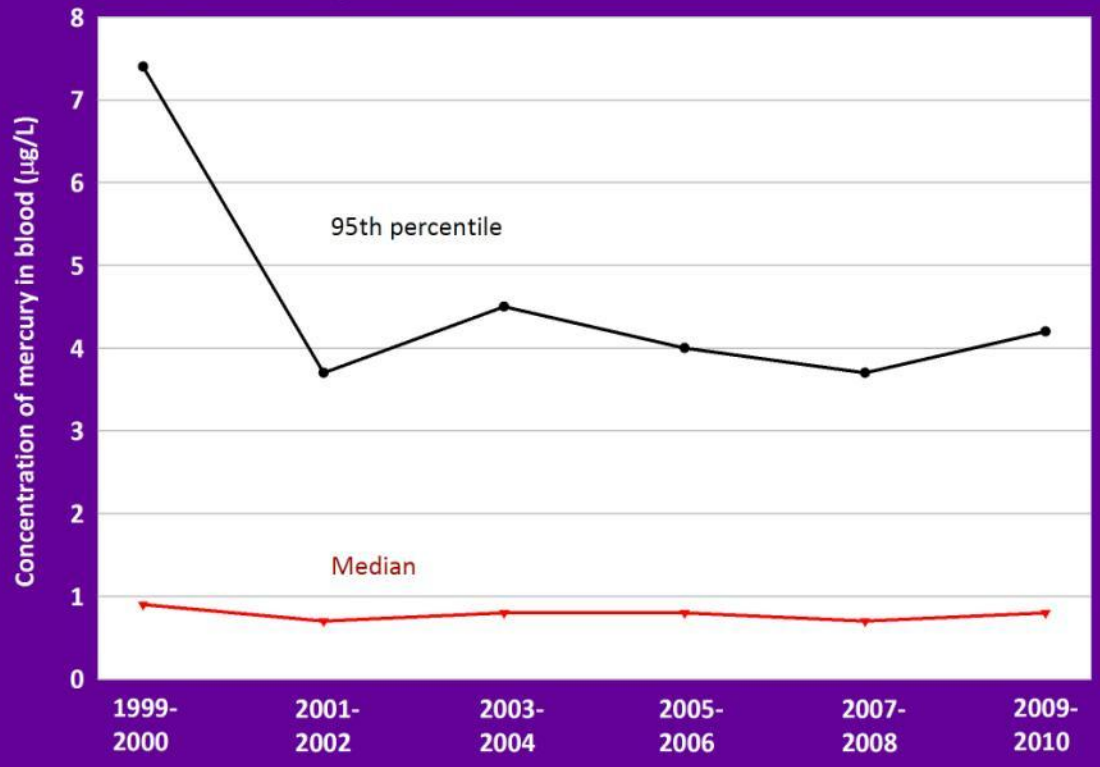
Additional information showing how median and 95th percentile blood mercury levels vary by race/ethnicity and family income for women ages 16 to 49 years is presented in supplemental data tables for these indicators. Data tables also display the median and 95th percentile blood mercury levels for children ages 1 to 5 years over time and the median and 95th percentile blood mercury levels for children ages 1 to 17 years for 2007–2010.

Please see the Introduction to the Biomonitoring section for an explanation of the terms "median" and "95th percentile," a description of the race/ethnicity and income groups used in the ACE3 biomonitoring indicators, and information on the statistical significance testing applied to these indicators.

[ii] The percentage for women ages 16 to 49 years is calculated with the birth rate adjustment described below.
[iii] There may be multiple ways to implement an adjustment to the data that accounts for birth rates by age. The National Center for Health Statistics has not fully evaluated the method used in ACE, or any other method intended to accomplish the same purpose, and has not used any such method in its publications. NCHS and EPA are working together to further evaluate the birth rate adjustment method used in ACE and alternative methods.

Indicator B3

Mercury in women ages 16 to 49 years: Median and 95th percentile concentrations in blood, 1999-2010



Data: Centers for Disease Control and Prevention, National Center for Health Statistics and National Center for Environmental Health, National Health and Nutrition Examination Survey

Note: To reflect exposures to women who are pregnant or may become pregnant, the estimates are adjusted for the probability (by age and race/ethnicity) that a woman gives birth.

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Data characterization

- Data for this indicator are obtained from an ongoing continuous survey conducted by the National Center for Health Statistics.
- Survey data are representative of the U.S. civilian noninstitutionalized population.
- Mercury is measured in blood samples obtained from individual survey participants.

- The median concentration of total mercury in the blood of women ages 16 to 49 years has shown little change between 1999–2000 and 2009–2010, and was 0.8 µg/L in 2009–2010.
- Among women in the 95th percentile of exposure, the concentration of total mercury in blood decreased from 7.4 µg/L in 1999–2000 to 3.7 µg/L in 2001-2002. From 2001–2002 to 2009–2010, the 95th percentile of total blood mercury remained between 3.7 and 4.5 µg/L.

  - The decrease in the 95th percentile levels of blood mercury between 1999–2000 and 2001–2002 was statistically significant. From 2001-2002 to 2009-2010 there was no statistically significant change.
- In 1999–2000, the 95th percentile total mercury level was 8 times the median level. For the remaining years, the 95th percentile total mercury levels were about 5 times the median levels.

- For the years 2007–2010, women of "All Other Races/Ethnicities" had median blood mercury levels of 1.3 µg/L, compared with median mercury levels for the remaining race/ethnicity groups of 0.6–0.8 µg/L. (See Table B3a.)
  - The median blood mercury level in women of "All Other Races/Ethnicities" was statistically significantly higher than the median level for each of the remaining race/ethnicity groups.
- Among women in the 95th percentile of exposure, differences in total mercury in blood were observed across race/ethnicity groups. For the years 2007–2010, White non-Hispanic women had a blood mercury level of 3.7 µg/L, Black non-Hispanics had 2.9 µg/L, Mexican-American women had 2.3 µg/L, and women in the "All Other Races/Ethnicities" group had 6.7 µg/L. (See Table B3b.)
  - The differences between race/ethnicity groups were statistically significant after accounting for differences by income level and age.
- Among women in the 95th percentile of exposure, women living at or above the poverty level had higher blood levels of total mercury (4.0 µg/L) compared with women living below poverty level (2.9 µg/L), a difference that was statistically significant. (See Table B3b.)
- The median and 95th percentile values for women of child-bearing age were about 2 to 4 times those of children ages 1 to 5 years. (See Table B3 and Table B3c.)
- Among children ages 1 to 5 years in the 95th percentile of exposure, the concentration of total mercury in blood showed a decreasing trend from 2.3 µg/L in 1999–2000 to 1.3 µg/L in 2009–2010. The median blood mercury level for children ages 1 to 5 years stayed relatively constant for the same time period. (See Table B3c.)
  - The decreasing trend in 95th percentile blood mercury levels in children was statistically significant. There was no statistically significant change in median blood mercury levels in children.
- Among children ages 1 to 17 years, median and 95th percentile blood mercury levels generally increased with age in 2007–2010, with higher blood mercury levels among children ages 6 years and older. Children ages 16 to 17 years had a median level of mercury in blood of 0.5 µg/L and a 95th percentile of 2.8 µg/L. (See Table B3d.)
  - The differences by age group were statistically significant at both the median and the 95th percentile.

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Cotinine

Environmental tobacco smoke (ETS), commonly referred to as secondhand smoke, is a complex mixture of gases and particles and includes smoke from burning cigarettes, cigars, and pipe tobacco (sidestream smoke), as well as exhaled mainstream smoke.[1] There are at least 250 chemicals in ETS that are known to be toxic or carcinogenic, including acrolein, ammonia, benzene, carbon monoxide, formaldehyde, hydrogen cyanide, nicotine, nitrogen oxides, and sulfur dioxide.[1,2] In 1992, EPA classified ETS as a known human carcinogen.[3] Children can be exposed to ETS in their homes or in places where people are allowed to smoke, such as some restaurants in some locations throughout the United States.

According to the U.S. Surgeon General, there is no safe level of exposure to ETS, and breathing even a small amount can be harmful to human health.[1] The Surgeon General has concluded that exposure to ETS causes sudden infant death syndrome (SIDS), acute lower respiratory infection, ear problems, and more severe asthma in children. Smoking by parents causes respiratory symptoms and slows lung growth in their children.[1] Young children appear to be more susceptible to the respiratory effects of ETS than are older children.[3-5] It is also possible that early-life exposures to ETS may lead to adverse health effects in adulthood. Exposure to ETS in childhood has been reported to be associated with early emphysema in adulthood among nonsmokers.[6]

The exposure of a pregnant woman to ETS can also be harmful to her developing fetus. The Surgeon General has determined that exposure of pregnant women to ETS causes a small reduction in mean birth weight and the evidence is suggestive (but not sufficient to infer causation) of a relationship between maternal exposure to environmental tobacco smoke during pregnancy and preterm delivery.[1] In addition, the Surgeon General concluded the evidence is suggestive but not sufficient to infer a causal relationship between prenatal and postnatal exposure to ETS and childhood cancer.[1]

Exposure to ETS in the home is influenced by adult behaviors, including the decisions to smoke at home and to allow visitors to smoke inside the home. Children living in homes with smoking bans have significantly lower levels of cotinine (a biological marker of exposure to ETS) in urine than children living in homes without smoking bans.[7] Household smoking bans can significantly decrease children's exposures to ETS, but do not completely eliminate them.[8]

In recent years there has been a significant decline in children's exposures to ETS.[9] This reduction is in part attributable to a decline in the percentage of adults who smoke. In 2010, an estimated 19.3% of adults were current smokers, down from 24.7% in 1997.[10,11] In addition, the prevalence of smoke-free households increased from 43% of U.S. homes in 1992–1993 to 72% in 2003.[12] However, despite the increasing numbers of adults disallowing smoking in the home, approximately 34% of children live in a home with at least one smoker as of 2009.[13] The enactment of smoking bans in restaurants, bars, and other public places has led to a decrease in ETS exposure for both children and adults.[14] Recent studies suggest that smoking bans can

reduce the number of asthma-related emergency room visits and hospitalizations and reduce asthmatic symptoms, including persistent wheeze, wheeze-medication use, and chronic night cough in children.[15-18]

Cotinine is considered the best biomarker of exposure to tobacco smoke for both active smokers and those exposed to ETS.[19] The two indicators that follow use the best nationally representative data currently available on blood cotinine levels over time for women of child-bearing age and children. Indicator B4 presents median and 95th percentile blood serum levels of cotinine for children ages 3 to 17 years. Indicator B5 presents median and 95th percentile blood serum levels of cotinine for women ages 16 to 49 years.

Indicator B4: Cotinine in nonsmoking children ages 3 to 17 years: Median and 95th percentile concentrations in blood serum, 1988–2010

Indicator B5: Cotinine in nonsmoking women ages 16 to 49 years: Median and 95th percentile concentrations in blood serum, 1988–2010

About the Indicators: Indicators B4 and B5 present concentrations of cotinine in blood serum of U.S. children ages 3 to 17 years and women ages 16 to 49 years. Cotinine is a marker of exposure to environmental tobacco smoke (ETS). The data are from a national survey that collects blood specimens from a representative sample of the population every two years, and then measures the concentration of cotinine in the blood serum. Indicator B4 presents concentrations of cotinine in children's blood serum over time and Indicator B5 presents concentrations of cotinine in women's blood serum over time. The focus on both children and women of child-bearing age is based on concern for potential adverse effects in children exposed to ETS and in children born to women who have been exposed to ETS.

NHANES

The National Health and Nutrition Examination Survey (NHANES) provides nationally representative biomonitoring data for cotinine. NHANES is designed to assess the health and nutritional status of the civilian noninstitutionalized U.S. population and is conducted by the National Center for Health Statistics, part of the Centers for Disease Control and Prevention (CDC). Interviews and physical examinations are conducted with approximately 10,000 people in each two-year survey cycle. CDC's National Center for Environmental Health measures concentrations of environmental chemicals in blood and urine samples collected from NHANES participants. Summaries of the measured values for more than 200 chemicals are provided in the *Fourth National Report on Human Exposure to Environmental Chemicals*.[19]

Environmental Tobacco Smoke (ETS) and Cotinine

Indicators B4 and B5 present blood serum levels of cotinine as a marker of exposure to ETS. Nicotine is a distinctive component of tobacco that is found in large amounts in tobacco smoke, including ETS. Once nicotine enters the body, it is rapidly broken down in a matter of a few hours into other chemicals. Cotinine is a primary breakdown product of nicotine, and has a longer half-life. This characteristic makes cotinine a better indicator than nicotine of an individual's exposure to ETS.[20-22]

Measurement of cotinine in blood serum is a marker for exposure to ETS in the previous few days.[23] Some studies have shown that, given the same exposure to tobacco smoke, cotinine levels may differ by race/ethnicity and sex, and there may be genetic differences in the rate at which cotinine is removed from the body.[1,24-28]

These indicators present cotinine levels for non-tobacco-users only. Children and women who were active smokers, as indicated by a relatively high serum cotinine level, were excluded from these statistics. For these analyses, individuals with a serum cotinine level greater than 10 nanograms of cotinine per milliliter of serum (ng/mL) are considered active smokers, and all individuals with cotinine levels below 10 ng/mL are considered nonsmokers.[19] Active smokers will almost always have serum cotinine levels above 10 ng/mL, and sometimes those levels will be higher than 500 ng/mL.[19,29] Nonsmokers who are exposed to typical levels of ETS have serum cotinine levels of less than 1 ng/mL, whereas those nonsmokers with heavy exposure to ETS will have serum cotinine levels between 1 and 10 ng/mL.[19]

Concentrations of cotinine in blood serum have been measured in NHANES participants ages 4 years and older for the 1988–1991 and 1991–1994 survey cycles, and then for ages 3 years and older beginning with the 1999–2000 survey cycle.

For 2009–2010, NHANES collected cotinine biomonitoring data for 6,678 nonsmoking individuals ages 3 years and older, including 2,191 children ages 3 to 17 years and 1,395 women ages 16 to 49 years. Cotinine was detected in about 67% of all nonsmoking individuals sampled. The frequency of cotinine detection was 71% in children ages 3 to 17 years and 66% in women ages 16 to 49 years.[i] The median blood serum cotinine level for all nonsmoking NHANES participants in 2009–2010 was 0.03 ng/mL and the 95th percentile was 1.3 ng/mL.

Birth Rate Adjustment

Indicator B5 uses measurements of cotinine in blood serum of women ages 16 to 49 years to represent the distribution of ETS exposures to women who are pregnant or may become pregnant. For example, in 2003–2004, women aged 27 years had a 12% annual probability of giving birth, and women aged 37 years had a 4% annual probability of giving birth.[30] A birth rate-adjusted distribution of women's cotinine levels is used in calculating this indicator,[ii] meaning that the data are weighted using the age-specific probability of a woman giving birth.[31]

Data Presented in the Indicators

Indicator B4 presents median and 95th percentile concentrations of cotinine in blood serum over time as a marker of exposure to ETS among non-smoking children ages 3 to 17 years, using NHANES data from 1988–2010.

[i] The percentage for women ages 16 to 49 years is calculated with the birth rate adjustment described below.
[ii] There may be multiple ways to implement an adjustment to the data that accounts for birth rates by age. The National Center for Health Statistics has not fully evaluated the method used in ACE, or any other method intended to accomplish the same purpose, and has not used any such method in its publications. NCHS and EPA are working together to further evaluate the birth rate adjustment method used in ACE and alternative methods.

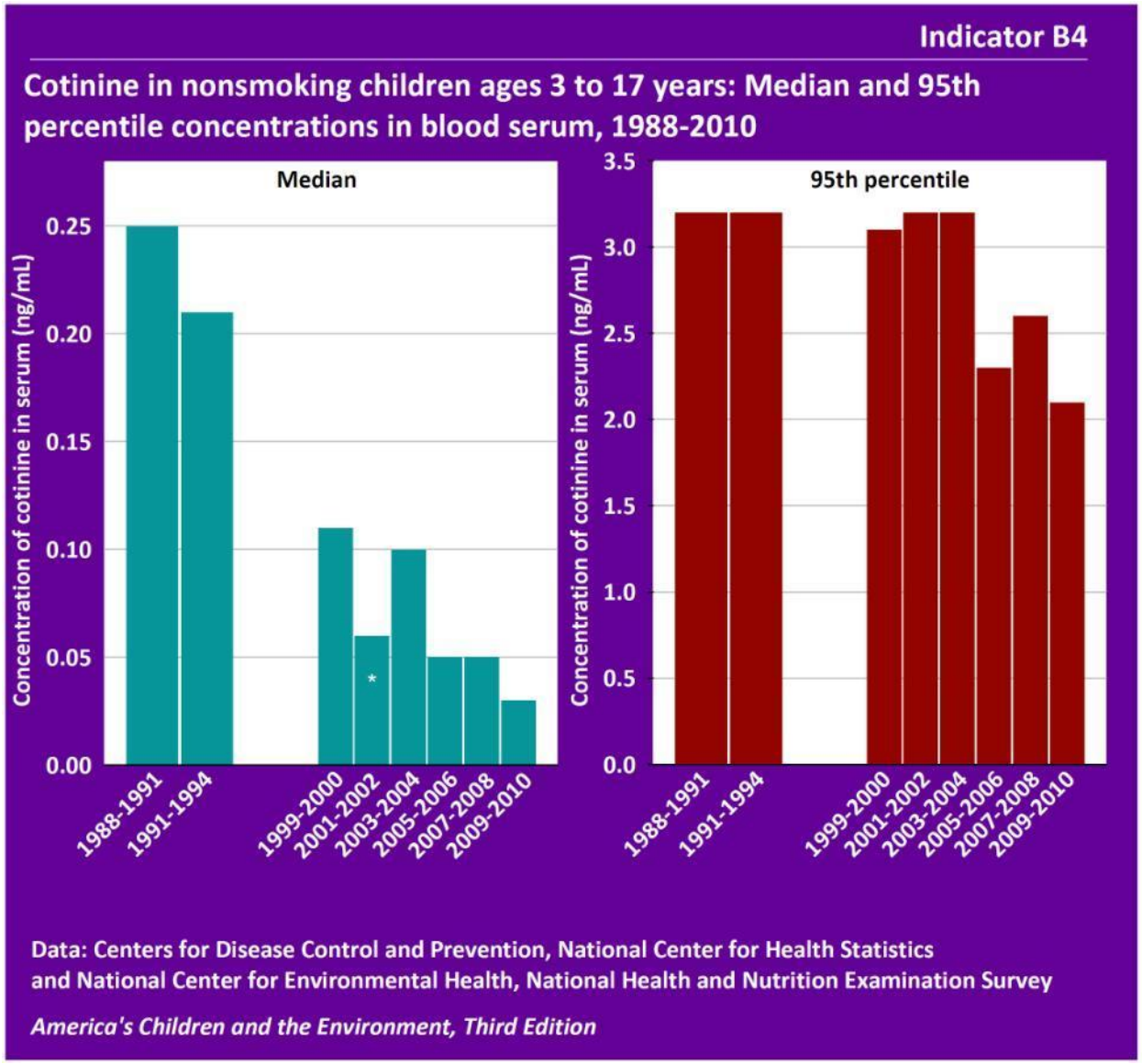
Indicator B5 presents median and 95th percentile concentrations of cotinine in blood serum over time as a marker of exposure to ETS among non-smoking women ages 16 to 49 years, using NHANES data from 1988–2010.

Although the sensitivity of measurement techniques has improved over the years spanned by Indicators B4 and B5, allowing increased detection of lower serum cotinine levels over time, these improvements do not affect the comparability of the median or 95th percentiles over time since the majority of children and women have had detectable levels of cotinine in each NHANES cycle.

Additional information showing how median and 95th percentile blood serum levels of cotinine vary by race/ethnicity, family income, and age for children ages 3 to 17 years is presented in the supplemental data tables for these indicators. Data tables also show how median and 95th percentile blood serum levels of cotinine vary by race/ethnicity and family income for women ages 16 to 49 years.

NHANES does not provide cotinine measurements for children under the age of 3 years (or under age 4 years prior to 1999), who may be especially sensitive to the effects of ETS exposure.

Please see the Introduction to the Biomonitoring section for an explanation of the terms "median" and "95th percentile," a description of the race/ethnicity and income groups used in the ACE3 biomonitoring indicators, and information on the statistical significance testing applied to these indicators.



*The estimate should be interpreted with caution because the standard error of the estimate is relatively large: the relative standard error, RSE, is at least 30% but is less than 40% (RSE = standard error divided by the estimate), or the RSE may be underestimated.

Data characterization

- Data for this indicator are obtained from an ongoing continuous survey conducted by the National Center for Health Statistics.
- Survey data are representative of the U.S. civilian noninstitutionalized population.
- Cotinine is measured in blood samples obtained from individual survey participants.

- The median level of cotinine measured in blood serum of nonsmoking children ages 3 to 17 years dropped from 0.25 ng/mL in 1988–1991 to 0.03 ng/mL in 2009–2010, a decrease of 88%. This decreasing trend was statistically significant.

- Cotinine values at the 95th percentile decreased by 34% from 1988–1991 to 2009–2010. This trend was also statistically significant.

- Children at the 95th percentile of cotinine levels had much higher levels than those at the median. In 1988–1991, the 95th percentile cotinine level (3.2 ng/mL) was 13 times the median level (0.25 ng/mL); in 2009–2010, the 95th percentile cotinine level (2.1 ng/mL) was 70 times the median level (0.03 ng/mL).

- In every time period measured, children at the 95th percentile had higher levels of cotinine in their blood than women at corresponding levels. (Compare with Indicator B5.)

- Eighty-seven percent of nonsmoking children ages 4 to 17 years had detectable levels (at or above 0.05 ng/mL) of cotinine in 1988–1991. Forty percent of nonsmoking children ages 3 to 17 years had levels at or above 0.05 ng/mL of cotinine in 2009–2010, although improvements in laboratory methods made it possible to detect cotinine at lower concentrations starting with the 2001–2002 survey cycle. (Data not shown.)

- In 2007–2010, median concentrations of cotinine in blood for nonsmokers were approximately 0.11 ng/mL for Black non-Hispanic children, 0.04 ng/mL for White non-Hispanic children, and 0.02 ng/mL for Mexican-American children. The differences between these race/ethnicity groups were statistically significant. (See Table B4a.)

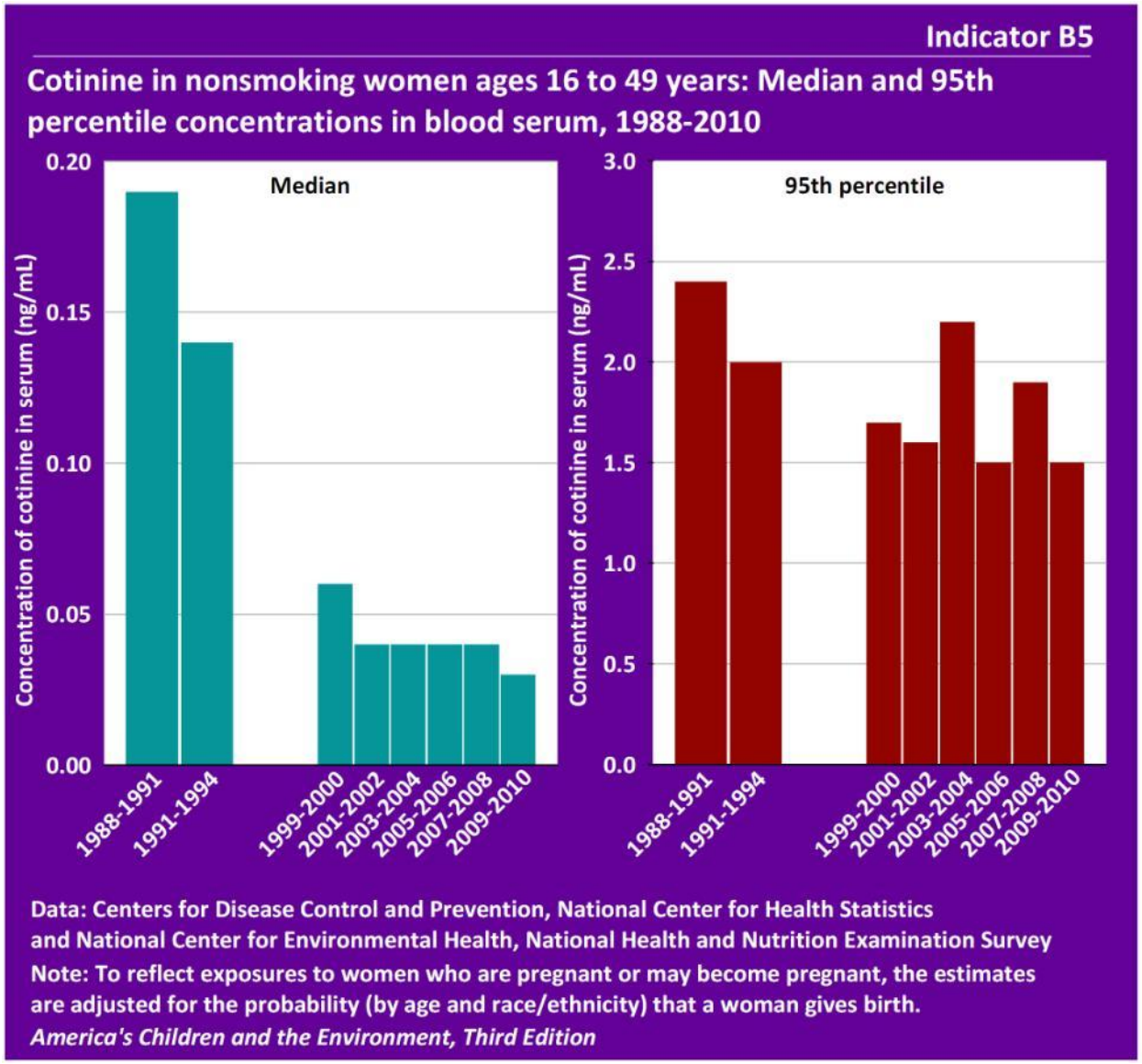
- In 2007–2010, the median concentration of cotinine in blood serum for nonsmoking children living below the poverty level (0.14 ng/mL) was about 5 times the median for nonsmoking children living at or above the poverty level (0.03 ng/mL). The differences between income groups were statistically significant. (See Table B4a.)

- In 2007–2010, 95th percentile concentrations of cotinine in blood for nonsmokers were 2.9 ng/mL for White non-Hispanic children and 2.6 ng/mL for Black non-Hispanic children, while Mexican-American children had levels that were more than 3 times lower (0.8 ng/mL). (See Table B4b.)

  - The differences between levels for Mexican-American children and both White non-Hispanic and Black non-Hispanic children were statistically significant.

- For the years 2007–2010, median levels of cotinine in younger nonsmoking children ages 3 to 5 years and 6 to 10 years were 0.06 and 0.05 ng/mL, respectively, compared with 0.03 and 0.04 ng/mL in older nonsmoking children ages 11 to 15 years and 16 to 17 years, respectively. (See Table B4c.)

  - The differences between the levels for the four age groups were not statistically significant.



Data characterization

- Data for this indicator are obtained from an ongoing continuous survey conducted by the National Center for Health Statistics.
- Survey data are representative of the U.S. civilian noninstitutionalized population.
- Cotinine is measured in blood samples obtained from individual survey participants.

- The median level of cotinine measured in blood serum of nonsmoking women of child-bearing age dropped from 3.2 ng/mL in 1988–1991 to 2.1 ng/mL in 2009–2010, a decrease of 86%. This decreasing trend was statistically significant.
- Cotinine values at the 95th percentile decreased by 35% from 1988–1991 to 2009–2010. This trend was also statistically significant.

- Women at the 95th percentile cotinine levels had much higher levels than those at the median. In 1988–1991, the 95th percentile cotinine level (2.3 ng/mL) was 11 times the median level (0.21 ng/mL); in 2009–2010, the 95th percentile cotinine level (1.5 ng/mL) was 50 times the median level (0.03 ng/mL).
- In 2007–2010, median concentrations of cotinine in blood for nonsmoking women were approximately 0.1 ng/mL for Black non-Hispanic women and 0.03 ng/mL for White non-Hispanic women and Mexican-American women.
  - The difference between Black non-Hispanic women and Mexican-American women was statistically significant. The difference between Black non-Hispanic and White non-Hispanic women was not statistically significant after adjusting for age and income differences. (See Table B5a.)
- Cotinine values at the 95th percentile were more than twice as high for nonsmoking women living below the poverty level (3.5 ng/mL) as for nonsmoking women living at or above the poverty level (1.4 ng/mL) in 2007–2010. The differences between income groups were statistically significant. (See Table B5b.)

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Perfluorochemicals (PFCs)

Perfluorochemicals (PFCs) are a group of synthetic chemicals that have been used in many consumer products.[1] The structure of these chemicals makes them very stable, hydrophobic (water-repelling), and oleophobic (oil-repelling). These unique properties have led to extensive use of PFCs in surface coating and protectant formulations for paper and cardboard packaging products; carpets; leather products; and textiles that repel water, grease, and soil. PFCs have also been used in fire-fighting foams and in the production of nonstick coatings on cookware and some waterproof clothes.[1] Due in part to their chemical properties, some PFCs can remain in the environment and bioconcentrate in animals.[2-8] Data from human studies suggest that some PFCs can take years to be cleared from the body.[9-13]

The PFCs with the highest production volumes in the United States have been perfluorooctane sulfonic acid (PFOS) and perfluorooctanoic acid (PFOA).[1] PFOS and PFOA are also two of the most frequently detected PFCs in humans.[14] Other PFCs include perfluorohexane sulfonic acid (PFHxS), which is a member of the same chemical category as PFOS; and perfluorononanoic acid (PFNA), which is a member of the same chemical category as PFOA.[15] Chemicals within a given PFC chemical category share similar chemical structures and uses. Although some studies have addressed PFHxS and PFNA specifically, the majority of scientific research has focused on PFOS and PFOA.[15]

In 2000, one of the principal perfluorochemical manufacturers, 3M, began phasing out the production of PFOA, PFOS, and PFOS-related compounds. The 3M phaseout of PFOS and PFHxS was completed in 2002, and its phaseout of PFOA was completed in 2008.[16] In 2006, to address PFOA production by other manufacturers, EPA launched the 2010/15 PFOA Stewardship Program, with eight companies voluntarily agreeing to reduce emissions and product content of PFOA, PFNA, and related chemicals by 95% no later than 2010. The industry participants also committed to work toward eliminating emissions and product content of these chemicals by 2015.[17] However, the fact that some of these chemicals may be persistent in the environment and have a long half-life in humans means that they may continue to persist in the environment and in people for many years, despite reductions in emissions.[2-13] EPA is currently evaluating the potential need for regulation of PFCs using the authorities of the Toxic Substances Control Act.[15]

The major sources of human exposure to PFCs are poorly understood, but may include food, water, indoor and outdoor air, breast milk, and dust.[4] Two recent studies pointed to food consumption as the primary pathway of exposure to PFOS and PFOA for Americans and Europeans.[18,19] PFC-treated food-contact packaging, such as microwave popcorn bags,[i] has

[i] The U.S. Food and Drug Administration recently worked with several manufacturers to remove grease-proofing agents containing C8 perfluorinated compounds from the marketplace. These manufacturers volunteered to stop distributing products containing these compounds in interstate commerce for food-contact purposes as of October

been a source of PFC exposure.[20] Meat and dairy products may also be contaminated with PFCs due to exposure of source animals to air, water, and feed contaminated with PFCs,[21-23] although a recent study reported that PFCs were undetected in nearly all milk samples tested in the United States.[24] In some areas, such as those near industrial facilities that either make or use PFCs, these contaminants have been found at high levels in drinking water, groundwater, and/or surface water.[25-31] PFCs have been detected in human breast milk.[32-36] PFCs have been measured in house dust as well, with some perfluorochemicals, such as PFOS, PFOA, PFHxS, perfluorobutane sulfonic acid (PFBuS), and perfluorohexanoic acid (PFHxA), found to be present in the majority of dust samples examined.[37-40] Infants and small children may be more highly exposed to the PFCs present in house dust than adults are, due to their frequent and extensive contact with floors, carpets, and other surfaces where dust gathers, as well as their frequent hand-to-mouth activity.[18,19,41,42] Children could have increased exposure to PFCs in carpet and carpet protectants, due to the amount of time they spend lying, crawling, and playing on carpet.[15,41] Limited available data on levels of PFCs in children's blood suggest that the blood serum levels of most PFCs are higher in children ages 3 to 11 years compared with other age groups.[43,44]

Some PFCs have been widely detected in pregnant women and in umbilical cord blood, suggesting that the developing fetus can be exposed to PFCs while in the womb. However, findings between studies vary. For example, PFOS and PFOA were detected in 99–100% of blood samples collected from both pregnant and non-pregnant women in 2003–2004.[45] Additionally, PFOS and PFOA were detected in 99% and 100% of umbilical cord blood samples, respectively, collected from newborns in Baltimore.[46] In another study conducted in Japan, the level of PFOS circulating in a pregnant woman's blood was highly correlated with the level in cord blood. However, PFOA was detected in maternal samples but was not detected in umbilical cord samples in the Japanese study.[47] Even though studies suggest that the correlation between maternal and fetal exposure may vary, the ubiquitous presence of PFOS, PFOA, and other PFCs in blood of women of child-bearing age and in umbilical cord blood may indicate that fetal exposure to these chemicals is widespread.[45,46,48]

Some human health studies have found associations between prenatal exposure to PFOS or PFOA and a range of adverse birth outcomes, such as low birth weight, decreased head circumference, reduced birth length, and smaller abdominal circumference.[49-52] However, there are inconsistencies in the results of these studies, and two other studies did not find an association between prenatal PFC exposure and birth weight.[53,54] The participants in all of these studies had PFC blood serum levels comparable to levels in the general population. Animal studies echo these findings, though typically at levels much higher than what humans are normally exposed to. Developmental and reproductive effects, including reduced birth weight, decreased gestational length, structural defects, delays in postnatal growth and development,

1, 2011. For more information, see http://www.fda.gov/Food/FoodIngredientsPackaging/FoodContactSubstancesFCS/ucm308462.htm.

increased neonatal mortality, and pregnancy loss have all been associated with prenatal rodent exposure to PFOS and PFOA.[55-65]

Findings from a limited number of studies suggest that exposure to PFOS or PFOA may have negative impacts on human thyroid function. However, there are inconsistencies in the findings between these studies. Some studies have found that PFC exposures are associated with alterations in thyroid hormone levels, as well as an increased risk of thyroid disease in the general public and in workers with occupational exposures.[66-68] However, a recent study of pregnant women with exposures comparable to those in the general population found that increasing levels of PFOS, PFOA, and PFHxS were not associated with differences in thyroid hormone levels.[69] The results from animal studies have been more consistent. Multiple animal studies have found that thyroid hormone levels are altered in animals exposed to PFOS.[57,62,63,65,70-74] One of these studies also found that PFOA-treated rats have altered thyroid hormone levels.[71] The health risks associated with maternal thyroid hormone disruption during pregnancy may make this a cause for concern. Moderate deficits in maternal thyroid hormone levels during early pregnancy have been linked to reduced childhood IQ scores and other neurodevelopmental effects, as well as unsuccessful or complicated pregnancies.[75]

Both animal and some human studies have found an association between PFCs exposure and cholesterol and/or triglyceride levels, although physiological differences between humans and experimental animals may cause lipid levels to vary in opposite directions.[76] Structurally, PFCs resemble fatty acids and can bind to receptors that play key roles in lipid metabolism and fat production.[77] In animal studies involving various species, PFCs are associated with decreased serum levels of these lipids;[64,65,73] in contrast some human studies show an increase in blood lipid levels with increased presence of PFCs, including PFOS, PFOA, PFHxS, and PFNA, while other human studies show no change in lipid levels with PFC exposure.[77-84] This could be a potential concern for children, because the mother's body provides a source of cholesterol and triglycerides to the developing fetus. Cholesterol and fatty acids support cellular growth, differentiation, and adipose accumulation during fetal development.[49,85] Finally, although human studies have not looked at the associations between PFC exposure and the immune system, animal studies have found an association between PFOS and PFNA exposure (in utero and in adulthood) and immune suppression, including alterations in function and production of immune cells and decreased lymphoid organ weights.[86-88]

The indicator that follows uses the best nationally representative data currently available on blood serum levels of perfluorochemicals over time for women of child-bearing age. Indicator B6 presents median blood serum levels of PFOS, PFOA, PFHxS, and PFNA for women ages 16 to 49 years.

Indicator B6: Perfluorochemicals in women ages 16 to 49 years: Median concentrations in blood serum, 1999–2008

About the Indicator: Indicator B6 presents concentrations of perfluorochemicals (PFCs) in blood serum of U.S. women ages 16 to 49 years. The data are from a national survey that collects blood specimens from a representative sample of the population every two years, and then measures the concentration of PFCs in the blood serum. The indicator presents concentrations of PFCs in blood serum over time. The focus on women of child-bearing age is based on concern for potential adverse effects in children born to women who have been exposed to PFCs.

NHANES

The National Health and Nutrition Examination Survey (NHANES) provides nationally representative biomonitoring data for PFCs. NHANES is designed to assess the health and nutritional status of the civilian noninstitutionalized U.S. population and is conducted by the National Center for Health Statistics, part of the Centers for Disease Control and Prevention (CDC). Interviews and physical examinations are conducted with approximately 10,000 people in each two-year survey cycle. CDC's National Center for Environmental Health measures concentrations of environmental chemicals in blood and urine samples collected from NHANES participants. Summaries of the measured values for more than 200 chemicals are provided in the *Fourth National Report on Human Exposure to Environmental Chemicals*.[2]

Perfluorinated Compounds

Indicator B6 presents blood serum levels of four important PFCs: perfluorooctane sulfonic acid (PFOS), perfluorooctanoic acid (PFOA), perfluorohexane sulfonic acid (PFHxS), and perfluorononanoic acid (PFNA). These four PFCs were chosen because they are commonly detected in humans, and the bulk of PFCs health effects research in both humans and laboratory animals has focused on these contaminants—especially PFOS and PFOA.

PFCs bind to proteins in the serum of blood. Because PFCs remain in the human body for years, blood serum levels of PFCs are reflective of long-term exposures to these contaminants. Serum accounts for about half the weight of whole blood, so the blood serum concentration of PFCs is about twice the concentration of PFCs in whole blood.[89] The blood serum PFC levels for this indicator are given in nanograms of PFC per milliliter of blood serum (ng/mL).[ii]

[ii] Most persistent organic pollutants (POPs) are lipophilic, meaning that they accumulate in fatty tissues; however, this is not the case for PFCs, which are both hydrophobic (water-repelling), and oleophobic (oil-repelling). They

Concentrations of 12 different PFCs, including PFOS, PFOA, PFHxS, and PFNA, have been measured in blood serum from a representative subset of NHANES participants ages 12 years and older beginning with the 1999–2000 survey cycle, although PFCs were not measured in the 2001–2002 cycle.

In 2007–2008, NHANES collected PFCs biomonitoring data for 2,100 individuals ages 12 years and older, including 495 women ages 16 to 49 years. The four selected PFCs were detected in 99% to 100% of the individuals sampled in NHANES 2007–2008. The median and 95th percentile of blood serum PFC levels for all NHANES participants in 2007–2008 were 14 ng/mL and 41 ng/mL, respectively, for PFOS; 4 ng/mL and 10 ng/mL, respectively, for PFOA; 2 ng/mL and 10 ng/mL, respectively, for PFHxS; 2 ng/mL and 4 ng/mL, respectively, for PFNA.

Birth Rate Adjustment

Indicator B6 uses measurements of PFCs in blood serum of women ages 16 to 49 years to represent the distribution of PFC exposures to women who are pregnant or may become pregnant. However, women of different ages have a different likelihood of giving birth. For example, in 2003–2004, women aged 27 years had a 12% annual probability of giving birth, and women aged 37 years had a 4% annual probability of giving birth.[90] A birth rate-adjusted distribution of women's PFC levels is used in calculating this indicator,[iii] meaning that the data are weighted using the age-specific probability of a woman giving birth.[91]

Data Presented in the Indicator

Indicator B6 presents median concentrations of PFOS, PFOA, PFHxS, and PFNA in blood serum over time for women ages 16 to 49 years, using NHANES data from 1999–2008 (excluding the years 2001–2002).

Additional information on the 95th percentile blood serum levels of PFOS, PFOA, PFHxS, and PFNA for women ages 16 to 49 years is presented in the supplemental data tables for this indicator, along with information showing how median and 95th percentile blood serum levels of PFCs in women of child-bearing age vary by race/ethnicity and family income.

Please see the Introduction to the Biomonitoring section for an explanation of the terms "median" and "95th percentile," a description of the race/ethnicity and income groups used in the ACE3 biomonitoring indicators, and information on the statistical significance testing applied to these indicators.

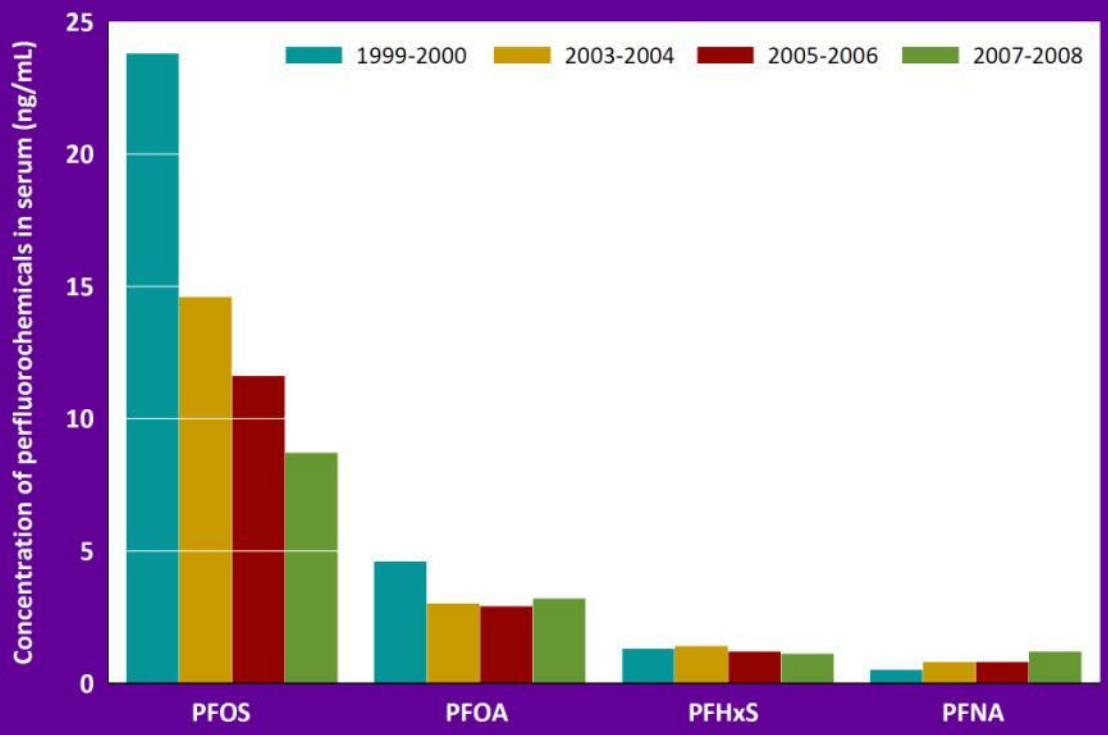
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instead bind to proteins in the serum of blood. While blood levels of lipophilic POPs are commonly lipid-adjusted, the PFC measurements in blood are not.

[iii] There may be multiple ways to implement an adjustment to the data that accounts for birth rates by age. The National Center for Health Statistics has not fully evaluated the method used in ACE, or any other method intended to accomplish the same purpose, and has not used any such method in its publications. NCHS and EPA are working together to further evaluate the birth rate adjustment method used in ACE and alternative methods.

Indicator B6

Perfluorochemicals in women ages 16 to 49 years: Median concentrations in blood serum, 1999-2008



Data: Centers for Disease Control and Prevention, National Center for Health Statistics and National Center for Environmental Health, National Health and Nutrition Examination Survey

Note: To reflect exposures to women who are pregnant or may become pregnant, the estimates are adjusted for the probability (by age and race/ethnicity) that a woman gives birth.

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Data characterization

- Data for this indicator are obtained from an ongoing continuous survey conducted by the National Center for Health Statistics.
- Survey data are representative of the U.S. civilian noninstitutionalized population.
- PFCs are measured in blood samples obtained from individual survey participants.

- Between 1999–2000 and 2007–2008, median blood serum levels of PFOS in women of child-bearing age declined from 24 ng/mL in 1999–2000 to 9 ng/mL in 2007–2008. Median blood serum levels of PFOA in women of child-bearing age declined from 5 ng/mL in 1999–2000 to 3 ng/mL in 2007–2008. These decreasing trends were statistically significant.
- The median blood serum levels of PFHxS and PFNA were lower than those of PFOS and PFOA in women of child-bearing age. Median levels of PFHxS have remained relatively

constant over time. Between 1999–2000 and 2007–2008, median blood serum levels of PFNA showed an increasing trend, from 0.5 ng/mL in 1999–2000 to 1.2 ng/mL in 2007–2008.

- The increasing trend in median PFNA levels was statistically significant.

- The concentration of PFOS in blood serum at the 95th percentile in women of child-bearing age showed a decreasing trend from 50 ng/mL in 1999–2000 to 23 ng/mL in 2007–2008. The concentration of PFOA in blood serum at the 95th percentile in women of child-bearing age remained relatively constant between 1999–2000 and 2007–2008. (See Table B6a.)
  - The decreasing trend in 95th percentile PFOS levels was statistically significant.
- For the years 2005–2008, women of child-bearing age living at or above poverty level had higher median and 95th percentile concentrations of PFOS and PFOA in their blood serum compared with women living below poverty level. (See Tables B6b and B6c.)
  - The differences between income groups were statistically significant after adjustment for differences in race/ethnicity and age.
- For the years 2005–2008, median concentrations of PFOA were higher in White non-Hispanic women of child-bearing age (3.5 ng/mL) compared with Black non-Hispanic women (2.7 ng/mL), Mexican-American women (2.3 ng/mL), and women of "All Other Races/Ethnicities" (2.4 ng/mL). (See Table B6b.)
  - These differences in median PFOA concentrations by race/ethnicity were statistically significant. The difference in median concentrations between White non-Hispanic and Black non-Hispanic women was no longer statistically significant after accounting for other demographic characteristics (differences in age and income).
- In 2005–2008, median and 95th percentile concentrations of PFOS were lower in Mexican-American women of child-bearing age at 7.4 ng/mL and 17.3 ng/mL, respectively, compared with White non-Hispanic women at 11.4 ng/mL and 28.4 ng/mL, respectively, Black non-Hispanic women at 11.2 ng/mL and 25.7 ng/mL, respectively, and women of "All Other Races/Ethnicities" at 8.3 ng/mL and 24.9 ng/mL, respectively. (See Tables B6b and B6c.)
  - These differences were statistically significant both with and without adjustment for other demographic characteristics, with the following exceptions: the difference between Mexican-American women and women of "All Other Races/Ethnicities" was statistically significant at the median only after accounting for differences by age and income; and was not statistically significant at the 95th percentile.

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Polychlorinated Biphenyls (PCBs)

Polychlorinated biphenyls (PCBs) are a family of industrial chemicals that were produced in the United States from 1929 to 1979 and used primarily as insulating fluids in capacitors, transformers, and other electrical equipment.[1] PCBs were also used as plasticizers in many paints, plastics, and rubber products, and had numerous applications in industry and building construction.[1]

Each PCB has a common structure of a biphenyl molecule with 1 to 10 chlorine atoms attached; each possible variant is called a congener. In theory, there could be as many as 209 PCB congeners; however, a smaller number of congeners were found in manufactured PCB mixtures, and measurements of PCBs in the environment and in human blood samples typically target a subset of the congeners.[2,3]

The PCB congeners are sometimes separated into two categories, "dioxin-like" or "non-dioxin-like," that are defined by structural differences and that act by different toxicological mechanisms.[4,5] The dioxin-like PCBs are structurally and toxicologically related to the chemical 2,3,7,8- tetrachlorodibenzo-p-dioxin, which has been studied very extensively in toxicological and epidemiological research. However, both categories have been associated with adverse health outcomes and it is unknown which congeners are the most potent, particularly for outcomes most relevant to children's health.[2,4]

Manufacture, sale, and use of PCBs was generally banned in the United States in 1979,[6] but EPA regulations have authorized their continued use in certain equipment manufactured prior to the ban. Due to their persistent nature, PCBs remain widely distributed in the environment, and they are also present at many Superfund sites.[2] The persistent nature of PCBs and their distribution through the food chain has resulted in continuing human exposure. However, dietary intake of PCBs and levels measured in blood serum have declined since the ban.[2,7,8] Measured levels of PCBs in human blood decreased by an estimated 87% from 1973–2003,[7] and levels of PCB-153, one of the major PCB congeners, also showed significant decline from the late 1980s to 2002.[8] Although levels of PCBs in environmental samples have declined from their peak, the rate of decline has slowed in recent years.[9,10]

A large body of health effects research comes from children born to mothers who were exposed to high concentrations of a mixture of PCBs and polychlorinated dibenzofurans (a class of dioxin-like chemicals) in accidental poisoning incidents in Taiwan and Japan. These prenatally exposed children exhibited a number of adverse health effects, including neurodevelopmental effects such as cognitive deficits, developmental delays, effects on motor skills, behavioral effects, immunological effects, and skin alterations ranging from irritation to chloracne,[2] a potentially serious inflammatory condition.[11-16]

Following the poisoning incidents, several studies have been conducted to examine the effects of PCBs at more typical exposure levels. Many of these studies have linked early-life exposure

to PCBs with neurodevelopmental effects, such as lowered intelligence, and behavioral deficits, including inattention and impulsive behavior.[17-23] The observed effects have been most frequently associated with exposure in the womb resulting from the mother having eaten food contaminated with PCBs,[24-29] but some studies have detected relationships between adverse effects and PCB exposure during infancy and childhood.[22,29-31] Although there is some inconsistency in the epidemiological literature, several reviews of the literature have concluded that the overall evidence supports a concern for effects of PCBs on children's neurological development.[16,30,32-34] The Agency for Toxic Substances and Disease Registry has determined that "Substantial data suggest that PCBs play a role in neurobehavioral alterations observed in newborns and young children of women with PCB burdens near background levels."[2] Research on dioxin-like chemicals in general also supports a concern for neurodevelopmental effects from the dioxin-like PCBs.[35] Similar outcomes have been observed in experimental animal studies, including behavioral changes and learning deficits in rats and monkeys exposed to PCBs in their diets.[2,36]

Prenatal PCB exposures have also been associated with immunological effects, such as increased infections, in multiple epidemiological studies,[37-43] with supporting evidence from the literature on effects of dioxin-like chemicals.[35] Possible other effects of exposure to PCBs—with limited or inconclusive evidence—include preterm birth and low birthweight,[16] as well as effects on the timing of puberty in both boys and girls.[44] PCBs are also considered "reasonably anticipated to be human carcinogens," based on experimental animal studies.[45]

Biomonitoring data in U.S. children under 12 years of age is limited. One study of 6- to 9-year-old girls from 2005–2007 in California and Ohio showed a median level of PCB-153, the congener with the highest concentration, of 7.4 nanograms per gram of lipid (ng/g lipid). The same congener was measured in a nationally representative sample of the U.S. population ages 12 years and older in 2003–2004. The median level of PCB-153 in males and females ages 12 to 19 years was 5.4 ng/g lipid and for adults ages 20 years and over the median level was 24.2 ng/g lipid.[46,47]

Due to the continued presence of PCBs in fish, especially salmon, meat, poultry, dairy products, and breast milk,[48] dietary intake is an important pathway of exposure for PCBs.[2] In infants, dietary intake is important since PCBs accumulate in the mother's body over many years and are stored in the fat in breast milk,[35] and breast-feeding infants are exposed to the PCBs in the milk.[49] PCBs can also cross the placental barrier to transfer prenatally from mother to fetus, and PCBs have been measured in cord blood.[2,50]

Recent findings also suggest that the presence of PCBs in indoor dust and indoor air may constitute an important exposure pathway for some portion of the population.[51-54] The importance of PCBs in indoor environments may be greater for toddlers than for adults and children of other ages, because toddlers tend to have more contact with house dust.[51] A study of homes with unusually high indoor air concentrations of PCBs found that a PCB-containing wood flooring finish applied in the 1950s and 1960s can be a major contributor to current elevations of PCBs in blood for people living in those homes.[53] PCBs have been found in caulk in

some schools and other buildings constructed or renovated before the late 1970s, which may contribute significantly to indoor air and dust levels of PCBs in those buildings.[55,56] Many schools have lighting systems containing PCBs that were produced before PCBs were banned. While well-contained lighting systems pose little risk, the PCB-containing ballasts are only expected to last 10–15 years. Existing ballasts from before the ban are past their life expectancy and are at a greater risk for leaks and fires, resulting in a greater risk of PCB exposure.[57] Finally, the inadvertent presence of PCBs has been found in pigments that are currently manufactured for use in paints, inks, textiles, paper, cosmetics, leather, and other materials.[58,59]

Blood levels of PCBs generally increase with age, because these chemicals are persistent.[60,61] However, the decline in levels of PCBs in the environment and in foods over the past three decades, suggests that young people today are exposed to lower levels of PCBs through the diet than were previous generations.[2,7,9,10]

Although environmental levels of PCBs have been declining, there are concerns that some past PCB emissions trapped in polar ice may be released to the environment in coming years with increasing ice melts.[62,63] Furthermore, environments where heavy PCB contamination previously occurred continue to be remediated, which may dislodge or expose additional PCBs. The Hudson River, contaminated with 1.3 million pounds of PCBs between 1947 and 1977, is undergoing remediation to remove PCB-contaminated sediments. After Phase 1 of the remediation in 2009 there was a short-term increase in the PCB levels in fish samples, but more recent samples from 2010 did not have increased PCB concentrations.[64,65]

The following indicator presents the best nationally representative data on PCB levels in women of child-bearing age. Indicator B7 presents median blood serum levels of PCBs for women ages 16 to 49 years. Although data are available only for two two-year survey periods at this time, the data provide a baseline that will be updated with PCB measurements over time from subsequent survey cycles. No indicator is presented for PCBs in children due to the limited availability of data.

Indicator B7: PCBs in women ages 16 to 49 years: Median concentrations in blood serum, by race/ethnicity and family income, 2001–2004

About the Indicator: Indicator B7 presents concentrations of PCBs in blood serum of U.S. women ages 16 to 49 years. The data are from a national survey that collects blood specimens from a representative sample of the population every two years, and then measures the concentration of PCBs in the blood serum. The indicator presents comparisons of PCBs in blood serum for women of different race/ethnicities, and for women of different income levels. The focus on women of child-bearing age is based on concern for potential adverse effects in children born to women who have been exposed to PCBs.

NHANES

The National Health and Nutrition Examination Survey (NHANES) provides nationally representative biomonitoring data for PCBs. NHANES is designed to assess the health and nutritional status of the civilian noninstitutionalized U.S. population and is conducted by the National Center for Health Statistics, part of the Centers for Disease Control and Prevention (CDC). Interviews and physical examinations are conducted with approximately 10,000 people in each two-year survey cycle. CDC's National Center for Environmental Health measures concentrations of environmental chemicals in blood and urine samples collected from NHANES participants. Summaries of the measured values for more than 200 chemicals are provided in the *Fourth National Report on Human Exposure to Environmental Chemicals*.[46]

PCB Congeners

Indicator B7 presents blood serum levels of PCBs in women of child-bearing age. There are 209 possible PCBs, referred to as "congeners," which are defined by the number of chlorine atoms (from 1 to 10) and their position in the chemical structure. Most of these congeners were not present in the manufactured PCB mixtures and have not been measured in environmental or human samples.

PCB concentrations are measured in blood serum. PCBs are lipophilic, meaning that they tend to accumulate in fat. Serum PCB concentrations are measured and expressed on a lipid-adjusted basis, as these values better represent the amount of PCBs stored in the body compared with unadjusted values.[46] The indicator uses lipid-adjusted concentrations, meaning that the concentration of PCBs in serum is divided by the concentration of lipid in serum. The resulting units are nanograms of PCB per gram of lipid (ng/g lipid) in serum.[i]

[i] Serum levels of PCBs can also be reported without lipid adjustment. Both the lipid-adjusted values and the unadjusted "whole weight" values are reported in CDC's Fourth National Report on Human Exposure to Environmental Chemicals.

Concentrations of PCBs in blood serum have been measured in a representative subset of NHANES participants ages 12 years and older beginning with the 1999–2000 survey cycle. NHANES sampled for 34 PCB congeners in 2001–2002, and added 4 congeners in 2003–2004 for a total of 38 congeners. Indicator B7 uses NHANES data on four specific congeners: PCBs 118, 138, 153, and 180. These four congeners are generally found at higher levels in the environment—and in human blood samples—than other PCB congeners. This combination of congeners has been frequently used to represent PCB exposure in the epidemiological studies described above that identified children's health concerns for PCBs. PCBs 118, 138, 153, and 180 were detected in the majority of samples for women ages 16 to 49 years in 2001–2002, and in virtually all samples for this population group in 2003–2004.

Indicator B7 was calculated by summing together the measured values of the 4 selected congeners for each woman 16 to 49 years sampled in NHANES; this approach is commonly used in studies assessing levels of PCBs in human blood samples and environmental samples.[2,30] If the congener was not detected in a sample, a default value below the detection limit was assigned for purposes of calculating the summed total.[ii] This assumption has a small impact on the indicator values, because all four congeners were detected in most samples in the combined four-year (2001–2004) data set.

In 2001–2004, a sum of measured PCBs 118, 138, 153, and 180 is available from NHANES for 4,205 individuals ages 12 years and older, including 1164 women ages 16 to 49 years. The four selected PCBs were detected in 81% of the individuals sampled in NHANES 2001-2004,[iii] and in 71% of women ages 16 to 49 years.[iv] The median sum of the four PCB congeners in blood serum among all NHANES participants in 2001-2004 was 71 ng/g lipid and the 95th percentile sum was 316 ng/g lipid.

Birth Rate Adjustment

Indicator B7 uses measurements of PCBs in the blood of women ages 16 to 49 years to represent the distribution of PCB exposures to women who are pregnant or may become pregnant. However, women of different ages have a different likelihood of giving birth. For example, in 2003–2004, women aged 27 years had a 12% annual probability of giving birth, and women aged 37 years had a 4% annual probability of giving birth.[66] A birth rate-adjusted distribution of women's PCB levels is used in calculating this indicator,[v] meaning that the data are weighted using the age-specific probability of a woman giving birth.[67]

[ii] The default value used for non-detect samples is equal to the limit of detection divided by the square root of 2.
[iii] In 2003–2004, PCBs were detected in 100% of the individuals sampled. The detection frequency was lower in 2001–2002 due to use of less sensitive measurement techniques.
[iv] The percentage for women ages 16 to 49 years is calculated with the birth rate adjustment described below.
[v] There may be multiple ways to implement an adjustment to the data that accounts for birth rates by age. The National Center for Health Statistics has not fully evaluated the method used in ACE, or any other method

Data Presented in the Indicator

Indicator B7 presents median concentrations of PCBs in blood serum, computed as the sum of PCBs 118, 138, 153, and 180, for women ages 16 to 49 years of different races/ethnicities and levels of family income, using NHANES data from 2001–2002 and 2003–2004.

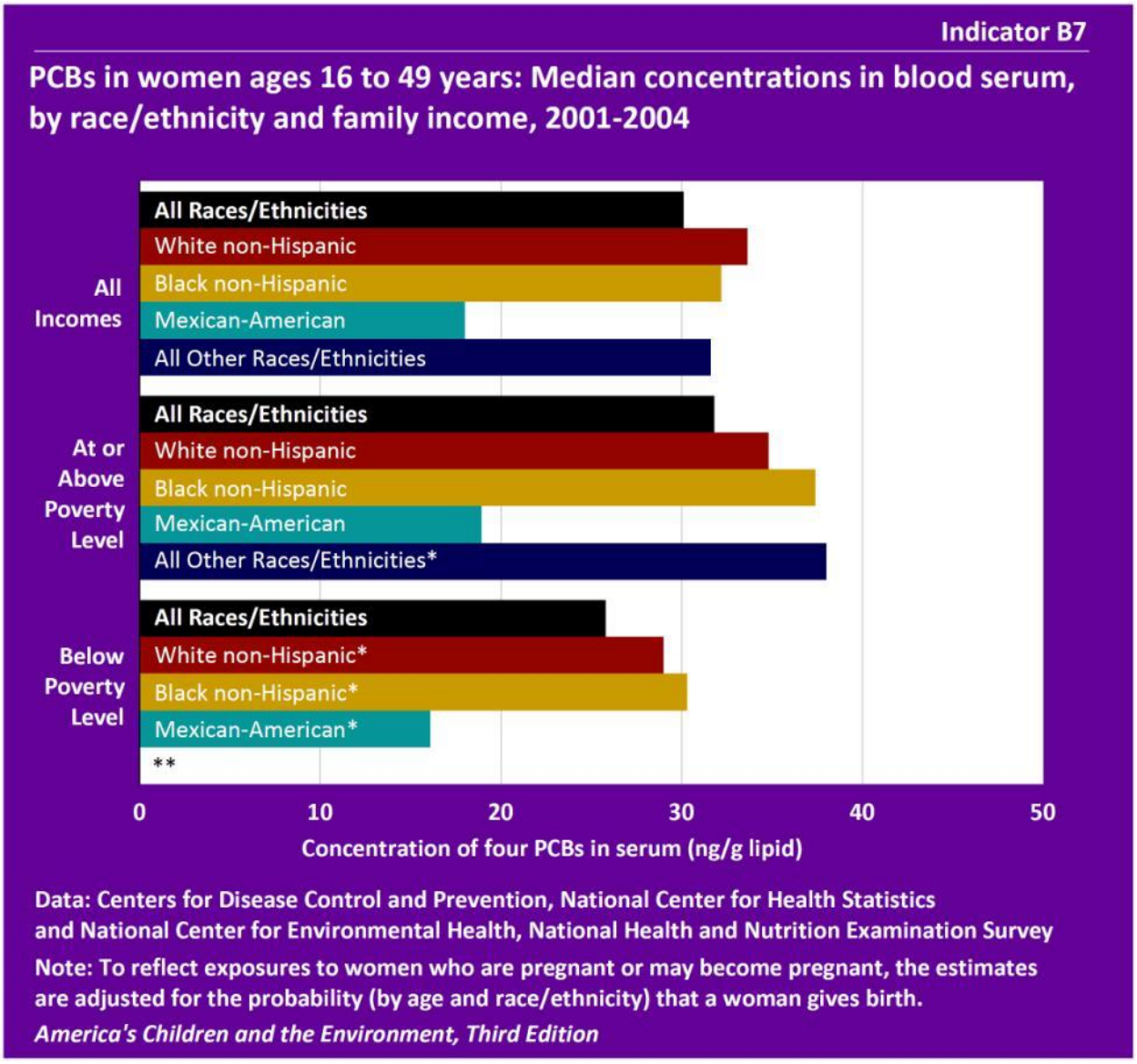
Data from 1999–2000 are not included in the indicator because less sensitive measurement techniques were used in those years, and PCB levels could not be determined in a large proportion of the blood samples. Improvements in measurement sensitivity were achieved in 2001–2002, with further improvements in 2003–2004 resulting in the detection of PCBs in a majority of samples.[61] The data from the 2001–2002 and 2003–2004 NHANES cycles are combined to increase the statistical reliability of the estimates for each race/ethnicity and income group, and to reduce any possible influence of geographic variability that may occur in two-year NHANES data. No time series is shown because data from only two NHANES cycles are too limited to depict possible changes over time.

Four race/ethnicity groups are presented in Indicator B7: White non-Hispanic, Black non-Hispanic, Mexican-American, and "All Other Races/Ethnicities." The "All Other Races/Ethnicities" category includes all other races and ethnicities not specified, together with those individuals who report more than one race. The limits of the sample design and sample size often prevent statistically reliable estimates for smaller race/ethnicity groups. The data are also tabulated across three income categories: all incomes, below the poverty level, and greater than or equal to the poverty level.

Additional information on how 95th percentile blood serum levels of PCBs vary by race/ethnicity and family income for women ages 16 to 49 years is presented in the supplemental data tables for this indicator

Please see the Introduction to the Biomonitoring section for an explanation of the terms "median" and "95th percentile," along with information on the statistical significance testing applied to these indicators.

intended to accomplish the same purpose, and has not used any such method in its publications. NCHS and EPA are working together to further evaluate the birth rate adjustment method used in ACE and alternative methods.



*The estimate should be interpreted with caution because the standard error of the estimate is relatively large: the relative standard error, RSE, is at least 30% but is less than 40% (RSE = standard error divided by the estimate), or the RSE may be underestimated.

** The estimate is not reported because it has large uncertainty: the relative standard error, RSE, is 40% or greater (RSE = standard error divided by the estimate), or the RSE cannot be reliably estimated.

Data characterization

- Data for this indicator are obtained from an ongoing continuous survey conducted by the National Center for Health Statistics.
- Survey data are representative of the U.S. civilian noninstitutionalized population.
- PCBs are measured in blood samples obtained from individual survey participants.

- In 2001–2004, the median level of PCBs in blood serum among women ages 16 to 49 years (the sum of PCBs 118, 138, 153 and 180) was 30 ng/g lipid.
- Median PCB levels were higher for women with higher incomes than for women with lower incomes, consistently for all race/ethnicity groups.
  - After accounting for other demographic differences (i.e., differences in age profile), the differences between income levels for each race/ethnicity group were not statistically significant except for the differences for White non-Hispanic women.
- Median PCB levels were lower among Mexican-American women than among women of any other race/ethnicity group.
  - These differences were statistically significant. After accounting for other demographic differences (i.e., differences in income or age profile), the differences remained statistically significant except for that between Mexican-American women and women of "All Other Races/Ethnicities."
- The 95th percentile concentration of PCBs among women ages 16 to 49 years was 106 ng/g lipid. Among women of "All Other Races/Ethnicities," the 95th percentile PCB concentration was substantially higher, at 245 ng/g lipid; the 95th percentile concentration among Mexican-American women was substantially lower at 49 ng/g lipid. (See Table B7a.)
  - These differences were statistically significant: the 95th percentile for women of "All Other Races/Ethnicities" was greater than the value for each of the remaining race/ethnicities; and the 95th percentile for Mexican-American women was less than the value for each of the remaining race/ethnicities.

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Polybrominated Diphenyl Ethers (PBDEs)

Polybrominated diphenyl ethers (PBDEs) are a group of brominated flame retardant chemicals that have been incorporated into a variety of manufactured products, including foam cushioning used in furniture and plastics used in televisions and computers. Flame retardants are intended to slow the rate of ignition and fire growth, allowing more time for people to escape from a fire or extinguish it.

All PBDEs have a common structure of a diphenyl ether molecule, which may have from 1–10 bromine atoms attached; each particular PBDE variant is referred to as a congener. In theory, there could be as many as 209 PBDE congeners, but a much smaller number of congeners are commonly found in the commercial PBDE mixtures and in measurements of PBDEs in humans and the environment.

Three commercial PBDE mixtures have been used in manufactured products since the 1960s and 1970s, when these chemicals came into use,[1] with each mixture made up of congeners with varying degrees of bromination. The commercial pentabromodiphenyl ether (pentaBDE) and octabromodiphenyl ether (octaBDE) mixtures have not been manufactured or imported in the United States since 2004. The pentaBDE mixture, made up primarily of four- and five-bromine congeners, was used almost entirely in flexible polyurethane foam in furniture, mattresses, carpet padding, and automobile seats; and the octaBDE mixture, made up primarily of seven- and eight-bromine congeners, was used in acrylonitrile-butadiene-styrene (ABS) plastic for certain electric and electronic devices.

A third product, the commercial decabromodiphenyl ether (decaBDE) mixture, is still manufactured and used in the United States. The decaBDE mixture, made up almost entirely of the 10-bromine congener, has been used primarily in high-impact polystyrene (HIPS) plastic that was frequently used to make the back part of television sets, and in other electronic devices. However, there are indications that the use of decaBDE in electronic devices has declined in recent years, particularly since restrictions on the use of decaBDE in electronics were implemented in Europe beginning in 2008. DecaBDE is also used as a flame retardant on certain types of textiles; in electrical products, including uses in vehicles and airplanes; and in certain building materials. The major U.S. importers and manufacturers of decaBDE have announced that this mixture will be phased out by the end of 2013.[2] As use of PBDEs is reduced, they are being replaced by other flame-retardant chemicals or by materials that are inherently resistant to fire. EPA has conducted an assessment of alternatives to commercial pentaBDE,[3] and is conducting a similar assessment of alternatives to commercial decaBDE.[4]

PBDEs can be released into the environment at various points in their lifecycle, from their production and application to consumer products to their release from discarded products in landfills. Since PBDEs are not chemically bound to the products in which they are used, they can easily migrate into the surrounding air, dust, soil, and water. Although production and use of the commercial PBDE mixtures has been phased out (pentaBDE and octaBDE) or will soon be

phased out (decaBDE), it is likely that PBDE congeners will continue to be present in the environment for many years. This is because products previously manufactured with PBDEs (e.g., sofas) will stay in use for many years. PBDEs will continue to be released from these products while they are in use, and these releases may continue when the products are disposed of or recycled. PBDEs are persistent in the environment, so even if there were no further releases they would continue to be detected for many years.

Exposure studies, focusing on selected PBDE congeners that were most predominant in the commercial mixtures or that are frequently measured in environmental samples, have concluded that the presence of PBDEs in house dust and in foods are both important contributors to PBDE exposures for people of all ages, and that exposures from house dust are generally greater than those from food.[1,5-11]

Studies conducted in multiple locations have consistently found PBDEs in U.S. house dust at levels greater than those found in other countries.[12-14] This is likely due to greater use of PBDE-containing products in homes in the United States than in other countries. Within the United States, the highest levels of three frequently measured PBDE congeners in dust have been found in California. The three congeners were all components of commercial pentaBDE mixtures, and the authors of these studies observed that the elevated levels may be due to California requirements for flame resistance in residential furniture that are not applicable in other states.[13,15] A study conducted in adults found a stronger association between direct contact with PBDE-containing materials and PBDE blood levels than between PBDE-contaminated house dust and PBDE blood levels.[16]

A second pathway of exposure to PBDEs is through diet. PBDEs are generally persistent chemicals that accumulate in fat tissue, so they are commonly found in foods derived from animals.[17-19] Information about how PBDEs enter the food web is limited, but release from manufacture of the PBDEs or of PBDE-containing products; release of PBDEs from products while they are in use; and release from products when disposed of or recycled are all likely contributors to PBDEs in the environment. PBDEs have been measured in a variety of supermarket foods, with the highest levels found in fish and other foods of animal origin.[18,20] A California study found associations between pork and poultry consumption and the levels of PBDEs measured in blood of children ages 2 to 5 years.[13]

Levels of PBDEs measured in blood are substantially greater in North America than in Europe and Asia, a difference that appears to be due to the higher levels of PBDEs in house dust in North America.[7,12,21,22] Studies comparing archived and current samples of blood and pooled serum from various locations in the United States have shown marked increases in PBDE levels since the late 1970s.[23,24]

Early-life exposures to PBDEs may be elevated in a number of ways. A child's exposure to PBDEs begins in the prenatal period, as PBDEs have been measured in cord blood, fetal blood, and placental tissue[25-27] and continues in early infancy due to the presence of PBDEs in breast milk.[11,22,28-31] Levels of PBDEs in breast milk are higher in North America than elsewhere,[12] and

estimated intakes of PBDEs are substantially greater for a breastfeeding infant than exposures that occur during other life stages.[6,22]

Exposures are also elevated for young children up to age 7 years. While few studies have measured concentrations of PBDEs in young children, one large study conducted in Australia found that levels of PBDEs in blood are greatest for children ages 2 to 5 years, compared with older children and adults.[32] A study of 20 young children (ages 1.5 to 4 years) in various locations throughout the United States found that their PBDE blood levels were consistently higher than those of their mothers.[33] A study of California children ages 2 to 5 years found PBDE blood levels generally greater than those measured in California residents ages 12 to 60 years.[13,15] A study of 7-year-old Mexican-American children living in California reported PBDE blood levels that were three times the levels found in their mothers during pregnancy.[34]

The elevated exposures observed for young children are likely due to increased exposure to house dust, based on several studies that have estimated exposures based on measured levels of PBDEs in house dust, air, and food.[6,7,9,35] Infants and small children may have the highest exposure to PBDEs in house dust due to their frequent and extensive contact with floors, carpets, and other surfaces where dust gathers, as well as their frequent hand-to-mouth activity.[36] However, children of all ages (as well as adults) are likely to be exposed to dust contaminants through hand-to-mouth activity and other ingestion pathways, such as the settling of dust onto food and food preparation surfaces in the kitchen, as well as inhalation and absorption of PBDEs through the skin.[1,9,22]

Concerns about the health effects of PBDEs are based largely on laboratory animal studies, along with findings of the limited number of human epidemiological studies that have been conducted to date. A primary concern from the animal studies is for effects on the developing brain and nervous system, including effects on learning, memory, and behavior.[37-39] A study of children in New York City found significant associations between children's prenatal exposure to PBDEs and performance on IQ tests at ages up through 6 years.[40] A second epidemiological study conducted in the Netherlands found that prenatal exposure to PBDEs was associated with reduced scores on some tests of neurological development and improved scores on other tests at ages 5 to 6 years.[41]

PBDEs are suspected endocrine disruptors.[37] Endocrine disruptors act by interfering with the biosynthesis, secretion, action, or metabolism of naturally occurring hormones.[42,43] Given the importance of hormones in human physiology, there is concern in the scientific community over the potential for endocrine disruptors to adversely affect children's health, particularly in reproduction, early and adolescent development, and behavior.

Animal and human studies indicate that PBDEs may alter circulating levels of thyroid hormones.[37,44-47] Moderate deficits in maternal thyroid hormone levels during early pregnancy have been linked to reduced childhood IQ scores and other neurodevelopmental effects, as well as unsuccessful or complicated pregnancies.[48] Animal studies have found that PBDE exposure at critical stages of fetal development reduced levels of male hormones or caused other changes

relevant to male reproductive development.[37,47,49-51] An epidemiological study of boys born in Denmark and Finland found that increased levels of PBDEs in breast milk were associated with an increased risk of cryptorchidism (undescended testes),[52] an effect that may be related to hormone disruption during critical stages of development.[53,54] Also, a study of Mexican immigrant women in California found effects on fertility (increased time to pregnancy) with increasing PBDE levels; this finding may be related to hormonal activity of PBDEs.[55]

The following indicator presents the best nationally representative data on PBDE levels in women of child-bearing age. Indicator B8 presents median blood serum levels of PBDEs for women ages 16 to 49 years. Although data are available only for a single two-year period at this time, the data provide a baseline that will be updated with PBDE measurements over time from subsequent survey cycles.

Indicator B8: PBDEs in women ages 16 to 49 years: Median concentrations in blood serum, by race/ethnicity and family income, 2003–2004

About the Indicator: Indicator B8 presents concentrations of PBDEs in blood serum of U.S. women ages 16 to 49 years. The data are from a national survey that collects blood specimens from a representative sample of the population every two years, and then measures the concentration of PBDEs in the blood serum. The indicator presents comparisons of PBDEs in blood serum for women of different race/ethnicities, and for women of different income levels. The focus on women of child-bearing age is based on concern for potential effects in children born to women who have been exposed to PBDEs.

NHANES

The National Health and Nutrition Examination Survey (NHANES) provides nationally representative biomonitoring data for PBDEs. NHANES is designed to assess the health and nutritional status of the civilian noninstitutionalized U.S. population and is conducted by the National Center for Health Statistics, part of the Centers for Disease Control and Prevention (CDC). Interviews and physical examinations are conducted with approximately 10,000 people in each two-year survey cycle. CDC's National Center for Environmental Health measures concentrations of environmental chemicals in blood and urine samples collected from NHANES participants. Summaries of the measured values for more than 200 chemicals are provided in the *Fourth National Report on Human Exposure to Environmental Chemicals*.[56]

PBDE Congeners

Indicator B8 presents blood serum levels of PBDEs in women of child-bearing age. There are 209 possible PBDEs, referred to as "congeners," which are defined by the number of bromine atoms (from 1 to 10) and their position in the chemical structure. Each congener is assigned a specific brominated diphenyl ether (BDE) number, such as BDE-47 (a tetrabromodiphenyl ether congener – four bromine atoms). Most of these congeners have not been detected in the manufactured PBDE mixtures and have not been measured in environmental or human samples.

PBDE concentrations are measured in blood serum. PBDEs are lipophilic, meaning that they tend to accumulate in fat. Serum PBDEs concentrations are measured and expressed on a lipid-adjusted basis, as these values better represent the amount of PBDEs stored in the body compared with unadjusted values.[56] The indicator uses lipid-adjusted concentrations, meaning

that the concentration of PBDEs in serum is divided by the concentration of lipid in serum. The resulting units are nanograms of PBDE per gram of lipid (ng/g lipid) in serum.[i]

Concentrations of PBDEs in blood serum have been measured in a representative subset of NHANES participants ages 12 years and older in the 2003–2004 survey cycle. NHANES sampled for 10 PBDE congeners in 2003–2004, including those most frequently measured in environmental and human samples. These include BDEs 17, 28, 47, 66, 85, 99, 100, 153, 154, and 183. Most of these 10 congeners were components of the pentaBDE mixture that was used in polyurethane foam for furniture, mattresses, and automotive seating. Some of the congeners measured in NHANES were components of the octaBDE mixture, used in plastics for some household electric devices. The primary congener comprising the decaBDE formulation, BDE-209, was not measured in NHANES in 2003–2004.

Indicator B8 was calculated by summing together the measured values of the 10 congeners for each woman 16 to 49 years sampled in NHANES; this approach is commonly used in studies assessing levels of PBDEs in human blood samples and environmental samples.[1] Data are insufficient at this time to assess and quantify differences in toxicity of the measured PBDE congeners, or to inform approaches other than an unweighted summation of the 10 congeners.

If a congener was not detected in a particular blood sample, a default value below the detection limit was assigned for purposes of calculating the summed total for the sampled individual.[ii] This assumption has a small impact on the reported blood levels of PBDEs, because almost all women sampled had values well above the detection limit for at least some congeners. BDEs 47, 100 and 153 were each detected in more than 90% of women ages 16 to 49 years.

In 2003–2004, a sum of the 10 measured PBDEs is available from NHANES for 2,040 individuals ages 12 years and older, including 540 women ages 16 to 49 years. One or more PBDE congeners were detected in 99% of the individuals sampled in NHANES 2003–2004, and in 99% of women ages 16 to 49 years.[iii] The median sum of the ten PBDE congeners among NHANES participants in 2003–2004 was 38 ng/g lipid and the 95th percentile sum was 307 ng/g lipid.

Birth Rate Adjustment

Indicator B8 uses measurements of PBDEs in blood of women ages 16 to 49 years to represent the distribution of PBDE exposures to women who are pregnant or may become pregnant. However, women of different ages have a different likelihood of giving birth. For example, in

[i] Serum levels of PBDEs can also be reported without lipid adjustment. Both the lipid-adjusted values and the unadjusted "whole weight" values are reported in CDC's Fourth National Report on Human Exposure to Environmental Chemicals.
[ii] The default value used for non-detect samples is equal to the limit of detection divided by the square root of 2.
[iii] The percentage for women ages 16 to 49 years is calculated with the birth rate adjustment described below.

2003–2004, women aged 27 years had a 12% annual probability of giving birth, and women aged 37 years had a 4% annual probability of giving birth.[57] A birth rate-adjusted distribution of women's PBDE levels is used in calculating this indicator,[iv] meaning that the data are weighted using the age-specific probability of a woman giving birth.[58]

Data Presented in the Indicator

Indicator B8 presents median concentrations of PBDEs in blood serum for women ages 16 to 49 years of different races/ethnicities and levels of family income, using NHANES data from 2003–2004.[v]

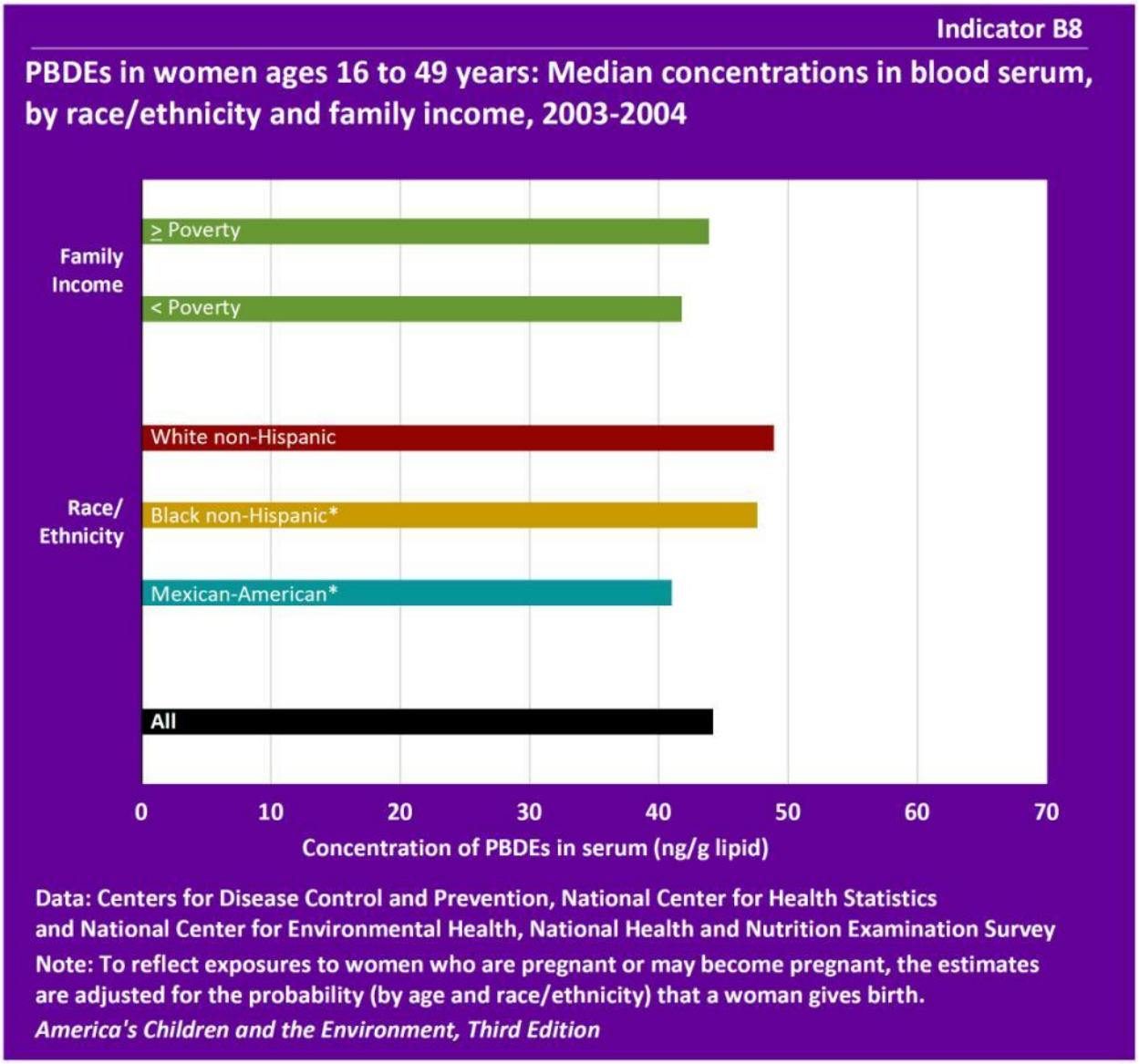
Three race/ethnicity groups are presented in Indicator B8: White non-Hispanic, Black non-Hispanic, and Mexican-American. The data are also tabulated across three income categories: all incomes, below the poverty level, and greater than or equal to the poverty level.

Additional information on how median blood serum levels of PBDEs vary by race/ethnicity and family income for children ages 12 to 17 years is presented in a supplemental data table for this indicator.

Please see the Introduction to the Biomonitoring section for an explanation of the term "median" and information on the statistical significance testing applied to this indicator.

[iv] There may be multiple ways to implement an adjustment to the data that accounts for birth rates by age. The National Center for Health Statistics has not fully evaluated the method used in ACE, or any other method intended to accomplish the same purpose, and has not used any such method in its publications. NCHS and EPA are working together to further evaluate the birth rate adjustment method used in ACE and alternative methods.

[v] Unlike other biomonitoring indicators in this report, 95th percentile PBDE levels are not provided in a supplementary table. This is because most 95th percentile PBDE values do not meet ACE statistical reliability criteria. There is more uncertainty in 95th percentile estimates for PBDEs than for other chemicals because data are only available for two years (2003–2004) at this time. Similarly, separate values are not provided considering both race/ethnicity and income simultaneously, nor are values provided for the "All Other Races/Ethnicities" category, because (with data from only one NHANES cycle available at this time) such estimates lack statistical reliability.



*The estimate should be interpreted with caution because the standard error of the estimate is relatively large: the relative standard error, RSE, is at least 30% but is less than 40% (RSE = standard error divided by the estimate), or the RSE may be underestimated.

Data characterization

- Data for this indicator are obtained from an ongoing continuous survey conducted by the National Center for Health Statistics.
- Survey data are representative of the U.S. civilian noninstitutionalized population.
- PBDEs are measured in blood samples obtained from individual survey participants.

- The median concentration of PBDEs in blood serum of women ages 16 to 49 years was 44 ng/g lipid in 2003–2004.

- White non-Hispanic women and Black non-Hispanic women had the highest median PBDE levels at 49 and 48 ng/g lipid, respectively.
  - The differences by race/ethnicity were generally not statistically significant without accounting for differences by age and income across race/ethnicity groups. After accounting for differences by age and income across race/ethnicity groups, PBDE levels in White non-Hispanic women were statistically significantly greater than levels in Black non-Hispanic women. Also after adjustment, PBDE levels in Black non-Hispanic women were statistically significantly greater than levels in Mexican-American women.
- Among women of child-bearing age, there was little difference in median PBDE concentrations in blood serum between income groups.
- The median concentration of PBDEs in children ages 12 to 17 years overall was 53 ng/g lipid. The median concentration of PBDEs for children with family incomes below the poverty level was 63 ng/g lipid, and 50 ng/g lipid for children at or above poverty level. (See Table B8a.)
  - The difference in median PBDE concentration between the income groups was not statistically significant.
- Among children ages 12 to 17, White non-Hispanic children and Black non-Hispanic children had the lowest median PBDE levels at 48 and 50 ng/g lipid. Mexican-American children had median PBDE levels of 63 ng/g lipid. (See Table B8a.)
  - These differences by race/ethnicity were not statistically significant.

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Phthalates

Phthalates are a class of manufactured chemicals commonly used to increase the flexibility of plastics in a wide array of consumer products. More than 470 million pounds of phthalates are produced or imported in the United States each year.[1]

By far the most common use of phthalates is in the production of polyvinyl chloride (PVC) products.[2] PVC is the second most commonly used plastic in the world, and is present in pipes and tubing, construction materials, packaging, electrical wiring, and thousands of consumer goods.[3,4] Phthalates are or have been used in wall coverings, tablecloths, floor tiles, furniture upholstery, carpet backings, shower curtains, garden hoses, rainwear, pesticides, some toys, shoes, automobile upholstery, food packaging, medical tubing, and blood storage bags.[5-8] Phthalates are not strongly bound in these products and can therefore leach out.[4-10] Some phthalates are also present in cosmetics, nail polish, hair products, skin care products, and some medications.[4,6,7,11,12]

The Consumer Product Safety Improvement Act of 2008 (CPSIA) banned the use of three phthalates in toys and child care articles at concentrations greater than 0.1 percent: di-2-ethylhexyl phthalate (DEHP), dibutyl phthalate (DBP), and butyl benzyl phthalate (BBzP). CPSIA also restricts the use of di-isononyl phthalate (DINP), di-isodecyl phthalate (DIDP), and di- n-octyl phthalate (DnOP) in toys that can be mouthed and child care articles. The Consumer Product Safety Commission has also appointed a Chronic Hazard Advisory Panel to examine the cumulative health risks of phthalates and phthalate substitutes, and to recommend whether to continue the ban of DINP, DIDP, and DnOP and whether any other phthalates or phthalate substitutes should be banned.[1] As use of phthalates is reduced, they are being replaced by other chemicals, such as di-isononylcyclohexane-1,2-dicarboxylate (DINCH) and di(2-ethylhexyl) terephthalate (DEHT), that also increase the flexibility of plastics.[13-15] EPA is planning to conduct an assessment of alternatives to several phthalates.[1]

For most phthalates, the major route of exposure is food ingestion.[4,16-19] However, personal care product use and inhalation are major routes of exposure for certain phthalates.[4-8,11,20] Some phthalates have been found at higher levels in fatty foods such as dairy products, fish, seafood, and oils.[8] Phthalates in a mother's body can enter her breast milk. Ingestion of that breast milk and infant formula containing phthalates may also contribute to infant phthalate exposure.[21] The phthalates that may be present in dust can be ingested by infants and children through hand-to-mouth activities.[10,22] Finally, infants and small children can be exposed to phthalates by sucking on toys and objects made with phthalate-containing plastics.[10]

Other minor routes of phthalate exposure include inhalation, drinking contaminated water, and absorption through the skin.[16,17] Phthalates can be released in small amounts to the air people breathe inside homes or schools from the many consumer products that contain them.[7,20] People living near phthalate-producing factories or hazardous waste sites may be exposed to phthalates released into the air or ground water where they live.[5,7,8] Individuals may be

exposed to phthalates during the use of many personal care products containing phthalates, such as hair products, cosmetics, and lotions.[11,23,24] Phthalates in these products may be absorbed through contact with the skin or may be inhaled if some of the product is present in the air.[5] In addition, certain medical devices, such as intravenous tubing or flexible bags containing blood, medications, or nutritional products, contain phthalates. These can be a source of phthalate exposure to children and women of child-bearing age when the tubing or bags are used to administer medications, nutritional products, or blood to the individual. This can be a very significant route of exposure, especially for premature infants in intensive care units.[25-27]

Phthalate exposures, assessed from urinary concentrations of phthalate metabolites (i.e., breakdown products), appear to be higher for children compared with adolescents and adults. Studies of phthalate metabolites in children's urine are limited, but the few that have been published have found children's urinary phthalate metabolite levels to be higher than levels in adults and to decrease with age (i.e., younger children had more phthalate metabolites in their urine than older children did).[28-30] The exception is monoethyl phthalate (MEP), a metabolite of diethyl phthalate, which has been found to be present in higher levels in adult urine compared with children's urine.[28] Levels of MEP are most likely associated with use of consumer products that contain diethyl phthalate, such as detergents, soaps, cosmetics, shampoos, and perfumes.[5,28]

Some phthalates are suspected endocrine disruptors.[31-35] Endocrine disruptors act by interfering with the biosynthesis, secretion, action, or metabolism of naturally occurring hormones.[32,36] Given the importance of hormones in human physiology, there is concern in the scientific community over the potential for endocrine disruptors to adversely affect children's health, particularly in reproduction, development, and behavior. Male laboratory animals exposed to high doses of some phthalates have been known to display elements of "phthalate syndrome," which includes infertility, decreased sperm count, cryptorchidism (undescended testes), hypospadias (malformation of the penis in which the urethra does not open at the tip of the organ), and other reproductive tract malformations.[4] A number of animal studies have reported associations between exposure to certain phthalates and changes in male hormone production, altered sexual differentiation, and changes to reproductive organs, including hypospadias.[37-45] These findings in animal studies, although typically occurring at exposure levels much higher than what the general population may be exposed to, suggest a potential concern for health effects in children as well. The National Research Council has concluded that prenatal exposure to certain phthalates produces reproductive tract abnormalities in male rats, and also concluded that the same effects could plausibly occur in humans.[4]

There are only a limited number of human studies looking at the relationship between phthalate exposure and hormonal and reproductive health changes. In one study, prenatal exposure to some phthalates at typical U.S. population levels was associated with changes in physical measures of the distance between the anus and the genitals (anogenital distance) in male infants.[46,47] A shorter anogenital distance has been associated with decreased fertility in animal experiments[48,49] and a recent human study reported that a shorter anogenital distance

in men was associated with decreased semen quality and low sperm count.[50] Another study reported an association between increased concentrations of phthalate metabolites in breast milk and altered reproductive hormone levels in newborn boys. The same study did not find an association between breast milk phthalate metabolite concentrations and cryptorchidism.[51]

Exposure to some phthalates has been associated with neurodevelopmental problems in children in some studies. Two studies of a group of New York City children ages 4 to 9 years reported associations between prenatal exposure to certain phthalates and behavioral deficits, including effects on attention, conduct, and social behaviors.[52,53] Studies conducted in South Korea of children ages 8 to 11 years reported that children with higher levels of certain phthalate metabolites in their urine were more inattentive and hyperactive, displayed more symptoms of ADHD, and had lower IQ compared with those who had lower levels.[54,55] The exposure levels in these studies are comparable to typical exposures in the U.S. population.

A handful of studies have reported associations between prenatal exposure to some phthalates and preterm birth, shorter gestational length, and low birth weight;[56-59] however, one study reported phthalate exposure to be associated with longer gestational length and increased risk of delivery by Cesarean section.[60]

Finally, some researchers have hypothesized that phthalate exposure in homes may contribute to asthma and allergies in children. Two research groups have conducted studies, primarily in Europe, and reported associations between surrogates for potential phthalate exposure in the home and risk of asthma and allergies in children.[61] Examples of the exposure indicators and outcomes considered in these studies include an association between some phthalates in surface dust and increased risk of runny nose, eczema, and asthma,[62] and increased risk of bronchial obstruction associated with the presence of PVC in the home.[63]

The two indicators that follow use the best nationally representative data currently available on urinary phthalate metabolite levels over time for women of child-bearing age and children. The indicators focus on three important phthalates: di-2-ethylhexyl phthalate (DEHP), dibutyl phthalate (DBP), and butyl benzyl phthalate (BBzP). These three phthalates were chosen because their metabolites are commonly detected in humans and their potential connection to adverse children's health outcomes is supported by the scientific literature summarized in the following paragraphs.

DEHP is currently the only phthalate plasticizer used in PVC medical devices such as blood bags and plastic tubing. DEHP is also currently used in flooring, wallpaper, and raincoats and has been used in toys, auto upholstery, and food packaging.[64] DBP is used primarily in latex adhesives, cellulose plastics, dyes, and cosmetics and other personal care products.[65] The largest use of BBzP is in the production of PVC flooring materials, but it is also used in the manufacture of automotive materials, artificial leather, and food conveyor belts.[66,67]

In 2006, the National Toxicology Program (NTP) concluded that there is "concern" for effects on reproductive tract development in male infants less than one year old exposed to DEHP. In

addition, the NTP also concluded that there is "some concern" (the midpoint on a five-level scale ranging from "negligible" to "serious" concern) [i] for effects on reproductive tract development in male children older than one year old exposed to DEHP, and also that there is "some concern" for effects of prenatal DEHP exposure on reproductive tract development in males. Concern was greater for males exposed to high levels of DEHP in the womb or early in life. These conclusions were based primarily on findings from animal studies, as human data are limited and were determined to be insufficient for evaluating the reproductive effects of DEHP.[64] Some studies have also reported associations of DEHP exposure with increased risk of asthma and bronchial obstruction, increased risk of ADHD symptoms, and altered pregnancy durations.[55,56,58,60,62,63] Human health studies have reported associations between exposures to DBP and altered reproductive hormone levels in newborn boys, and shifts in thyroid hormone levels in pregnant women.[51,68] Signs of feminization in young boys (as measured by reduced anogenital distance), altered hormone levels in newborn boys, and increased risk of rhinitis and eczema are health effects that have been associated with BBzP exposure in some studies.[46,47,51,62] The exposure levels in these studies are comparable to typical exposures in the U.S. population. It is important to note that while the following indicators present data on individual phthalate metabolites, evidence suggests that exposures to multiple phthalates may contribute to common adverse outcomes. The National Research Council has concluded that multiple phthalates may act cumulatively to adversely impact male reproductive development.[4]

Indicator B9 presents median concentrations of metabolites of DEHP, DBP, and BBzP in urine for women ages 16 to 49 years. Indicator B10 presents median metabolite levels of the same phthalates (DEHP, DBP, and BBzP) in urine for children ages 6 to 17 years.

[i] More information on NTP concern levels is available at http://www.niehs.nih.gov/news/media/questions/sya-bpa.cfm.

Indicator B9: Phthalate metabolites in women ages 16 to 49 years: Median concentrations in urine, 1999–2008

Indicator B10: Phthalate metabolites in children ages 6 to 17 years: Median concentrations in urine, 1999–2008

About the Indicators: Indicators B9 and B10 present concentrations of phthalate metabolites in urine of U.S. women ages 16 to 49 years and children ages 6 to 17 years. The data are from a national survey that collects urine specimens from a representative sample of the population every two years, and then measures the concentration of phthalate metabolites in the urine. Indicator B9 presents concentrations of phthalate metabolites in women's urine over time and Indicator B10 presents concentrations of phthalate metabolites in children's urine over time. The focus on both women of child-bearing age and children is based on concern for potential adverse effects in children born to women who have been exposed to phthalates and in children exposed to phthalates.

NHANES

The National Health and Nutrition Examination Survey (NHANES) provides nationally representative biomonitoring data for several phthalates. NHANES is designed to assess the health and nutritional status of the civilian noninstitutionalized U.S. population and is conducted by the National Center for Health Statistics, part of the Centers for Disease Control and Prevention (CDC). Interviews and physical examinations are conducted with approximately 10,000 people in each two-year cycle. CDC's National Center for Environmental Health measures concentrations of environmental chemicals in blood and urine samples collected from NHANES participants. Summaries of the measured values for more than 200 chemicals are provided in the *Fourth National Report on Human Exposure to Environmental Chemicals*.[69]

Phthalate Metabolites

Indicators B9 and B10 present urinary metabolite levels of three important phthalates: di-2-ethylhexyl phthalate (DEHP), dibutyl phthalate (di-n-butyl phthalate and di-isobutyl phthalate) (DBP), and butyl benzyl phthalate (BBzP).

In NHANES and many research studies, biomonitoring of phthalates is conducted by measuring phthalate metabolites in urine rather than the phthalates themselves. This is because phthalates may be present in the sampling and laboratory equipment used to study human exposure levels, and contamination of samples could occur. Also phthalate metabolism is so rapid that the parent phthalate may not appear in urine.[5-8,16,70,71] Furthermore, the phthalate metabolites, and not the parent phthalates, are generally considered to be the biologically active molecules.[5-8,16,72] Unlike other contaminants that have a tendency to accumulate in the human body, phthalates are metabolized and excreted quickly, with elimination half-lives on the order of hours.[5-8,71] Therefore, phthalate metabolites measured in humans are indicative of

recent exposures. All values are reported as micrograms of phthalate metabolites per liter of urine (µg/L).

Concentrations of phthalate metabolites, including those for DEHP, DBP, and BBzP, have been measured in urine from a representative subset of NHANES participants ages 6 and older beginning with the 1999–2000 survey cycle. For DEHP, three metabolites are included: mono-2-ethylhexyl phthalate (MEHP), mono-(2-ethyl-5-oxohexyl) phthalate (MEOHP), and mono-(2-ethyl-5-hydroxyhexyl) phthalate (MEHHP).[ii] The urinary levels of MEHP, MEOHP, and MEHHP are summed together, as is common in phthalates research, to provide a more complete picture of an individual's total DEHP exposure than is given by any individual metabolite.[57,73-75] The primary urinary metabolites of DBP are mono-n-butyl phthalate (MnBP) and mono-isobutyl phthalate (MiBP). The urinary levels of MnBP and MiBP were measured together for the NHANES 1999–2000 survey cycle, but for the following years were measured separately. Indicators B9 and B10 present the combined urinary levels of MnBP and MiBP for each survey cycle. The primary urinary metabolite of BBzP is mono-benzyl phthalate (MBzP).

Calculation of the DEHP metabolite and DBP metabolite indicator values involves summing together separate measured values (3 metabolites of DEHP, and 2 metabolites of DBP in the survey cycles following 1999–2000). If a metabolite included in the sum was not detected in a sample, a default value below the detection limit was assigned for purposes of calculating the summed total.[iii]

In 2007–2008, NHANES collected phthalates biomonitoring data for 2,604 individuals ages 6 years and older, including 571 women ages 16 to 49 years and 690 children ages 6 to 17 years. DEHP metabolites were detected in about 67% of all individuals sampled. The frequency of DEHP metabolites detection was 75% in women ages 16 to 49 years[iv], and 69% in children ages 6 to 17 years. DBP metabolites and BBzP metabolite were detected in 98% of all individuals sampled. The frequency of DBP metabolites detection was 98% in women ages 16 to 49 years, and 99% in children ages 6 to 17 years. The frequency of BBzP metabolite detection was 97% in women ages 16 to 49 years, and 99% in children ages 6 to 17 years. The median and 95th percentile of phthalate levels in urine for all NHANES participants in 2007–2008 were 35 µg/L and 406 µg/L, respectively, for DEHP; 29 µg/L and 147 µg/L, respectively, for DBP; and 12 µg/L and 82 µg/L, respectively, for BBzP. The widespread detection of phthalate metabolites, combined with the fact that phthalates have short half-lives, indicates that phthalate exposure is widespread and relatively continuous.

[ii] A fourth DEHP metabolite, mono(2-ethyl-5-carboxypentyl) phthalate (MECPP), is now measured in NHANES but was not measured prior to 2003–2004. At least one other DEHP metabolite has been measured in laboratory studies but is not measured in NHANES.
[iii] The default value used for non-detect samples is equal to the limit of detection divided by the square root of 2.
[iv] The percentage for women ages 16 to 49 years is calculated with the birth rate adjustment described below.

Individual Variability in Urinary Measurements

NHANES data for phthalates are based on measurements made using a single urine sample for each person surveyed. Due to normal changes in an individual's urinary output throughout the day, this variability in urinary volume, among other factors related to the measurement of chemicals that do not accumulate in the body,[76] may mask differences between individuals in levels of phthalates. Since phthalates do not appear to accumulate in bodily tissues, the distribution of NHANES urinary phthalate levels may overestimate high-end exposures (e.g., at the 95th percentile) as a result of collecting one-time urine samples.[71,77,78] Many studies account for differences in hydration levels by reporting the chemical concentration per gram of creatinine. Creatinine is a byproduct of muscle metabolism that is excreted in urine at a relatively constant rate, independent of the volume of urine, and can in some circumstances partially account for the measurement variability due to changes in urinary output.[79] However, urinary creatinine concentrations differ significantly among different demographic groups, and are strongly associated with an individual's muscle mass, age, sex, diet, health status (specifically renal function), body mass index, and pregnancy status.[80,81] Thus, these indicators present the unadjusted phthalate concentrations so that any observed differences in concentrations between demographic groups are not due to differences in creatinine excretion rates. These unadjusted urinary levels from a single sample may either over- or underestimate urinary levels for a sampled individual. However, for a representative group, it can be expected that a median value based on single samples taken throughout the day will provide a good approximation of the median for that group. Furthermore, due to the large number of subjects surveyed, we expect that differences in the concentrations of phthalates that might be attributed to the volume of the urine sample would average out within and across the various comparison groups.

Birth Rate Adjustment

Indicator B9 uses measurements of phthalate metabolites in urine of women ages 16 to 49 years to represent the distribution of phthalate exposures to women who are pregnant or may become pregnant. However, women of different ages have a different likelihood of giving birth. For example, in 2003–2004, women aged 27 years had a 12% annual probability of giving birth, and women aged 37 years had a 4% annual probability of giving birth.[82] A birth rate-adjusted distribution of women's phthalate metabolite levels is used in calculating this indicator,[v] meaning that the data are weighted using the age-specific probability of a woman giving birth.[83]

[v] There may be multiple ways to implement an adjustment to the data that accounts for birth rates by age. The National Center for Health Statistics has not fully evaluated the method used in ACE, or any other method intended to accomplish the same purpose, and has not used any such method in its publications. NCHS and EPA are working together to further evaluate the birth rate adjustment method used in ACE and alternative methods.

Data Presented in the Indicators

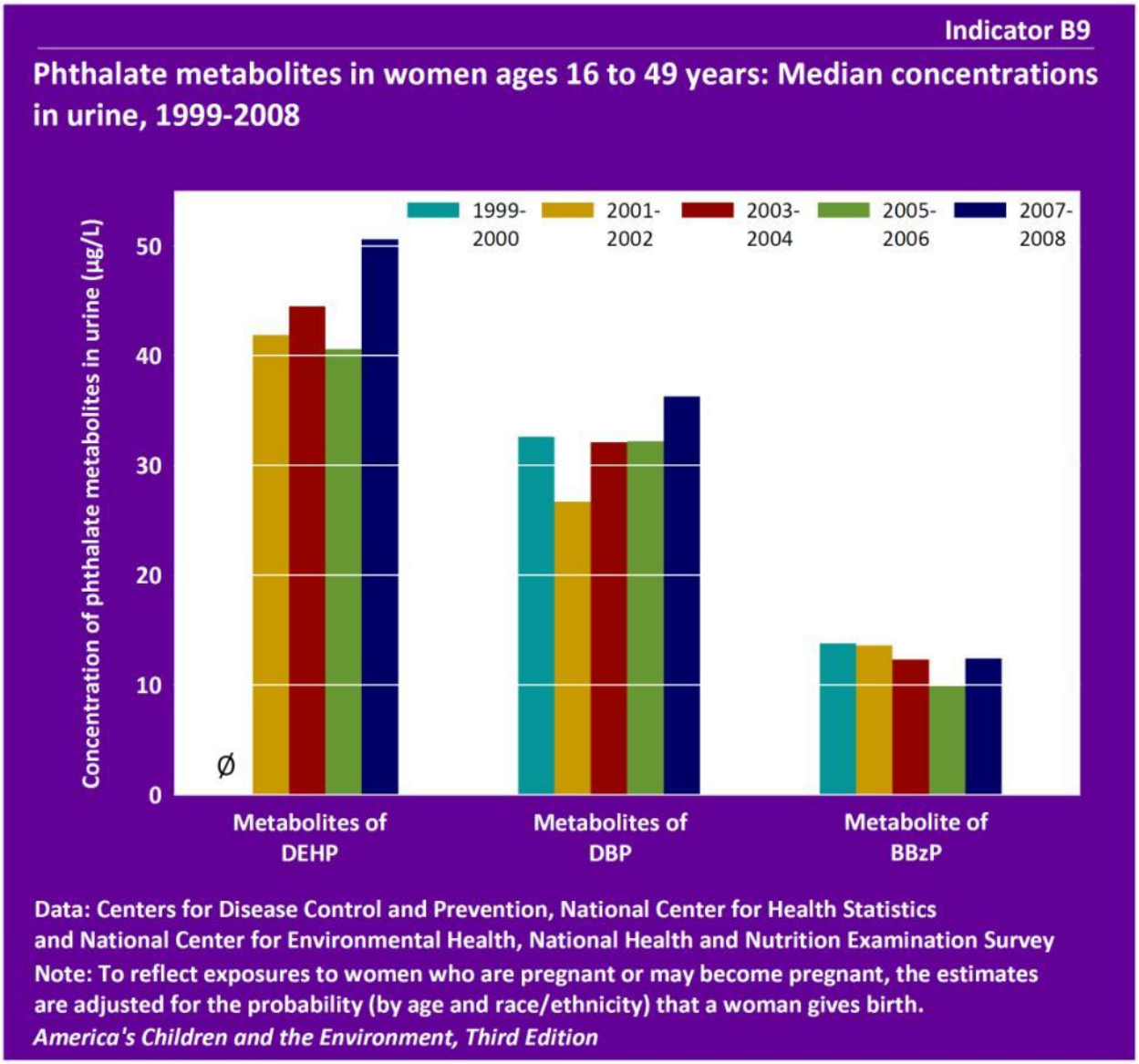
Indicator B9 presents median concentrations of DEHP, DBP, and BBzP metabolites in urine over time for women ages 16 to 49 years, using NHANES data from 1999–2008.

Indicator B10 presents median concentrations of DEHP, DBP, and BBzP metabolites in urine over time for children ages 6 to 17 years, using NHANES data from 1999–2008.

Additional information on the 95th percentile levels of urinary phthalates and how median levels of phthalate metabolites vary by race/ethnicity and family income for women ages 16 to 49 years is presented in the supplemental data tables for this indicator. Data tables also display the 95th percentile levels of phthalate metabolites and how median levels of phthalate metabolites vary by race/ethnicity, family income and age for children ages 6 to 17 years.

NHANES only provides phthalate metabolite data for children ages 6 years and older, which means that the indicator is not able to capture the exposure of premature infants, some of whom may have high levels of phthalate exposure due to the use of medical equipment containing phthalates; or young children, whose play and mouthing behaviors may increase their exposure to phthalates in toys and house dust.

Please see the Introduction to the Biomonitoring section for an explanation of the terms "median" and "95th percentile," a description of the race/ethnicity and income groups used in the ACE3 biomonitoring indicators, and information on the statistical significance testing applied to these indicators.



Ø The estimate is not reported because the metabolites MEOHP and MEHHP were not measured in 1999–2000.

Data characterization

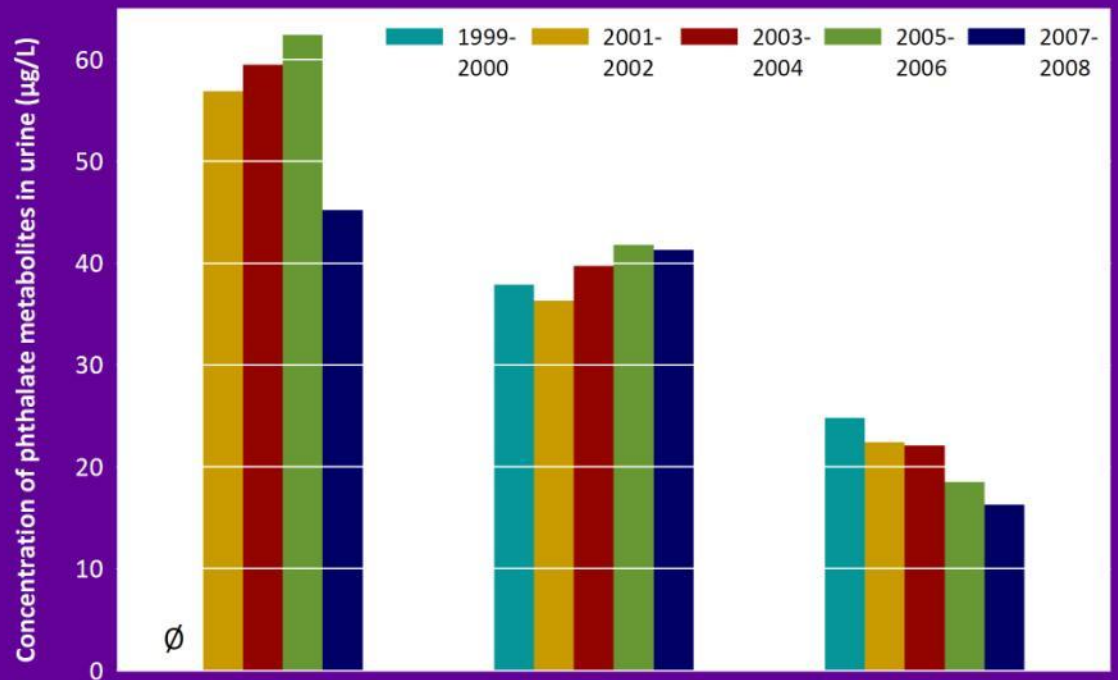
- Data for this indicator are obtained from an ongoing continuous survey conducted by the National Center for Health Statistics.
- Survey data are representative of the U.S. civilian noninstitutionalized population.
- Phthalate metabolites are measured in urine samples obtained from individual survey participants.

- From 2001–2002 to 2007–2008, the median level of DEHP metabolites in urine of women ages 16 to 49 years varied between 41 µg/L and 51 µg/L, and was 51 µg/L in 2007–2008. There was no statistically significant trend in median DEHP metabolites over 2001–2002 to 2007–2008.

- From 1999–2000 to 2007–2008, the median level of DBP metabolites in urine of women ages 16 to 49 years varied between 27 µg/L and 36 µg/L, and was 36 µg/L in 2007–2008.
- From 1999–2000 to 2007–2008, the median level of BBzP metabolites in urine of women ages 16 to 49 years varied between 10 µg/L and 14 µg/L, and was 12 µg/L in 2007–2008.
- From 2001–2002 to 2007–2008, the concentration of DEHP metabolites in the 95th percentile varied between 462 µg/L and 578 µg/L, and was 567 µg/L in 2007–2008. There was an increasing trend in the 95th percentile concentration of DBP metabolites in women of child-bearing age, from 128 µg/L in 2001–2002 to 160 µg/L in 2007–2008. From 1999–2000 and 2007–2008, the concentration of BBzP metabolite varied between 68 µg/L and 100 µg/L, and was 70 µg/L in 2007–2008. (See Table B9a.)
  - The increasing trend for DBP metabolites at the 95th percentile from 2001–2002 to 2007–2008 was statistically significant after accounting for differences by age, race/ethnicity, and income.
- The concentrations of DEHP metabolites in the 95th percentile ranged from 10 to 14 times higher than the median levels presented in this graph. The concentrations of DBP metabolites and BBzP metabolite in urine at the 95th percentile ranged from 4 to 7 times higher than the median levels presented in this graph. (See Table B9 and B9a.)
- For the years 2005–2008, Black non-Hispanic women of child-bearing age had higher median concentrations of all the phthalate metabolites shown here compared with White non-Hispanic women, Mexican-American women, and women of "All Other Races/Ethnicities," although these differences were frequently not statistically significant. (See Table B9b.)
- Median levels of urinary phthalate metabolites varied by family income. Women living below the poverty level had higher concentrations of phthalate metabolites in their urine compared with women living at or above the poverty level. (See Table B9b.)
  - The difference between income groups was statistically significant for the DBP metabolites after accounting for differences by race/ethnicity or age profile above and below poverty. The difference between income groups for the BBzP metabolite was statistically significant before accounting for race/ethnicity and age. The difference between income groups was not statistically significant for the DEHP metabolites.

Indicator B10

Phthalate metabolites in children ages 6 to 17 years: Median concentrations in urine, 1999-2008



Data: Centers for Disease Control and Prevention, National Center for Health Statistics and National Center for Environmental Health, National Health and Nutrition Examination Survey

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Ø The estimate is not reported because the metabolites MEOHP and MEHHP were not measured in 1999–2000.

Data characterization

- Data for this indicator are obtained from an ongoing continuous survey conducted by the National Center for Health Statistics.
- Survey data are representative of the U.S. civilian noninstitutionalized population.
- Phthalate metabolites are measured in urine samples obtained from individual survey participants.

- From 2001–2002 to 2007–2008, the median level of DEHP metabolites in urine of children ages 6 to 17 years varied between 45 µg/L and 62 µg/L, and was 45 µg/L in 2007–2008.
- From 1999–2000 to 2007–2008, the median level of DBP metabolites in urine of children ages 6 to 17 years varied between 36 µg/L and 42 µg/L, and was 41 µg/L in 2007–2008.

- The median level of BBzP metabolite in urine of children ages 6 to 17 years decreased from 25 µg/L in 1999–2000 to 16 µg/L in 2007–2008. This decreasing trend was statistically significant.
- At the 95th percentile, there was an increasing trend in the concentration of DEHP metabolites in children, from 387 µg/L in 1999-2000 to 564 µg/L in 2007–2008. From 1999–2000 to 2007–2008, the concentration of DBP metabolites varied between 166 µg/L and 191 µg/L, and was 191 µg/L in 2007–2008. The concentration of BBzP metabolites varied between 104 µg/L and 151 µg/L, and was 107 µg/L in 2007–2008. (See Table B10a.)
  - The increasing trend for DEHP metabolites from 1999–2000 to 2007–2008 was statistically significant.
- Among children ages 6 to 17 years, the concentration of DEHP metabolites in urine at the 95th percentile ranged from 7 to 12 times higher than the median levels presented in this graph. The concentrations of metabolites of DBP and BBzP in the 95th percentile ranged from 4 to 7 times higher than the median levels. (See Table B10 and B10a.)
- Children living below the poverty level had higher median concentrations of DBP metabolites detected in their urine compared with children living at or above the poverty level. Median concentrations of DEHP metabolites and BBzP metabolite were similar among children living below the poverty level and children living at or above the poverty level. (See Table B10b.)
  - The difference between income groups for DBP metabolites was statistically significant.
- For the years 2005–2008, Mexican-American children had lower median concentrations of all the phthalate metabolites shown here compared with White non-Hispanic children and Black non-Hispanic children. (See Table B10b.)
  - Testing for differences by race/ethnicity found that Mexican-American children had statistically significantly lower median concentrations of phthalate metabolites as follows: for DEHP and BBzP, lower than both White non-Hispanic and Black non-Hispanic children; for DBP, lower than Black non-Hispanic children, and lower than White non-Hispanic children after accounting for differences in age, sex, and income.
- Children ages 6 to 10 years had higher median levels of phthalate metabolites in their urine compared to adolescents ages 16-17 years. These differences were relatively small for DEHP metabolites and DBP metabolites but greater for BBzP metabolite. (See Table B10c.)
  - The age group differences for BBzP were statistically significant.

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Bisphenol A

Bisphenol A (BPA) is a high-volume industrial chemical used in the production of epoxy resins and polycarbonate plastics. Polycarbonate plastics may be encountered in many products, notably food and drink containers, while epoxy resins are frequently used as inner liners of metallic food and drink containers to prevent corrosion. The use of BPA in food contact materials is regulated by the U.S. Food and Drug Administration.[1,2] BPA also serves as a coating on some types of thermal paper that are often used as receipts from cash registers, automatic teller machines, and other similar devices. It is used in the polyvinyl chloride (PVC) industries as well as in metal foundries where it is used to make casts and moldings. The primary route of human exposure to BPA is believed to be through diet, when BPA migrates from food and drink containers.[3-5] Migration is more likely to occur when the container is heated or washed.[5,6] Other possible sources of BPA exposure include air, dust, water, and dental sealants.[3,5]

Biomonitoring studies demonstrate that BPA exposure is prevalent in the United States, with detectable levels of BPA present in 93% of tested urine samples.[7] Because BPA is metabolized quickly in the body,[8] the high frequency of detection indicates that exposures are occurring regularly within the U.S. population. Exposures to BPA of infants and children up to age 6 years are estimated to be greater than BPA exposures in older children and adults.[3]

Much of the scientific interest in BPA is related to published research suggesting that BPA may be an endocrine disrupting chemical.[9,10] Endocrine disruptors act by interfering with the biosynthesis, secretion, action, or metabolism of naturally occurring hormones.[9,11] Given the importance of hormones in human physiology, there is concern in the scientific community over the potential for endocrine disruptors to adversely affect children's health, particularly in reproduction, early and adolescent development, and behavior.[9] BPA is described as a "weakly estrogenic" chemical, because its affinity for binding to estrogen receptors is approximately 10,000-fold weaker than natural estrogen.[12]

Recent attention to the developmental effects of BPA is based on several laboratory studies and a better understanding of the mechanisms by which BPA exerts an estrogenic effect.[10,13-15] In animal studies, exposure to high levels of BPA during pregnancy or lactation resulted in reduced birth weight, slowed growth, reduced survival, and delayed time to the onset of puberty in offspring.[16-19] Animal studies have also found that low-dose BPA exposure was associated with insulin resistance.[20,21] In addition, one study found that low-dose BPA exposure in pregnant animals was associated with symptoms similar to gestational diabetes, suggesting that BPA exposures may have adverse effects in pregnant women.[22] Other studies have found relationships between prenatal or early-life BPA exposure and neurological effects as well as the development of breast and prostate cancer in adult animals.[23-25] The effects of low-dose exposure to BPA in lab animals are debated within the scientific community, with some researchers finding no developmental effects, while others have identified behavioral and neural effects, abnormal urinary tract development, development of lesions in the prostate gland, and early onset of puberty in females.[3,23,26-36] Differences in reported results on the

timing of puberty between low and high dose studies may be a result of dose differences, study design, or species of animal.[3] Based on a critical review of the existing scientific literature, in 2008 the National Toxicology Program (NTP) determined that there was "some concern" (the midpoint on a five-level scale ranging from "negligible" to "serious")[i] for effects of BPA on the brain, behavior, and prostate gland in fetuses, infants, and children; "minimal concern" for effects on the mammary gland and onset of puberty in females; and "negligible concern" for fetal or neonatal mortality, birth defects, or reduced birth weight and growth.[3]

Epidemiological data on the effects of BPA in human populations are limited. Studies of the U.S. general population have reported that adults with higher recent BPA exposure (as represented by urinary BPA concentrations) are more likely to have coronary heart disease, diabetes, immune dysfunction, and liver enzyme abnormalities.[37-39] Some of these associations are postulated to be due to non-estrogenic effects of BPA,[38] although there is limited understanding of the mechanisms by which BPA exposure may lead to an adverse health effect. Studies of workers in China reported an association between exposure to high levels of BPA and an increased risk of self-reported sexual dysfunction,[40,41] and that BPA exposure to pregnant workers was associated with decreased offspring birthweight.[42] A study of children in Ohio reported an association between prenatal BPA exposure, at levels typical for the general population, and aggression and hyperactivity in 2-year-old children.[43] Similar associations between behavioral effects and BPA exposure have been seen in animal studies.[3,44,45] However, another study of prenatal BPA exposure conducted in New York City found no association between prenatal BPA exposure and social behavior deficits in children at ages 7 to 9 years.[46] In 2009, the National Institutes of Health announced that it would spend $30 million over two years to better understand the link between low-dose BPA exposure and human health effects.

Studies have shown that detectable levels of BPA are present in human urine samples from all age groups including infants, toddlers, children and adults.[3,47-52] BPA has been identified in the blood of pregnant women,[53] and also can cross the placenta, potentially exposing the fetus.[54] Previous studies have identified higher levels of BPA in the urine of children ages 6 to 11 years compared with adults,[47,49,50] and found that consumption of soda and school lunches was also associated with higher urinary BPA concentrations.[50] Infants and young children also have a higher estimated daily intake of BPA compared with adults.[3,48] Although less information is available on BPA levels in infants than in older children, one study found that premature infants in intensive care units had greater urinary BPA concentrations than those observed in other infants or even older children, though the route of exposure for the premature infants is unclear.[55] Some laboratory animal studies have found that younger animals are less effective at metabolizing BPA than older animals are; while it has been proposed that such findings may apply to human infants and developing fetuses, this hypothesis is debated in the scientific literature.[3,36,52,56-61] One important part of ongoing research is to better understand how BPA is

[i] More information on NTP concern levels is available at http://www.niehs.nih.gov/news/media/questions/sya-bpa.cfm.

absorbed, distributed, metabolized, and excreted by the body, and how those processes change with age and with route of exposure.[56-58,60-62] Interpretation of these data will allow us to understand how environmental exposure equates to the internal dose routinely measured in biomonitoring studies.

The two indicators that follow use the best nationally representative data currently available on urinary BPA levels over time for women of child-bearing age and children. Indicator B11 presents median and 95th percentile concentrations of BPA in urine for women ages 16 to 49 years. Indicator B12 presents median and 95th percentile concentrations of BPA in urine for children ages 6 to 17 years.

Indicator B11: Bisphenol A in women ages 16 to 49 years: Median and 95th percentile concentrations in urine, 2003–2010

Indicator B12: Bisphenol A in children ages 6 to 17 years: Median and 95th percentile concentrations in urine, 2003–2010

About the Indicators: Indicators B11 and B12 present concentrations of bisphenol A (BPA) in urine of U.S. women ages 16 to 49 years and children ages 6 to 17 years. The data are from a national survey that collects urine specimens from a representative sample of the population every two years, and then measures the concentration of total BPA in the urine. Indicator B11 presents concentrations of BPA in women's urine over time and Indicator B12 presents concentrations of BPA in children's urine over time. The focus on both women of child-bearing age and children is based on concern for potential adverse effects in children born to women who have been exposed to BPA and in children exposed to BPA.

NHANES

The National Health and Nutrition Examination Survey (NHANES) provides nationally representative biomonitoring data for BPA. NHANES is designed to assess the health and nutritional status of the civilian noninstitutionalized U.S. population and is conducted by the National Center for Health Statistics, part of the Centers for Disease Control and Prevention (CDC). Interviews and physical examinations are conducted with approximately 10,000 people in each two-year survey cycle. CDC's National Center for Environmental Health measures concentrations of environmental chemicals in blood and urine samples collected from NHANES participants. Summaries of the measured values for more than 200 chemicals are provided in the *Fourth National Report on Human Exposure to Environmental Chemicals*.[63]

Bisphenol A and its Metabolites

Indicators B11 and B12 present urinary levels of BPA in women of child-bearing age and children. The reported measurements of BPA in urine represent "total BPA," which includes both free BPA and non-estrogenic metabolites of BPA (only free BPA is considered active based on measures of estrogenicity). Measured levels in the U.S. population may be composed predominantly of these metabolites,[64] but total BPA levels reflect previous exposure to the biologically active form of BPA and there is debate in the scientific community over the potential for conversion of non-estrogenic metabolites back to free BPA in various tissues.[65] Recent work has also highlighted the potential for conversion of non-estrogenic metabolites of BPA to the active form when crossing the placenta, increasing the relevance of total BPA measurements to children's health.[54,66] All values are reported as micrograms of BPA per liter of urine (µg/L).

Concentrations of BPA in urine have been measured in a representative subset of NHANES participants ages 6 years and older beginning with the 2003–2004 survey cycle. In 2009–2010, NHANES collected BPA biomonitoring data for 2,749 individuals ages 6 years and older, including 608 women ages 16 to 49 years and 727 children ages 6 to 17 years. BPA was detected in about 90% of all individuals sampled. The frequency of BPA detection was 92% in women ages 16 to 49 years,[ii] and 92% in children ages 6 to 17 years. The median and 95th percentile BPA levels in urine for all NHANES participants in 2009–2010 were 2 µg/L and 10 µg/L, respectively. The widespread detection of BPA, combined with the fact that BPA has a short half-life, indicates that BPA exposure is widespread and relatively continuous.

Individual Variability in Urinary Measurements

NHANES data for BPA are based on measurements made using a single urine sample for each person surveyed. Due to normal changes in an individual's urinary output throughout the day, this variability in urinary volume, among other factors related to the measurement of chemicals that do not accumulate in the body,[67] may mask differences between individuals in levels of BPA. Since BPA does not appear to accumulate in bodily tissues, the distribution of NHANES urinary BPA levels may overestimate high-end exposures (e.g., at the 95th percentile) as a result of collecting one-time urine samples.[8,68,69] Many studies account for differences in hydration levels by reporting the chemical concentration per gram of creatinine. Creatinine is a byproduct of muscle metabolism that is excreted in urine at a relatively constant rate, independent of the volume of urine, and can in some circumstances partially account for the measurement variability due to changes in urinary output.[70] However, urinary creatinine concentrations differ significantly among different demographic groups, and are strongly associated with an individual's muscle mass, age, sex, diet, health status (specifically renal function), body mass index, and pregnancy status.[71,72] Thus, these indicators present the unadjusted BPA concentrations so that any observed differences in concentrations between demographic groups are not due to differences in creatinine excretion rates. These unadjusted urinary levels from a single sample may either over- or underestimate urinary levels for a sampled individual. However, for a representative group, it can be expected that a median value based on single samples taken throughout the day will provide a good approximation of the median for that group. Furthermore, due to the large number of subjects surveyed, we expect that differences in the concentrations of BPA that might be attributed to the volume of the urine sample would average out within and across the various comparison groups.

Birth Rate Adjustment

Indicator B11 uses measurements of BPA in urine of women ages 16 to 49 years to represent the distribution of BPA exposures to women who are pregnant or may become pregnant. However, women of different ages have a different likelihood of giving birth. For example, in

[ii] The percentage for women ages 16 to 49 years is calculated with the birth rate adjustment described below.

2003–2004, women aged 27 had a 12% probability of giving birth, and women aged 37 had a 4% probability of giving birth.[73] A birth rate-adjusted distribution of women's BPA levels is used in calculating this indicator,[iii] meaning that the data are weighted using the age-specific probability of a woman giving birth.[74]

Data Presented in the Indicators

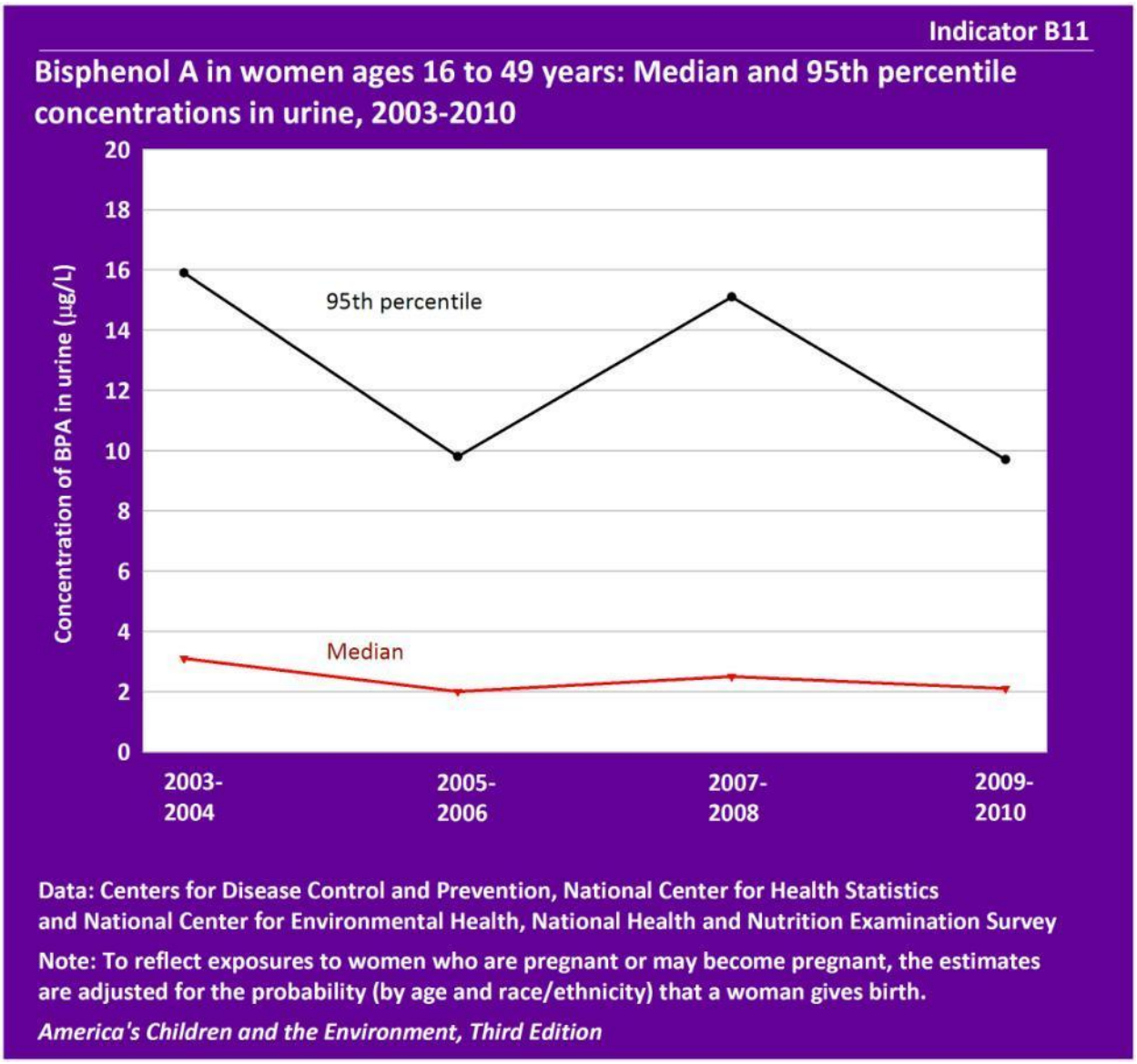
Indicators B11 presents median and 95th percentile concentrations of BPA in urine over time for women ages 16 to 49 years, using NHANES data from 2003–2010.

Indicator B12 presents median and 95th percentile concentrations of BPA in urine over time for children ages 6 to 17 years, using NHANES data from 2003–2010.

Additional information showing how the median and 95th percentile levels of BPA in urine vary by race/ethnicity and family income for women ages 16 to 49 years is presented in supplemental data tables for these indicators. Data tables also display information showing how the median and 95th percentile levels of BPA in urine vary by race/ethnicity, family income, and age for children ages 6 to 17 years.

Please see the Introduction to the Biomonitoring section for an explanation of the terms "median" and "95th percentile," a description of the race/ethnicity and income groups used in the ACE3 biomonitoring indicators, and information on the statistical significance testing applied to these indicators.

[iii] There may be multiple ways to implement an adjustment to the data that accounts for birth rates by age. The National Center for Health Statistics has not fully evaluated the method used in ACE, or any other method intended to accomplish the same purpose, and has not used any such method in its publications. NCHS and EPA are working together to further evaluate the birth rate adjustment method used in ACE and alternative methods.

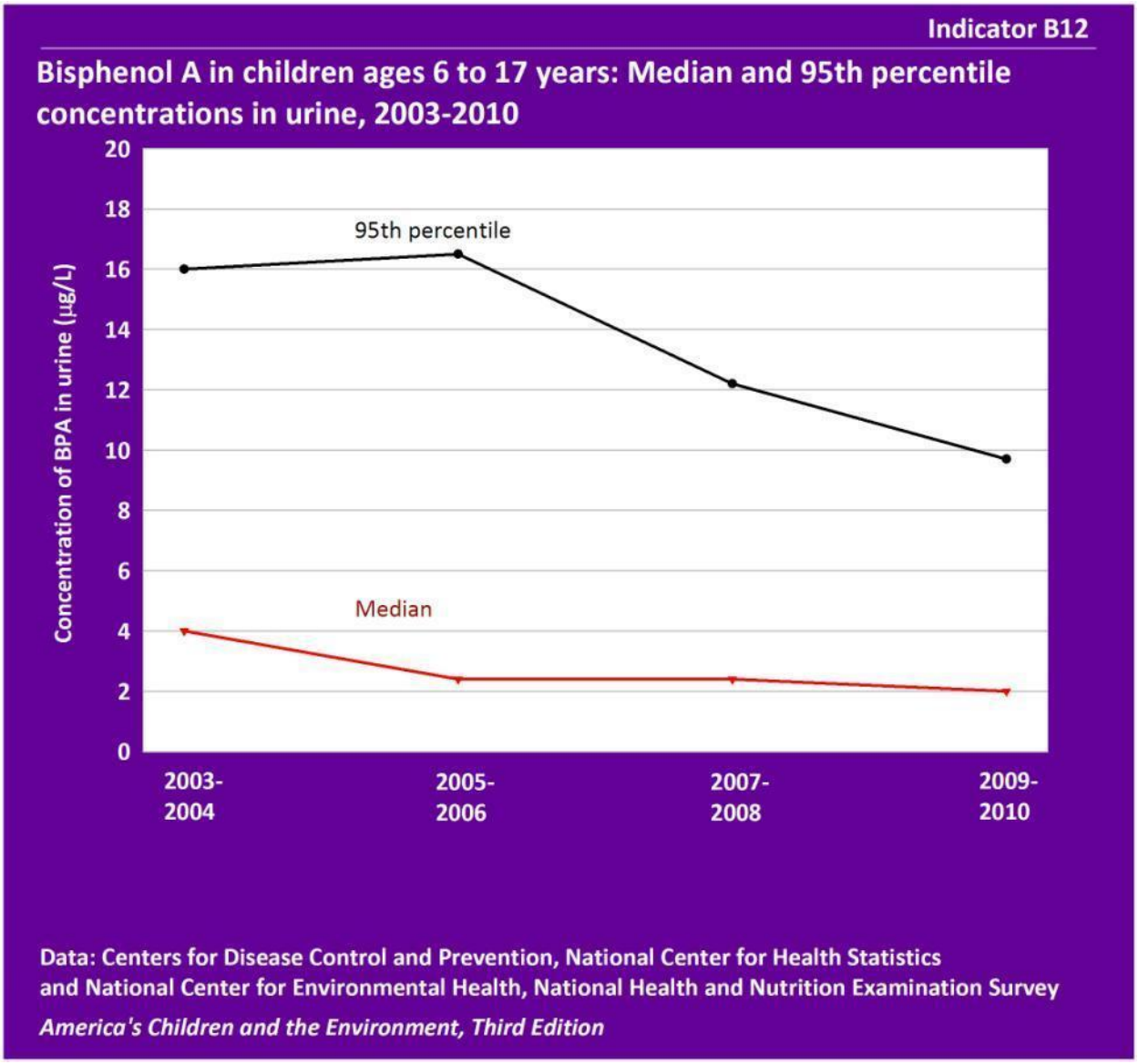


Data characterization

- Data for this indicator are obtained from an ongoing continuous survey conducted by the National Center for Health Statistics.
- Survey data are representative of the U.S. civilian noninstitutionalized population.
- BPA is measured in urine samples obtained from individual survey participants.

- From 2003–2004 to 2009–2010, the median concentration of BPA in urine among women ages 16 to 49 years varied between2 µg/L and 3 µg/L. There was no statistically significant trend in median BPA levels over the years shown.

- From 2003–2004 to 2009–2010, the concentrations of BPA in urine at the 95th percentile varied between 10 µg/L and 16 µg/L, and was 10 µg/L in 2009–2010. There was no statistically significant trend in 95th percentile concentrations of BPA over the years shown.
- Between 2003–2004 and 2009–2010, the concentrations of BPA in the 95th percentile ranged from 5 to 6 times the median levels for women ages 16 to 49 years.
- In 2007–2010, the median concentration of BPA in urine of Black non-Hispanic women was about 4 µg/L, which was higher than the median concentrations in White non-Hispanic women, Mexican-American women, and women of "All Other Races/Ethnicities." The differences between Black non-Hispanic women and women in other race/ethnicity groups were statistically significant. (See Table B11a.)
  Women living below the poverty level had higher median concentrations of BPA in urine than those living at or above poverty level, a difference that was statistically significant. (See Table B11a.)
- Among White non-Hispanic women and women of "All Other Races/Ethnicities," those with family incomes below poverty level had higher median concentrations of BPA in urine than those at or above poverty level. The differences between the income groups were statistically significant. (See Table B11a.)
- Higher concentrations of BPA were observed in the urine of women below the poverty level at the 95th percentile (15 µg/L) compared with women at or above the poverty level (11 µg/L). This difference was statistically significant after adjustment for differences in age and race/ethnicity. (See Table B11b.)
- White non-Hispanic women at the 95th percentile (10 µg/L) had lower concentrations of BPA in urine than Black non-Hispanic women (15 µg/L) and Mexican-American women (15 µg/L). These differences were statistically significant after adjustment for differences in age and income. (See Table B11b.)



Data characterization

- Data for this indicator are obtained from an ongoing continuous survey conducted by the National Center for Health Statistics.
- Survey data are representative of the U.S. civilian noninstitutionalized population.
- BPA is measured in urine samples obtained from individual survey participants.

- Among children ages 6 to 17 years, the median concentration of BPA in urine of children ages 6 to 17 years decreased from 4 µg/L in 2003–2004 to 2 µg/L in 2009–2010. The concentration of BPA in urine at the 95th percentile decreased from 16 µg/L in 2003–2004 to 10 µg/L in 2009–2010. These decreasing trends were statistically significant.
- Between 2003–2004 and 2009–2010, the concentrations of BPA in the 95th percentile ranged from 4 to 7 times the median levels for children ages 6 to 17 years.

- In 2007–2010, median concentrations of BPA in urine of Black non-Hispanic children ages 6 to 17 years were higher than in White non-Hispanic children, Mexican-American children, and children of "All Other Races/Ethnicities." These differences were statistically significant. (See Table B12a.)
- BPA concentrations at the 95th percentile were similar for Black non-Hispanic, White-non Hispanic, and Mexican-American children ages 6 to 17 years in 2007–2010. (See Table B12b.)
- In 2007–2010, BPA concentrations were similar for age groups 6 to 10 years, 11 to 15 years, and 16 to 17 years, both at the median and at the 95th percentile. (See Table B12c.)

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Perchlorate

Perchlorate is a naturally occurring and man-made chemical that is used to manufacture fireworks, explosives, flares, and rocket fuel.[1,2] It is found naturally in groundwater and soils throughout many regions in the United States and other arid regions of the world.[3,4] Perchlorate is presumed to migrate into groundwater during the process of irrigation,[5] and has also been found in groundwater supplies near military and industrial facilities where perchlorate was used.[6] Perchlorate has been detected in surface water; dairy products; and in some food crops, including lettuce, spinach, grapes, carrots, tomatoes, and other fruits and vegetables, produced in the United States and internationally.[5,7-12] Perchlorate has been detected in some fertilizers produced in Chile; however, fertilizers appear to be a negligible source of perchlorate in the United States.[1,13-17] The numerous sources of perchlorate located across the United States result in widespread exposures of perchlorate to the U.S. population.[3,4]

Perchlorate has been detected in human breast milk, urine, blood, amniotic fluid, and saliva.[18-23] A national study representative of the U.S. population ages 6 years and older found perchlorate in the urine of 100% of the more than 5,000 people sampled; children had higher median urinary levels compared with those of adults, including women of child-bearing age.[3,24] Infants are exposed to perchlorate through both breast milk and formula, but those who are fed breast milk may have higher exposures to perchlorate compared with those who are fed cow- or soy-based formula.[25] When comparing perchlorate doses (daily intakes per kilogram of body weight, estimated from urine samples), infants less than 2 months of age experience higher perchlorate doses compared with older infants, and estimated doses of perchlorate in infants are more than twice as high as estimated doses for adults.[3,25,26] A study conducted in China found that blood samples of infants less than 1 year of age have higher mean perchlorate values than blood samples of both older children and adults.[27,28]

Children might be directly exposed to perchlorate through perchlorate-contaminated water and foods containing perchlorate. Surveys conducted by the U.S. Food and Drug Administration have detected varying levels of perchlorate in many foods that may be consumed by both women and young children. The surveys, conducted in 22 states, tested 27 different types of food products and found the highest levels of perchlorate in spinach and tomatoes.[12] Some infant formulas have also been found to contain perchlorate, and the perchlorate content of the formula is increased if it is prepared with perchlorate-contaminated water.[29-32] However, computer modeling studies have concluded that exposure to perchlorate from food consumption is much greater than exposure from drinking water in the United States.[33,34] These modeled predictions are consistent with empirical studies that attribute the majority of perchlorate intake dose in U.S. residents to food consumption.[8,35-37]

Exposure to high doses of perchlorate has been shown to block the uptake of iodide into the thyroid gland.[38,39] Exposure to perchlorate and other thyroid-disrupting chemicals is of particular concern for women of child-bearing age, because thyroid hormones are important for growth and development of the central nervous system in fetuses and infants.[1,40-42] The

transfer of iodide from blood into the thyroid gland is an essential step in the synthesis of thyroid hormones that regulate how the body uses energy; influence bone growth; and influence the development of the brain, reproductive, and cardiovascular systems.[43] When this transfer of iodide into the thyroid gland is blocked, the thyroid may not have enough iodide to make thyroid hormones. Reduction in a woman's thyroid hormone levels during the first and second trimester puts the fetus at risk for impaired physical and mental development, with the severity of the impairment depending upon the degree of hormone deficiency.[40,41] Moderate deficits in maternal thyroid hormone levels during early pregnancy have been linked to reduced childhood IQ scores and other neurodevelopmental effects, as well as unsuccessful or complicated pregnancies.[44] Prenatal and newborn hypothyroidism (low thyroid hormone levels) is a risk factor for intellectual disability (mental retardation) and other forms of impaired neurodevelopment.[45] In 2005–2008, approximately 38% of women ages 15 to 44 years in the United States had insufficient iodine intake,[46] potentially increasing the risk for effects on fetal development from exposure to perchlorate.[1]

Associations between perchlorate exposure and thyroid hormones have been based on both epidemiological and animal-based studies. Animal studies have shown that exposure to high doses of perchlorate result in decreased thyroid hormone levels and physical alterations to the thyroid gland,[1] and have also found that these effects of perchlorate can be enhanced with exposure to other chemicals that block uptake of iodide.[47] In 2005, the National Research Council (NRC) concluded that the available epidemiological evidence concerning non-medical exposure to perchlorate did not indicate an association with thyroid disorders in adults or infants, and was inadequate for assessing the potential for adverse associations between prenatal perchlorate exposure and adverse neurodevelopmental outcomes in children.[1] The NRC also indicated that there was a lack of studies to evaluate potential effects of prenatal perchlorate exposures in infants and children, particularly in vulnerable populations.[1]

Some further epidemiological research has been conducted since the NRC report was completed. A study of urinary perchlorate and thyroid hormone levels in more than 11,000 U.S. females ages 12 years and older in 2001–2002 found that increasing levels of perchlorate in urine were associated with decreased thyroid hormone levels.[26] Further analysis of this data set found that tobacco smoke and perchlorate may interact to affect thyroid function at commonly occurring perchlorate levels.[48] In contrast, a study of first-trimester pregnant women identified as iodine-deficient, and a long-term exposure study of women in early pregnancy and late pregnancy in Chile, found that exposure to low levels of perchlorate did not result in decreased levels of thyroid hormones.[49,50]

Other studies have evaluated relationships between drinking water perchlorate levels and thyroid hormone levels in newborns. A study of California infants born in 1998 reported that babies born to mothers in communities with higher drinking water perchlorate levels were more likely to have elevated levels of thyroid stimulating hormone, which is an indication of reduced thyroid hormone levels.[51] An earlier study of the same population and other studies have not found associations between drinking water perchlorate levels and neonatal thyroid hormone function.[50,52-56]

In January 2009, EPA issued an interim health advisory level to help state and local officials manage local perchlorate contamination issues in a health-protective manner, in advance of a final EPA regulatory determination.[2,57] In February 2011, EPA decided to develop a federal drinking water standard for perchlorate, based on the concern for effects on thyroid hormones and the development and growth of fetuses, infants, and children.[2,58] The process for developing the standard will include receiving input from key stakeholders as well as submitting any formal rule to a public comment process. California and Massachusetts have both set their own standards for perchlorate in drinking water.[59] No standards exist for perchlorate in food.

The indicator that follows uses the best nationally representative data currently available on urine perchlorate levels over time for women of child-bearing age. Indicator B13 presents median and 95th percentile urinary perchlorate levels for women ages 16 to 49 years.

Indicator B13: Perchlorate in women ages 16 to 49 years: Median and 95th percentile concentrations in urine, 2001–2008

About the Indicators: Indicator B13 presents concentrations of perchlorate in urine of U.S. women ages 16 to 49 years. The data are from a national survey that collects urine specimens from a representative sample of the population every two years, and then measures the concentration of perchlorate in the urine. The indicator presents concentrations of perchlorate in urine over time. The focus on women of child-bearing age is based on concern for potential adverse effects in children born to women who have been exposed to perchlorate.

NHANES

The National Health and Nutrition Examination Survey (NHANES) provides nationally representative biomonitoring data for perchlorate. NHANES is designed to assess the health and nutritional status of the civilian noninstitutionalized U.S. population and is conducted by the National Center for Health Statistics, part of the Centers for Disease Control and Prevention (CDC). Interviews and physical examinations are conducted with approximately 10,000 people in each two-year cycle. CDC's National Center for Environmental Health measures concentrations of environmental chemicals in blood and urine samples collected from NHANES participants. Summaries of the measured values for more than 200 chemicals are provided in the *Fourth National Report on Human Exposure to Environmental Chemicals*.[24]

Perchlorate

Indicator B13 presents urinary levels of perchlorate in women of child-bearing age. Perchlorate passes quickly through the body unchanged and is excreted in urine, with an elimination half-life on the order of hours.[3] Therefore, perchlorate measured in humans is indicative of recent exposures. All values are reported as micrograms of perchlorate per liter of urine (µg/L).

Concentrations of perchlorate in urine have been measured in a representative subset of NHANES participants ages 6 years and older in 2001–2002 and 2003–2004, and in all NHANES participants ages 6 years and older in 2005–2006 and 2007–2008.[19]

For 2007-2008, NHANES collected perchlorate biomonitoring data for 7,629 individuals ages 6 years and older, including 1,608 women ages 16 to 49 years. Perchlorate was detected in 100% of the individuals sampled in NHANES 2007-2008. The median and 95th percentile of urinary perchlorate levels for all NHANES participants in 2007-2008 were 4 µg/L and 17 µg/L, respectively. The widespread detection of perchlorate, combined with the fact that perchlorate has a short half-life, indicates that perchlorate exposure is widespread and relatively continuous.

Individual Variability in Urinary Measurements

NHANES data for perchlorate are based on measurements made using a single urine sample for each person surveyed. Due to normal changes in an individual's urinary output throughout the day, this variability in urinary volume, among other factors related to the measurement of chemicals that do not accumulate in the body,[60] may mask differences between individuals in levels of perchlorate. Since perchlorate does not appear to accumulate in bodily tissues, the distribution of NHANES urinary perchlorate levels may over estimate high-end exposures (e.g., at the 95th percentile) as a result of collecting one-time urine samples.[34,61,62] Many studies account for differences in hydration levels by reporting the chemical concentration per gram of creatinine. Creatinine is a byproduct of muscle metabolism that is excreted in urine at a relatively constant rate, independent of the volume of urine, and can in some circumstances partially account for the measurement variability due to changes in urinary output.[63] However, urinary creatinine concentrations differ significantly among different demographic groups, and are strongly associated with an individual's muscle mass, age, sex, diet, health status (specifically renal function), body mass index, and pregnancy status.[64,65] Thus, this indicator presents the unadjusted perchlorate concentrations so that any observed differences in concentrations between demographic groups are not due to differences in creatinine excretion rates. These unadjusted urinary levels from a single sample may either over- or underestimate urinary levels for a sampled individual. However, for a representative group, it can be expected that a median value based on single samples taken throughout the day will provide a good approximation of the median for that group. Furthermore, due to the large number of subjects surveyed, we expect that differences in the concentrations of perchlorate that might be attributed to the volume of the urine sample would average out within and across the various comparison groups.

Birth Rate Adjustment

Indicator B13 uses measurements of perchlorate in urine of women ages 16 to 49 years to reflect the potential distribution of perchlorate exposures to women who are pregnant or may become pregnant. However, women of different ages have a different likelihood of giving birth. For example, in 2003–2004, women aged 27 years had a 12% annual probability of giving birth, and women aged 37 years had a 4% annual probability of giving birth.[66] A birth rate-adjusted distribution of women's perchlorate levels is used in calculating this indicator,[i] meaning that the data are weighted using the age-specific probability of a woman giving birth.[67]

[i] There may be multiple ways to implement an adjustment to the data that accounts for birth rates by age. The National Center for Health Statistics has not fully evaluated the method used in ACE, or any other method intended to accomplish the same purpose, and has not used any such method in its publications. NCHS and EPA are working together to further evaluate the birth rate adjustment method used in ACE and alternative methods.

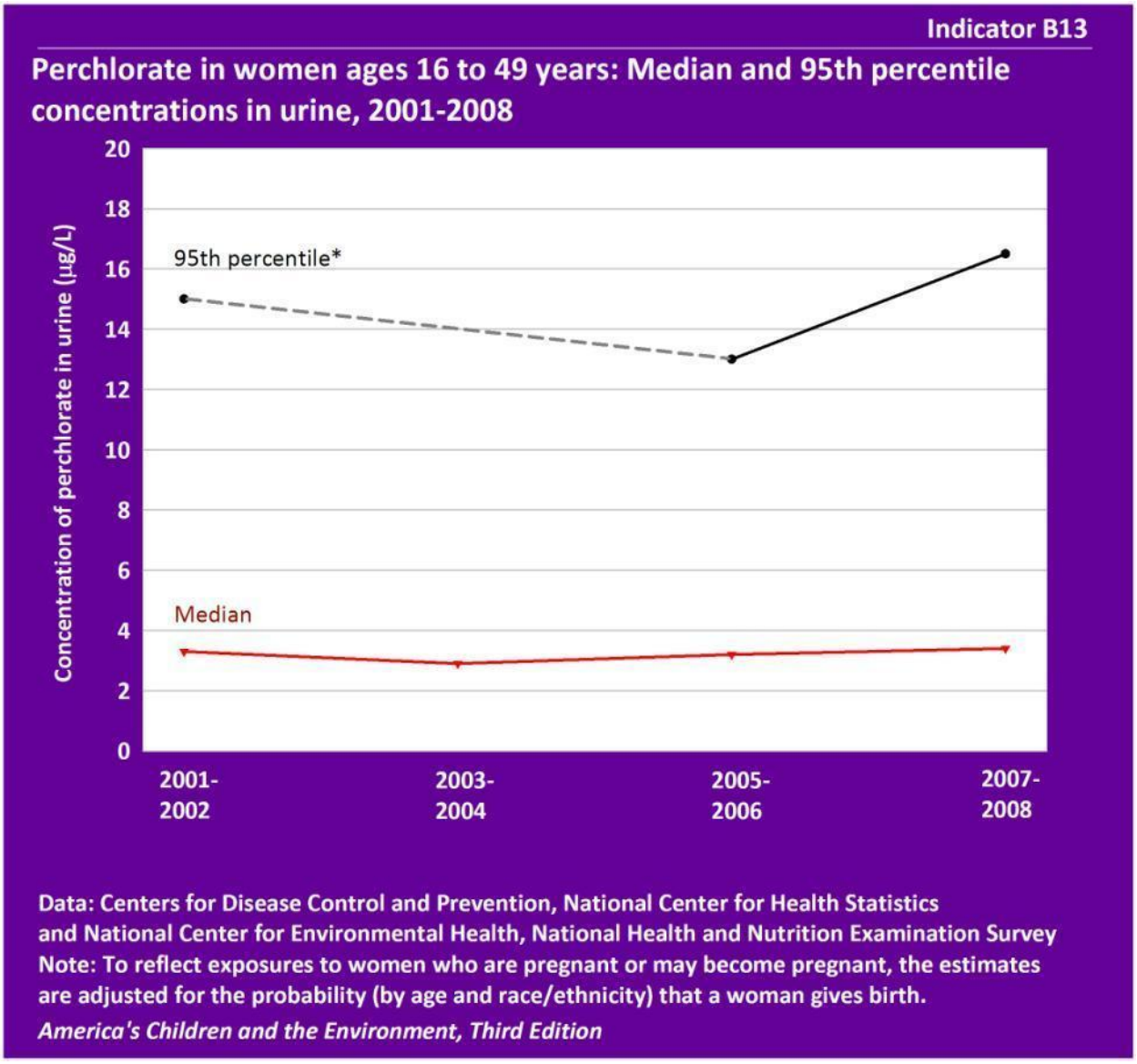
Data Presented in the Indicator

Indicator B13 presents median and 95th percentile concentrations of perchlorate in urine over time for women ages 16 to 49 years, using NHANES data from 2001–2008.

Additional information showing how the median and 95th percentile levels of perchlorate in urine vary by race/ethnicity and family income for women ages 16 to 49 years is presented in supplemental data tables for these indicators. Data tables also display information on the median and 95th percentile levels of perchlorate in urine for children ages 6 to 17 years, including how levels vary by race/ethnicity, family income, and age.

NHANES does not collect urine samples from children less than 6 years of age, and thus cannot assess the exposure of infants, who may be exposed to unhealthy levels of perchlorate due to the presence of perchlorate in breast milk and some infant formula.[18,20,21,31,32]

Please see the Introduction to the Biomonitoring section for an explanation of the terms "median" and "95th percentile," a description of the race/ethnicity and income groups used in the ACE3 biomonitoring indicators, and information on the statistical significance testing applied to these indicators.



*The 95th percentile concentration for 2003-2004 is not reported because it has large uncertainty: the relative standard error, RSE, is 40% or greater (RSE = standard error divided by the estimate).

Data characterization

- Data for this indicator are obtained from an ongoing continuous survey conducted by the National Center for Health Statistics.
- Survey data are representative of the U.S. civilian noninstitutionalized population.
- Perchlorate is measured in urine samples obtained from individual survey participants.

- From 2001–2002 to 2007–2008, the median level of perchlorate in urine among women ages 16 to 49 years was 3 µg/L with little variation over time. Over the same period, the 95th percentile varied between 13 and 17 µg/L.

- In 2005–2008, there was little variation in median or 95th percentile perchlorate levels by race/ethnicity or income among women ages 16 to 49 years. (See Tables B13a and B13b.)
- From 2001–2002 to 2007–2008, the median level of perchlorate among children ages 6 to 17 years was 5 µg/L with little variation over time. The 95th percentile perchlorate level among children increased from 15 µg/L in 2001–2002 to 19 µg/L in 2007–2008. (See Table B13c.)
  - The increasing trend in children's 95th percentile perchlorate levels was statistically significant.
- The median perchlorate level among children ages 6 to 17 years was about 42% higher than the level found in women of childbearing age in 2005–2008, while the 95th percentile level among children ages 6 to 17 years was about 19% higher than in women of childbearing age. (See Tables B13 and B13c.)
- Differences in urinary perchlorate levels by race/ethnicity and income among children ages 6 to 17 years were relatively limited. (See Tables B13d and B13e.)
- There were minimal differences in urinary perchlorate levels by age group among children ages 6 to 17 years. (See Table B13f.)

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Introduction

Why is EPA tracking children's health outcomes in *America's Children and the Environment*?

The central goal of efforts to protect children's environmental health is the reduction of disease, disability, and mortality. Many different factors contribute to children's health, including nutrition, prenatal and childhood exposure to toxins in the environment, genetics, socioeconomic status, access to medical care, and exercise. Data on children's health outcomes can provide important information about changes over time and differences between demographic groups. In particular, monitoring children's health outcomes for which causes are unknown or not well established can help stimulate hypotheses, some of which may point to environmental factors, which then can be examined rigorously in future studies.

What health outcomes are included in America's Children and the Environment, Third Edition (ACE3)?

Health outcomes were selected for ACE3 based on: (1) magnitude of prevalence and/or trend in prevalence, and severity of health outcome; (2) research findings that indicate environmental contaminants or characteristics may be contributing factors; and (3) the availability of nationally representative data suitable for constructing an indicator. EPA obtained input from its Children's Health Protection Advisory Committee to assist in selecting topics from among the many diseases and health disorders that affect children. The ACE3 Health indicators address the following topics:

- Respiratory diseases
- Childhood cancer
- Neurodevelopmental disorders
- Obesity
- Adverse birth outcomes

What data sources were used to develop the Health indicators?

Data for all of the selected health outcomes, with the exception of childhood cancer, were based on surveys and registries conducted/maintained by the National Center for Health Statistics (NCHS). These include the National Health Interview Survey (NHIS), National Hospital Ambulatory Medical Care Survey (NHAMCS), National Hospital Discharge Survey (NHDS), National Health and Nutrition Examination Survey (NHANES), and the National Vital Statistics System (NVSS). Data on childhood cancer were obtained from the National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) Program.

NHIS and NHANES collect health information from a probability sample of the U.S. civilian, noninstitutionalized population, and survey data are weighted to yield national estimates describing this population. NHAMCS and NHDS collect patient visit information from a sample of hospitals, and the survey data are weighted to estimate the rates of respiratory-related emergency room visits and

hospital admissions in the United States. NVSS is a registry that captures virtually all births that occur in the United States, and thus does not rely on sampling. The SEER program has attributes of both a survey and a registry. It is based on a collection of registries located across the United States that record all tumors that occur in specific geographical regions. The registry information is then used to estimate the occurrence of cancer, including childhood cancer, for the entire country.

What can we learn from the Health indicators?

The indicators presented in this report focus on health outcome data collected over multiple years, which allow determination of whether the reported prevalence or rate of each outcome is increasing, decreasing, or not changing over time. An additional focus is whether particular groups (defined by race/ethnicity and income) within the population are disproportionately affected by a given health outcome. Such trends and comparisons can generate hypotheses and help identify opportunities for future action.

The topic text provided before the indicators reviews the scientific evidence regarding environmental factors and other factors contributing to the disease or disorder, providing context that informs the interpretation of the indicators. For some of the selected health outcomes, the scientific evidence suggests that environmental contaminants may play a role in the development of the disease or disorder. For other health outcomes, available evidence is less clear as to whether environmental contaminants are involved. The inclusion of the selected health outcomes in this report does not imply that environmental contaminants or other environmental factors definitely play a role in the selected health outcomes. It can be very difficult to develop conclusive evidence that environmental factors cause or contribute to the incidence of childhood health effects, and research is ongoing. Where available, we rely on authoritative reviews of the literature and report their conclusions regarding the strength of the evidence for a causal role of specific environmental factors in the development of childhood diseases and disorders. When such reviews are unavailable, we summarize important findings from individual studies that address the potential role of environmental factors in contributing to an effect.

Furthermore, the inclusion of the selected health outcomes in this report does not imply that environmental factors, in cases where they do play a role, are the only cause of the disease or disorder. Most often, health outcomes are a result of multiple causes that may include genetics, nutrition, and socioeconomic factors, as well as prenatal and childhood exposure to environmental contaminants, and other environmental factors. The various factors may also interact, such as a genetic predisposition that makes a person more susceptible to the effects of an environmental exposure.

In some instances, the indicators show that the prevalence of a health outcome is increasing while important environmental exposures are decreasing. Although this could suggest that the environmental exposures addressed in the indicators are unrelated to the health outcomes being measured, it could also result from a lag between environmental improvements and changes in related health outcomes, or changes in other important environmental exposures that are not currently measured by the indicators in the report. The Health indicators are therefore not intended as a basis for concluding that an environmental factor is or is not related to a particular children's health outcome.

What information is provided for each Health topic?

An introduction section explains the relevance of the topic to children's environmental health, including a description of the health outcomes and a discussion of evidence indicating or suggesting that environmental agents may play a role in contributing to the outcomes. The introduction is followed by a description of the indicators, including a summary of the data available and brief information on how each indicator was calculated. Two to four indicators, each a graphical presentation of the available data, are included for each topic. All Health topics include an indicator that presents a time series. Some of the topics also include indicators that show a comparison of the most current health outcome data by race/ethnicity and income level. Beneath each figure are explanatory bullet points describing dataset characteristics and key findings presented in the figure, along with key data from any supplemental data tables. References are provided for each topic at the end of the report.[i]

Data tables are provided in Appendix A. The tables include all indicator values depicted in the indicator figures, along with additional data of interest not shown in the figures. Metadata describing the data sources are provided in Appendix B. Documents providing details of how the indicators were calculated are available on the ACE website (www.epa.gov/ace).[ii]

Many of the topics presented in the Health indicators are addressed in Healthy People 2020, which provides science-based, 10-year national objectives for improving the health of all Americans. Appendix C provides examples of the alignment of the Health topics presented in ACE3 with objectives in Healthy People 2020.

What race/ethnicity groups are used in reporting indicator values?

For each topic in the Health section, indicator values are provided for defined race/ethnicity groups—either in the indicator figures or in the data tables. The race/ethnicity groups vary to some extent across the indicators, depending on the extent to which the data support reporting indicator values for specific race/ethnicity groups. Where possible, the Health indicators provide data for the following races/ethnicities:

- White non-Hispanic
- Black non-Hispanic
- Hispanic[iii]
- Asian or Pacific Islander
- American Indian or Alaska Native

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[i] In the current copy, references are provided at the end of each topic section. In the final printed report, references will appear, separated by topic, at the end of the report.

[ii] The Methods documents will be posted to the ACE website when the final ACE3 report is posted in early 2013.

[iii] For the Obesity indicators, values are provided for "Mexican-American" ethnicity rather than "Hispanic" ethnicity because in all years up to 2006, NHANES was designed to provide statistically reliable estimates for Mexican-Americans rather than all Hispanics. NHANES now oversamples Hispanics instead of Mexican-Americans, beginning with NHANES 2007–2008.
Please see http://www.cdc.gov/nchs/nhanes/nhanes2007-2008/sampling_0708.htm/.

Specific races/ethnicities for which the data support stratified group analysis are described in the text preceding each Health indicator.

What income groups are used in reporting indicator values?

For indicators presenting the prevalence of asthma, neurodevelopmental disorders, and obesity, indicator values are presented for income groups defined on the basis of the federal poverty level. Poverty level is defined by the federal government, and is based on income thresholds that vary by family size and composition. In 2010, for example, the poverty threshold was $22,113 for a household with two adults and two related children.[1] These Health indicators (in figures and/or data tables) provide data separately for individuals in families with incomes below poverty level, and those in families with incomes at or above poverty level.

Further detail is provided in the data tables by dividing those above poverty level into two groups: 100–200% of poverty level, and greater than 200% of poverty level. The category of incomes between the poverty level and twice the poverty level (sometimes referred to as "near poor") represents households that have relatively low incomes but are not below the officially defined poverty level, and is frequently used by NCHS in its reporting of health data.

For indicators of respiratory emergency room visits and hospitalization, childhood cancer, and adverse birth outcomes, no income group comparisons can be provided because income data are not collected for these data sets.

How were the indicators calculated and presented?

Data files: The indicators were calculated from publicly available data files obtained from the NCHS and SEER websites. The files include various information such as survey responses (NHIS), diagnosis codes (NHAMCS and NHDS), type of cancer (SEER), gestational age and birth weight (NVSS), and body measurements (NHANES). Depending on the data set, the files may also include information on age, sex, race/ethnicity, and income level (that is, family income above or below poverty level). For the survey data, each individual observation also has a sample weight that is used in calculating population statistics; the weight equals the number of people in the U.S. population represented by the particular observation.

Population age groups: The age groups covered by the indicators differ among the Health topics. The indicators for respiratory diseases used data for children ages 17 years and younger. The indicators for childhood cancer used data for children ages 19 years and younger. The indicators for neurodevelopmental disorders used data for children ages 5 to 17 years. The indicators for adverse birth outcomes used data ascertained at birth. The indicators for obesity used data for children ages 2 to 17 years.

Calculated prevalence or rate of occurrence for each health outcome: Depending on the nature of the available data, some of the indicators present prevalence data while others express the occurrence of health outcomes as a rate. The main difference between these two measures is that prevalence presents data occurring at one point in time. These prevalence measures are proportions, such as the percentage of children who currently have asthma or the percentage of children classified as obese. Rates, on the other hand, express the number of events, such as emergency room visits, hospital

admissions, new cancer cases, or cancer deaths, that occur over a definite time period (one year for all of the ACE3 indicators), per the population at risk for the event. The population at risk is either all children or all births, depending on the indicator.

Statistical considerations in presenting and characterizing the indicators: Statistical analysis has been applied to the ACE3 Health indicators to evaluate trends over time in indicator values (for example, percentage of children with asthma) or differences in indicator values between demographic groups. These analyses use a 5% significance level, meaning that a conclusion of statistical significance is made only when there is no more than a 5% probability that the observed trend or difference occurred by chance ($p \leq 0.05$).

The statistical analysis of trends over time for an ACE3 Health indicator is dependent on how the indicator values vary over time, the number of survey years included in the analysis, the number of observations in each survey year, and various aspects of the survey design. The evaluation of trends over time incorporates annual data from each year within the time period reported. A finding of statistical significance for differences in indicator values between demographic groups depends on the magnitude of the difference, the number of observations in each group, and various aspects of the survey design. For example, if the prevalence of a health effect is different between two groups, the statistical test is more likely to detect a difference when data have been obtained from a larger number of people in those groups. It should be noted that when statistical testing is conducted for differences among multiple demographic groups (for example, considering both race/ethnicity and income level), the large number of comparisons involved increases the probability that some differences identified as statistically significant may actually have occurred by chance.

A finding of statistical significance is useful for determining that an observed trend or difference was unlikely to have occurred by chance. However, a determination of statistical significance by itself does not convey information about the magnitude of the increase, decrease, or difference in indicator values. Furthermore, a lack of statistical significance means only that occurrence by chance cannot be ruled out. Thus, a conclusion about statistical significance is only part of the information that should be considered when determining the public health implications of trends or differences in indicator values.

In some cases, calculated indicator values have substantial uncertainty. Uncertainty in these estimates is assessed by looking at the relative standard error (RSE), a measure of how large the variability of the estimate is in relation to the estimate (RSE = standard error divided by the estimate).[iv] The estimate should be interpreted with caution if the RSE is at least 30%; a notation is provided for such estimates in the indicator figures and tables. If the RSE is greater than 40%, the estimate is considered to have very large uncertainty and is not reported.[v]

[iv] Standard errors for all health indicator values are provided in a file available on the ACE website (www.epa.gov/ace). (This file will be posted to the ACE website when the final ACE3 report is posted in early 2013.)

[v] For respiratory emergency room and hospital visits (indicator H3), an estimate is also considered to have very large uncertainty and is not reported if it is based on fewer than 30 sampled visits. For obesity (indicators H10 and H11), values are not reported if the RSE cannot be reliably estimated.

References

1. U.S. Census Bureau. *Poverty Thresholds by Size of Family and Number of Related Children Under 18 Years: 2010*. U.S. Census Bureau. Retrieved August 20, 2011 from http://www.census.gov/hhes/www/cpstables/032011/pov/new35_000.htm.

Respiratory Diseases

Respiratory diseases and illness, such as asthma, bronchitis, pneumonia, allergic rhinitis, and sinusitis, can greatly impair a child's ability to function and are an important cause of missed school days and limitations of activities. Symptoms associated with both mild and more severe manifestations of these respiratory conditions, such as cough, wheeze, congestion, chest pain, shortness of breath, respiratory distress, and death in the most extreme cases, are responsible for substantial morbidity and a large cost burden to families and society.

Outdoor and indoor air pollution can adversely affect children's respiratory health.[1-7] Studies have shown that air pollution can exacerbate existing respiratory conditions such as asthma and upper airway allergies.[1,8-10] Increasing evidence suggests that exposure to certain air pollutants may contribute to the onset of asthma in children, although studies relating to the exacerbation of pre-existing asthma are more prevalent because they are easier to conduct.[11-13] Air pollution also increases a child's risk of developing respiratory infections, most likely by causing inflammation and/or impaired immune response.[14-16]

EPA sets health-based National Ambient Air Quality Standards for six air pollutants.[17] These pollutants, referred to as criteria air pollutants, are particulate matter (PM), ground-level ozone, nitrogen oxides, sulfur oxides, carbon monoxide (CO), and lead. Four of these pollutants have extensive evidence linking them to respiratory diseases in children (PM, ground-level ozone, nitrogen oxides, and sulfur oxides). The evidence for respiratory effects is weaker for CO, and lead has not been linked to adverse respiratory outcomes.

PM is associated with significant respiratory problems in children, including aggravated asthma; exacerbation of allergic symptoms; reduced growth of lung function; and increased hospital admissions, emergency room visits, and doctor visits for respiratory diseases, especially in children with lung diseases such as asthma.[6] Particulate air pollution has also been associated with respiratory-related infant mortality, even at relatively low PM levels that are commonly experienced in the United States.[18,19]

Short-term exposure to ground-level ozone can cause a variety of respiratory health effects, including airway inflammation; reduced lung function; increased susceptibility to respiratory infection; and respiratory symptoms such as cough, wheezing, chest pain, and shortness of breath.[3,20,21] Ozone exposure can decrease the capacity to perform exercise and has been associated with the aggravation of respiratory illnesses such as asthma and bronchitis, leading to increased use of medication, absences from school, doctor and emergency department visits, and hospital admissions.[3] Studies have also found that long-term ozone exposure may contribute to the development of asthma, especially among children with certain genetic susceptibilities and children who frequently exercise outdoors.[22-24]

Nitrogen dioxide (NO_2) is an odorless gas that can irritate the eyes, nose, and throat, and can cause shortness of breath. EPA has concluded that exposure to NO_2 can lead to increased respiratory illnesses and symptoms, more severe asthma symptoms, and an increase in the number of emergency department visits and hospital admissions for respiratory causes, especially asthma.[4]

Short-term exposures of persons with asthma to elevated levels of sulfur dioxide (SO_2) while exercising at a moderate level may result in breathing difficulties, accompanied by symptoms such as wheezing,

chest tightness, or shortness of breath. Studies also provide consistent evidence of an association between short-term SO_2 exposure and increased respiratory symptoms in children, especially those with asthma or chronic respiratory symptoms. Short-term exposures to SO_2 have also been associated with respiratory-related emergency department visits and hospital admissions, particularly for children.[5]

Exposure to CO reduces the capacity of the blood to carry oxygen, thereby decreasing the supply of oxygen to tissues and organs such as the heart. Research suggests correlations between CO exposure and the exacerbation of asthma; however, CO levels are highly correlated with other combustion-related pollutants, especially in locations near roads. Few analyses clearly distinguish the contributions of CO from those of the larger traffic-related air pollutant mixture, thus it is uncertain whether the observed health effects are truly attributable to CO or whether they are due to other co-occurring air pollutants.[7,10,25]

In addition to the criteria air pollutants, EPA regulates 187 hazardous air pollutants (HAPs) that are known or suspected to cause serious health effects or adverse environmental effects. For many of these pollutants, information on health effects is scarce. HAPs that may be of particular concern for the induction and exacerbation of asthma include acrolein, formaldehyde, nickel, and chromium.[26] Acrolein has been identified as a HAP of particular concern for possible respiratory effects at levels commonly found in outdoor air in the United States.[27-29] Acrolein can cause respiratory irritation in individuals who do not have asthma.[30]

Pollution from traffic-related sources, a mix of criteria air pollutants and HAPs, appears to pose particular threats to a child's respiratory system. Many studies have found a correlation between proximity to traffic (or to traffic-related pollutants) and occurrence of new asthma cases or exacerbation of existing asthma and other respiratory symptoms, including reduced growth of lung function during childhood.[11-13,31-36] A report by the Health Effects Institute concluded that living close to busy roads appears to be an independent risk factor for the onset of childhood asthma. The same report also concluded that the evidence was "sufficient" to infer a causal association between exposure to traffic-related pollution and exacerbations of asthma in children.[37] Some studies have suggested that traffic-related pollutants may contribute to the development of allergic disease, either by affecting the immune response directly or by increasing the concentration or biological activity of the allergens themselves.[38-40]

Children can also be exposed to air pollution inside homes, schools, and other buildings. Indoor air pollutants from biological sources such as mold; dust mites; pet dander (skin flakes); and droppings and body parts from cockroaches, rodents, and other pests or insects, can lead to allergic reactions, exacerbate existing asthma, and have been associated with the development of respiratory symptoms.[1,41,42] Furthermore, the Institute of Medicine concluded that exposure to dust mites can cause asthma in susceptible children, and exposure to cockroaches may cause asthma in young children.[1]

PM and NO_2, discussed previously as outdoor air pollutants, also pollute indoor air when they are emitted from gas stoves, gas or oil furnaces, fireplaces, wood stoves, and kerosene or gas space heaters. Indoor concentrations of these combustion byproducts can reach very high levels in developing countries where solid fuels are used extensively for cooking and home heating, but may also affect the respiratory health of children in developed countries, especially during the winter when use of fireplaces and space heaters is more common.[43] Environmental tobacco smoke (ETS), also known as secondhand smoke, is an air pollutant mixture that includes particles and NO_2 as well as thousands of other

chemicals. The Surgeon General has concluded that exposure to ETS causes sudden infant death syndrome (SIDS), acute lower respiratory infection, ear problems, and more severe asthma in children. Smoking by parents causes respiratory symptoms and slows lung growth in their children.[2]

A number of air pollutants emitted indoors by a variety of household items such as building materials and home furnishings, recently dry-cleaned clothes, cleaning supplies, and room deodorizers, have been associated with respiratory symptoms and may play a role in the exacerbation or development of childhood asthma.[44,45] A recent systematic review of seven studies concluded that there is a significant association between exposure to formaldehyde—a chemical released from particle board, insulation, carpet, and furniture—and self-reported or diagnosed asthma in children.[46]

Air pollutants can enter the bloodstream of pregnant women and cross the placenta to reach the developing fetus; thus the period of fetal development may be a window of special vulnerability for respiratory effects of some air pollutants. Studies indicate that prenatal exposure to ETS may increase the risk of developing asthma during childhood and/or lead to impaired lung function, especially among children with asthma.[2,47-50] Studies have also found that prenatal exposure to polycyclic aromatic hydrocarbons (hazardous air pollutants found in diesel exhaust, ETS, and smoke from burning organic materials) is associated with childhood respiratory illnesses and the development of asthma, particularly when in combination with prenatal or postnatal exposure to ETS.[51-53] Limited studies of prenatal exposure to criteria air pollutants have found that exposure to PM, CO, and oxides of nitrogen and sulfur may increase the risk of developing asthma as well as worsen respiratory outcomes among those children who do develop asthma.[11,54,55] However, it is difficult to distinguish the effects of prenatal and early childhood exposure because exposure to air pollutants is often very similar during both periods.

Asthma

Asthma is a chronic inflammatory disease of the airways. When children with asthma are exposed to an asthma trigger, the airway walls become inflamed and secrete more mucus, and the muscles around the airways tighten. This exaggerates the normal airway constriction that occurs on exhalation, trapping air in the lungs and compromising normal oxygen exchange. These physiological changes can result in wheezing, coughing, difficulty in breathing, chest tightness, and pain.

Asthma is one of the most common chronic diseases among children: in the year 2009, it affected 7.1 million (or about 10% of) children in the United States.[56] It is costly in both human and monetary terms: estimated national costs of asthma in 2007 were $56 billion.[57] The percentage of children with asthma increased substantially from 1980–1996 and remains high.[58] Researchers do not completely understand why children develop asthma or why the prevalence has increased.

Asthma is a complex disease with many factors, including genetic factors and environmental factors, that interact to influence its development and severity. The percentage of children reported to have current asthma differs by age, family history of asthma and allergies, racial and ethnic group, and family income. Children of color and children of lower-income families are more likely to be diagnosed with asthma. Because minority populations are more likely to be of low socioeconomic status, it is difficult to establish whether racial/ethnic group is an independent risk factor for the development of asthma. While some research has suggested that variations in asthma prevalence among racial groups can be explained by socioeconomic factors,[59,60] another study suggested that the difference persists even after accounting for socioeconomic factors.[61] Other researchers have proposed that the greater prevalence of

asthma among Black children can be explained by their disproportionate presence in urban environments.[62]

Children living in poverty are more likely to have poorly maintained housing, which can present risk factors for asthma development and exacerbation. The Institute of Medicine concluded that exposure to dust mites causes asthma in susceptible children, and that cockroaches may cause asthma in young children.[1] Research suggests that lower-income children are more likely to live in homes with high levels of cockroach allergens and homes where someone smokes regularly.[63-66] A nationally representative survey of allergens in U.S. housing reported higher levels of dust mite allergen in bedding from lower-income families.[67] Household mouse allergen was also found at higher concentrations in low-income homes, mobile homes, and older homes.[68] In addition, total dust weight itself has been found to contribute to respiratory symptoms, including asthma and wheeze. Households with lower income, older homes, household pets, a smoker in the house, and less frequent cleaning are more likely to have higher dust weight levels.[69] Furthermore, children living in poverty may also face barriers to medical care, have less access to routine medical care and instructions for asthma management, or may be less likely to use asthma control medications.[70-76] These factors may increase asthma morbidity, as evidenced by increased asthma symptoms among those diagnosed with the disease.

Asthma indicators provide data on the percentage of children who have asthma as well as health outcomes for children with asthma. Indicators H1 and H2 focus on the prevalence of asthma among children. Indicator H1 provides the best nationally representative data available on prevalence of asthma over time among children ages 0 to 17 years. It provides two measures of asthma prevalence by year, from 1997–2010: current asthma prevalence and asthma attack prevalence. While the former measure reports on the percentage of children who have asthma each year, the latter measure presents data on children who had asthma attacks in the past year, and thus represents outcomes for children with asthma by identifying the proportion of children with ongoing or uncontrolled symptoms. Indicator H2 provides the best nationally representative data available to compare the prevalence of current asthma among children 0 to 17 years by race/ethnicity and family income for the years 2007–2010.[vi]

Emergency Room Visits and Hospitalizations for Respiratory Diseases

Children who visit emergency rooms or are hospitalized for respiratory diseases (including asthma and upper and lower respiratory infections such as bronchiolitis and pneumonia) usually represent the most severe cases of respiratory disease. Although only a fraction of children with respiratory diseases are admitted to the hospital, asthma is the third leading cause of hospitalization for children in the United States and bronchiolitis is the leading cause of acute illness and hospitalization in infants.[77,78]

Emergency room visits and hospital admissions for respiratory diseases can be related to a number of factors. These factors include exposure to asthma triggers, lack of access to primary health care, lack of or inadequate insurance, inadequate instructions for asthma management, or inadequate compliance with given instructions.[79-83] Changes in emergency room visits and hospital admissions over time may also reflect changes in medical practices, asthma therapy, and access to and use of care.[84,85]

[vi] State-specific asthma information can be found in the CDC report, *The State of Childhood Asthma, United States, 1980–2005*, located at http://www.cdc.gov/nchs/data/ad/ad381.pdf.

For children with existing respiratory conditions, exposure to air pollution from indoor and outdoor sources can trigger the onset of symptoms and lead to difficulty in breathing, increased use of medication, school absenteeism, visits to the doctor's office, and respiratory-related hospitalizations and trips to the emergency room.[3-6]

Studies have suggested that exacerbation of asthma from exposure to air pollution can be more severe among people with low income compared with other populations,[86,87] and that the gap between Black and White children in both hospitalizations and deaths from asthma appears to be growing.[88-90] The asthma death rate among Black non-Hispanic children with asthma was 4.9 times higher than the rate for White non-Hispanic children with asthma in 2004–2005.[88] Asthma is the leading cause of emergency room visits, hospitalizations, and missed school days in New York City's poorest neighborhoods.[91] In Maryland, the rate of children's emergency room visits for asthma is twice as high for Baltimore City (an area with a relatively high percentage of lower income and Black children) than for any other jurisdiction.[92]

Indicator H3 provides the best nationally representative data available on the frequency with which children experienced asthma or respiratory symptoms resulting in an emergency room visit or hospitalization for the years 1996–2008. This indicator highlights the most severe cases of respiratory illness among children ages 0 to 17 years. Indicator H3 includes further information on health outcomes for children with asthma, in addition to the asthma attack prevalence information in Indicator H1, by reporting on trends in children's hospitalizations and emergency room visits due to asthma.

Indicator H1: Percentage of children ages 0 to 17 years with asthma, 1997–2010

Indicator H2: Percentage of children ages 0 to 17 years reported to have current asthma, by race/ethnicity and family income, 2007–2010

About the Indicators: Indicators H1 and H2 present the percentage of children ages 0 to 17 years with asthma. The data are from a national survey that collects health information from a representative sample of the population each year. Indicator H1 shows how children's asthma rates have changed over time. Indicator H2 shows how children's asthma rates vary by race/ethnicity and family income level.

National Health Interview Survey

The National Health Interview Survey (NHIS) provides nationally representative data on the prevalence of childhood asthma in the United States each year. NHIS is a large-scale household interview survey of a representative sample of the civilian noninstitutionalized U.S. population, conducted by the National Center for Health Statistics (NCHS). The interviews are conducted in person at the participants' homes. From 1997–2005, interviews were conducted for approximately 12,000–14,000 children annually. From 2006–2008, interviews were conducted for approximately 9,000–10,000 children per year. In 2009 and 2010, interviews were conducted for approximately 11,000 children per year.

With a major survey redesign implemented in 1997, the measurement of childhood asthma prevalence in NHIS was changed to reporting the percentage of children ever diagnosed with asthma (lifetime asthma prevalence) and children ever diagnosed with asthma that also had an asthma attack in the previous 12 months (asthma attack prevalence). The data are obtained by asking a parent or other knowledgeable household adult questions regarding the child's health status. NHIS asks "Has a doctor or other health professional ever told you that your child has asthma?" If the answer is YES to this question, NHIS then asks (1) "Does your child still have asthma?" and (2) "during the past 12 months, has your child had an episode of asthma or an asthma attack?" The question "Does your child still have asthma?" was introduced in 2001 and identifies children who were previously diagnosed with asthma and who currently have asthma (current asthma prevalence). Some children may have asthma when they are young and experience fewer symptoms as they get older, or their asthma may be well controlled through medication and by avoiding triggers of asthma attacks. In such cases, children may currently have asthma but may not have experienced any attacks in the previous year.

Data Presented in the Indicators

Indicator H1 presents two different measures of asthma prevalence using data from the NHIS: current asthma and asthma attack prevalence. Indicator H1 provides the annual estimates of asthma prevalence for all children 0 to 17 years of age for the years 1997–2010. Indicator H2 reports on the percentage of children ages 0 to 17 years reported to have current asthma, by race/ethnicity and family income, in 2007–2010. NHIS is also the source of data for this indicator. The 2007, 2008, 2009 and 2010 data are combined for this indicator in order to increase the statistical reliability of the estimates for each race/ethnicity and income group.

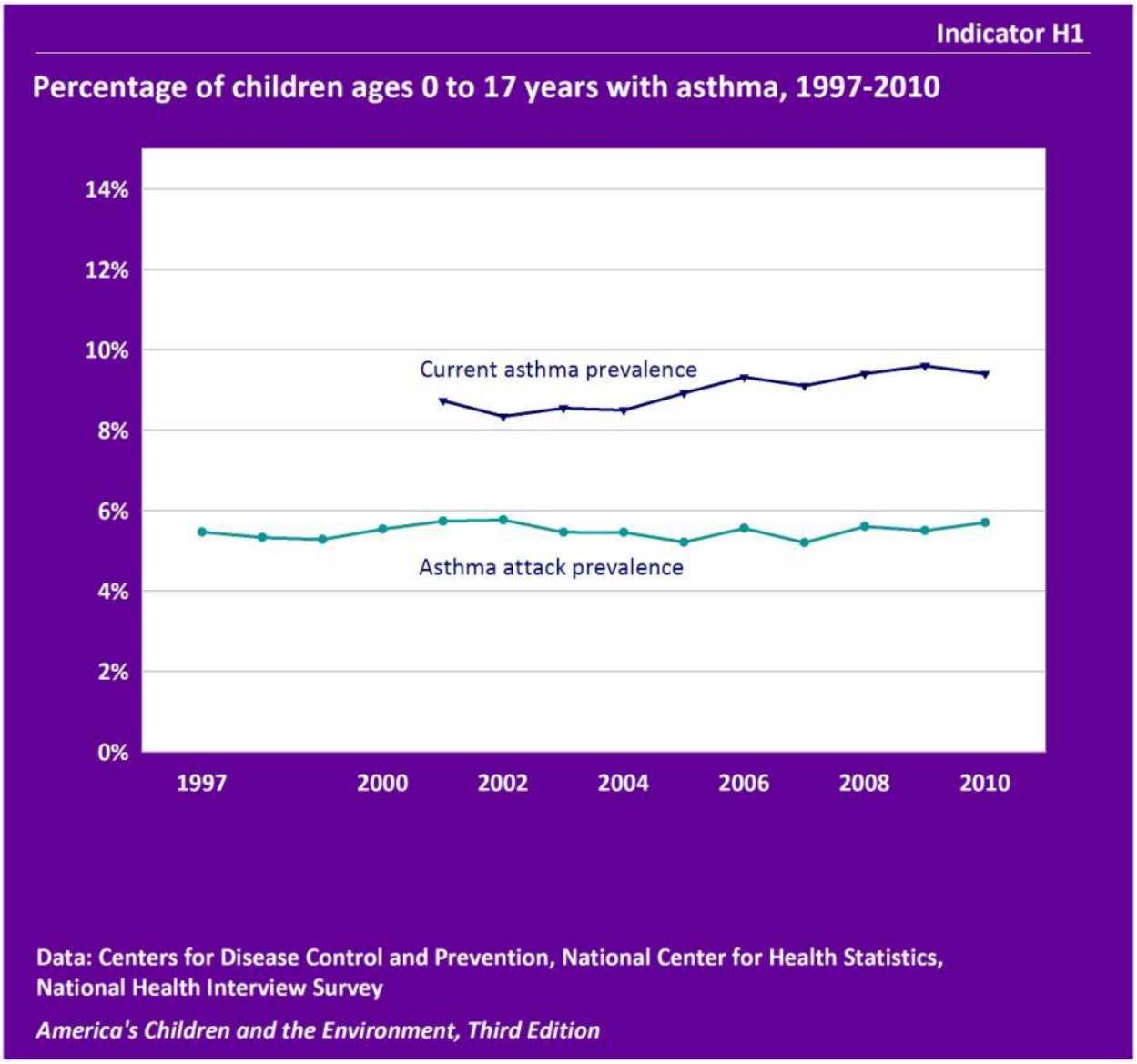
For Indicator H2, five race/ethnicity groups are presented: White non-Hispanic, Black non-Hispanic, Asian non-Hispanic, Hispanic, and "All Other Races." The "All Others Races" category includes all other races not specified, together with those individuals who report more than one race. The limits of the sample design and sample size often prevent statistically reliable estimates for smaller race/ethnicity groups. The data are also tabulated for three income groups: all incomes, below the poverty level, and greater than or equal to the poverty level. These prevalence data are based on a survey respondent reporting that asthma has been diagnosed by a health care provider. Accuracy of responses and access to health care providers may vary among population groups.[93,94]

In addition to the data shown in Indicator H1, a supplemental table shows data for the percentage of children who had asthma in the past 12 months (asthma period prevalence), for the years 1980–1996. Estimates for asthma period prevalence are not directly comparable to any of the three prevalence estimates collected since 1997 because of changes in the NHIS survey questions. The data table for Indicator H2 shows the prevalence of current asthma for an expanded set of race/ethnicity categories, including Mexican-American and Puerto Rican. A supplemental data table shows the prevalence of current asthma by age and sex for the years 2007-2010.

Please see the Introduction to the Health section for discussion of statistical significance testing applied to these indicators.

Other Estimates of Asthma Prevalence

In addition to NHIS, other NCHS surveys provide data on asthma prevalence. A telephone-based survey conducted in 2007 by NCHS along with state and local governments found that 11% of high school students currently had asthma.[95] The 2007 National Survey of Children's Health (NSCH) found that nationwide 9.0% of children ages 0 to 17 years currently had asthma, which is very similar to the estimate from NHIS for 2007. The 2007 NSCH also provides information at the state level: South Dakota has the lowest asthma rates, with only 5.2% of children currently having asthma. The District of Columbia has the highest asthma rates, with 14.4% of children currently having asthma.[96]



Data characterization

- Data for this indicator are obtained from an ongoing annual survey conducted by the National Center for Health Statistics.
- Survey data are representative of the U.S. civilian noninstitutionalized population.
- A parent or other knowledgeable adult in each sampled household is asked questions regarding the child's health status, including if they have ever been told the child has asthma, if the child has had an asthma attack in the past year, and if the child currently has asthma.

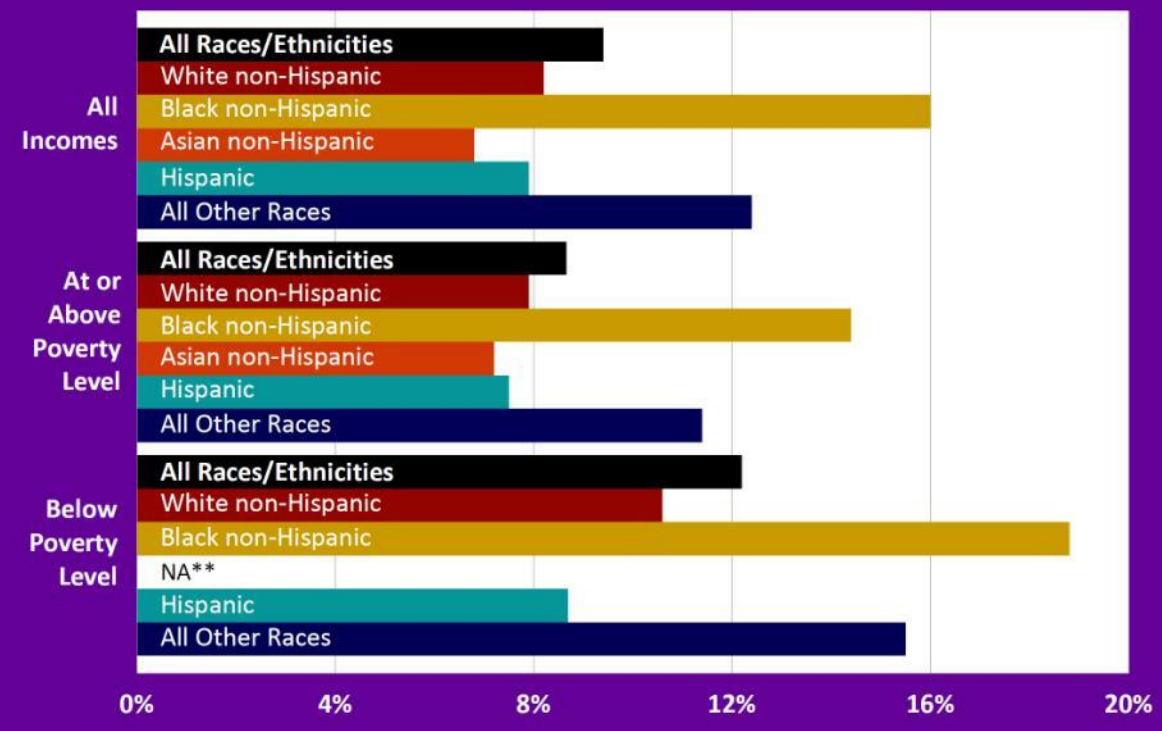
- The proportion of children reported to currently have asthma increased from 8.7% in 2001 to 9.4% in 2010.
- In 2010, 5.7% of all children were reported to have had one or more asthma attacks in the previous 12 months. There was little change in this rate between 1997 and 2010.

- In 2001, 61.7% of children with current asthma had one or more asthma attacks in the previous 12 months, and by 2010 this figure had declined to 58.3%.[vii] The decreasing trend from 2001 to 2010 was statistically significant. (See Table H1c.)
- Between 1980 and 1995 the percentage of children who had asthma in the past 12 months increased from 3.6% in 1980 to 7.5% in 1995. Methods for measurement of childhood asthma changed in 1997, so earlier data cannot be compared to the data from 1997–2010. (See Table H1b.)

[vii] See indicator H3 for further information on outcomes for children with asthma.

Indicator H2

Percentage of children ages 0 to 17 years reported to have current asthma, by race/ethnicity and family income, 2007-2010



Data: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health Interview Survey

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** Not available. The estimate is not reported because it has large uncertainty: the relative standard error, RSE, is 40% or greater (RSE = standard error divided by the estimate).

Data characterization

- Data for this indicator are obtained from an ongoing annual survey conducted by the National Center for Health Statistics.
- Survey data are representative of the U.S. civilian noninstitutionalized population.
- A parent or other knowledgeable adult in each sampled household is asked questions regarding the child's health status, including if they have ever been told the child has asthma, if the child has had an asthma attack in the past year, and if the child currently has asthma.

- In 2007–2010, 9.4% of all children were reported to currently have asthma.

- Among children living in families with incomes below the poverty level, 12.2% were reported to currently have asthma. Among children living in families with incomes at the poverty level and higher, 8.7% were reported to currently have asthma. This difference was statistically significant.
- In 2007–2010, the percentages of Black non-Hispanic children and children of "All Other Races" reported to currently have asthma, 16.0% and 12.4% respectively, were greater than for White non-Hispanic children (8.2%), Hispanic children (7.9%), and Asian non-Hispanic children (6.8%).
  - The differences in current asthma prevalence among Black non-Hispanic or "All Other Races" children, compared with current asthma prevalence among Hispanic, White non-Hispanic, or Asian non-Hispanic children, were statistically significant. These differences by race/ethnicity also hold true when considering only children below poverty level and only children at or above poverty level.
- Among Hispanic children, about 1 in 4 Puerto Rican children (23.3%) living in families with incomes below the poverty level were reported to currently have asthma. The rate of reported current asthma for Mexican-American children living in families with incomes below the poverty level is 6.6%. This difference was statistically significant. (See Table H2.)
- Among boys, 10.7% were reported to have current asthma compared with 8.0% of girls. This difference was statistically significant. (See Table H2a.)
- Among children ages 0 to 5 years, 7.1% were reported to have current asthma compared with 10.0% of children ages 6 to 10 years and 11.0% of children ages 11 to 17 years. The difference in current asthma by age group was statistically significant. (See Table H2a.)

Indicator H3: Children's emergency room visits and hospitalizations for asthma and other respiratory causes, ages 0 to 17 years, 1996–2008

About the Indicator: Indicator H3 presents information about the number of children's emergency room visits and hospitalizations for asthma and other respiratory causes. The data are from two national surveys that collect information from hospitals each year. Indicator H3 shows how the rates of children's emergency room visits and hospitalizations for respiratory causes have changed over time.

National Hospital Ambulatory Medical Care Survey and National Hospital Discharge Survey

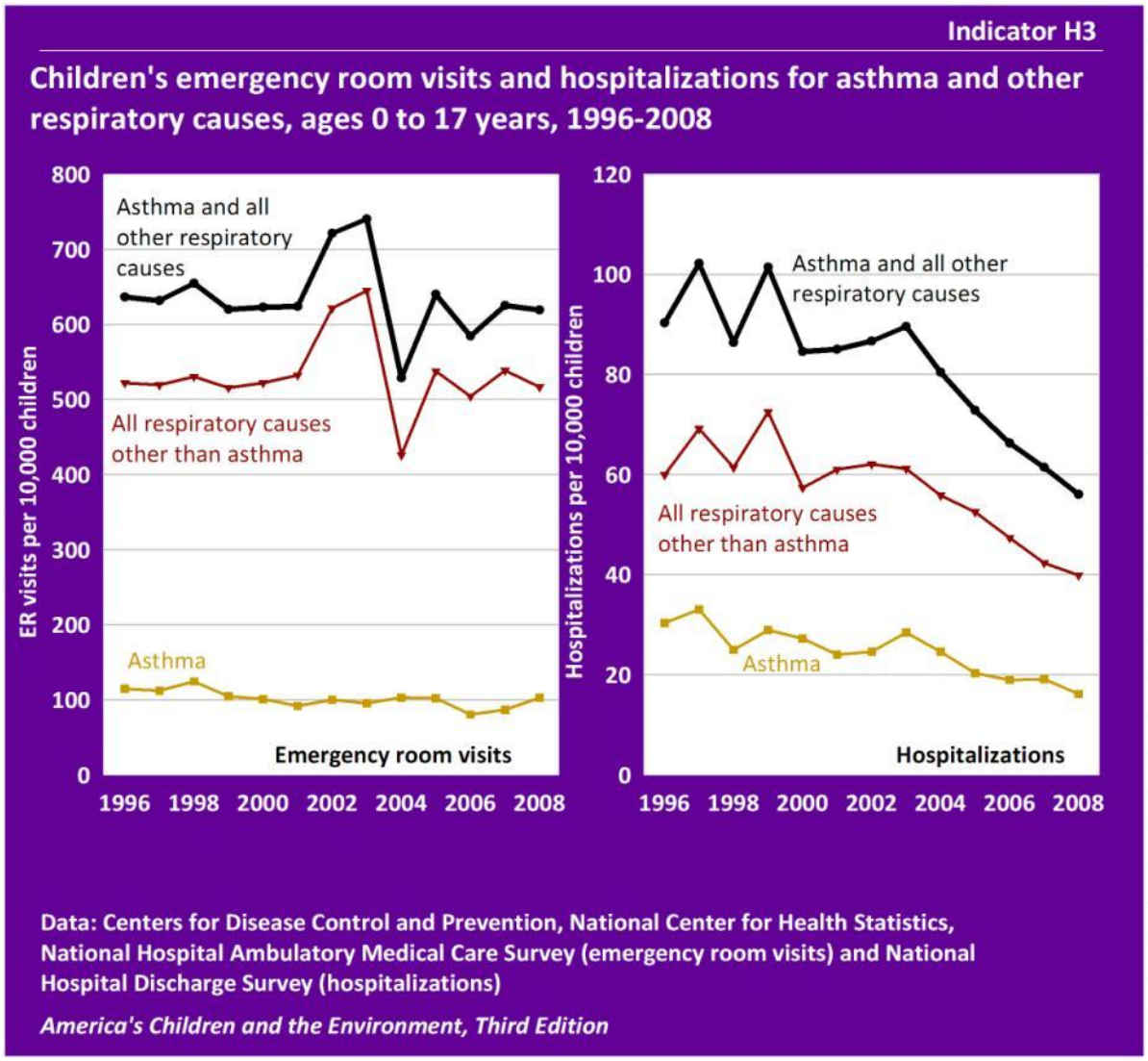
The National Hospital Ambulatory Medical Care Survey (NHAMCS) and the National Hospital Discharge Survey (NHDS), conducted by the National Center for Health Statistics of the Centers for Disease Control and Prevention, provide national data on emergency room visits and hospitalizations. The NHAMCS has collected data for physician diagnoses for visits to hospital emergency rooms and outpatient departments beginning in the year 1992, while the NHDS reports physician diagnoses for discharges from hospitals beginning in the year 1965. The diagnoses in both surveys include asthma and a number of other respiratory conditions. Both surveys exclude federal and military hospitals and report patient demographic information.

Data Presented in the Indicators

Indicator H3 uses data from NHAMCS and NHDS to display emergency room visits and hospitalizations for asthma and other respiratory conditions including bronchitis, pneumonia, and influenza. The top line in each graph represents the total number of children's emergency room visits or hospitalizations for asthma and all other respiratory causes, followed by lines for asthma and for all respiratory causes other than asthma. Indicator H3 presents annual survey results from 1996–2008. 1996 was selected as the initial year for the indicator because not all of the needed hospitalization data for earlier years are available online. The indicator provides data through 2008 because it is the most recent year for which data from both NHAMCS and NHDS are available.

In addition to the data shown in the Indicator H3 graph, supplemental tables show the annual average rates of children's emergency room visits and hospitalizations for asthma and all other respiratory causes, asthma, and all respiratory causes other than asthma (composed of the following subcategories: upper respiratory conditions, pneumonia or influenza, and other lower respiratory conditions besides asthma) by age and race/ethnicity for the years 2005–2008. For emergency room visits, five race/ethnicity groups are presented: White non-Hispanic, Black non-Hispanic, American Indian/Alaska Native non-Hispanic, Asian and Pacific Islander non-Hispanic, and Hispanic. For hospitalizations, race only is reported; the two groups presented are White and Black. The supplemental tables do not include income data, since neither of these surveys includes the patient's income or family income.

Please see the Introduction to the Health section for discussion of statistical significance testing applied to these indicators.



Data characterization

- Data for this indicator are obtained from two ongoing annual surveys conducted by the National Center for Health Statistics.
- Survey data are representative of U.S. population visits to emergency rooms and stays at non-federal hospitals.
- The surveys collect data on physician diagnoses of patients in sampled hospitals, including diagnoses of asthma and other respiratory conditions.

Emergency Room Visits

- In 2008, the rate of emergency room visits for asthma and all other respiratory causes was 619 visits per 10,000 children. The rate of emergency room visits for asthma alone was 103 visits per 10,000 children, and the rate for all respiratory causes other than asthma was 517 visits per 10,000 children.

- The rate of emergency room visits for asthma decreased from 114 visits per 10,000 children in 1996 to 103 visits per 10,000 children in 2008. This decreasing trend was statistically significant.
- Children's emergency room visits for asthma and all other respiratory causes vary widely by race/ethnicity. For the years 2005–2008, Black non-Hispanic children had a rate of 1,240 emergency room visits per 10,000 children, while Hispanic children had a rate of 672 emergency room visits per 10,000 children, American Indian/Alaska Native non-Hispanic children had a rate of 536 emergency room visits per 10,000 children, White non-Hispanic children had a rate of 487 emergency room visits per 10,000 children, and Asian and Pacific Islander non-Hispanic children had a rate of 371 emergency room visits per 10,000 children. (See Table H3a.)
  - The difference in rates of emergency room visits between Black non-Hispanic children and emergency room visits for each of the other race/ethnicity groups was statistically significant.
- Children's emergency room visits for asthma and all other respiratory causes vary widely by age. For the years 2005–2008, infants less than 12 months of age had a rate of 2,142 emergency room visits per 10,000 children, while children 16 to 17 years of age had a rate of 338 emergency room visits per 10,000 children. The differences between age groups were statistically significant. (See Table H3b.)

Hospitalizations

- Between 1996 and 2008, hospitalizations for asthma and for all other respiratory causes decreased from 90 hospitalizations per 10,000 children to 56 hospitalizations per 10,000 children. Between 1996 and 2008, hospitalizations for asthma alone decreased from 30 per 10,000 children to 16 per 10,000 children, and hospitalizations for all other respiratory causes decreased from 60 per 10,000 children to 40 per 10,000 children. These decreasing trends were statistically significant.
- Children's hospitalizations for asthma and all other respiratory causes vary widely by race. For the years 2005–2008, Black children had a rate of 84 hospitalizations for asthma and other respiratory causes per 10,000 children, while White children had a rate of 52 hospitalizations per 10,000 children. This difference was statistically significant. (See Table H3c.)
- Children's hospitalizations for asthma and all other respiratory causes vary widely by age. For the years 2005–2008, infants less than 12 months of age had a rate of 396 hospitalizations per 10,000 children, while children 16 to 17 years of age had a rate of 13 hospitalizations per 10,000 children. The differences between age groups were statistically significant. (See Table H3d.)

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Childhood Cancer

Cancer is not a single disease, but includes a variety of malignancies in which abnormal cells divide in an uncontrolled manner. These cancer cells can invade nearby tissues and can migrate by way of the blood or lymph systems to other parts of the body.[1] The most common childhood cancers are leukemias (cancers of the white blood cells) and cancers of the brain or central nervous system, which together account for more than half of new childhood cancer cases.[2]

Cancer in childhood is rare compared with cancer in adults, but still causes more deaths than any factor, other than injuries, among children from infancy to age 15 years.[2] The annual incidence of childhood cancer has increased slightly over the last 30 years; however, mortality has declined significantly for many cancers due largely to improvements in treatment.[2,3] Part of the increase in incidence may be explained by better diagnostic imaging or changing classification of tumors, specifically brain tumors.[4] However, the President's Cancer Panel recently concluded that the causes of the increased incidence of childhood cancers are not fully understood, and cannot be explained solely by the introduction of better diagnostic techniques. The Panel also concluded that genetics cannot account for this rapid change. The proportion of this increase caused by environmental factors has not yet been determined.[5]

The causes of cancer in children are poorly understood, though in general it is thought that different forms of cancer have different causes. According to scientists at the National Cancer Institute, established risk factors for the development of childhood cancer include family history, specific genetic syndromes (such as Down syndrome), high levels of radiation, and certain pharmaceutical agents used in chemotherapy.[4,6] A number of studies suggest that environmental contaminants may play a role in the development of childhood cancers. The majority of these studies have focused on pesticides and solvents, such as benzene. According to the President's Cancer Panel, "the true burden of environmentally induced cancer has been grossly underestimated."[5]

The development of cancer, or carcinogenesis, is a multistep process leading to the uncontrolled growth and division of cells. This process can begin with an inherited genetic mutation or DNA damage initiated by an exogenous agent, such as exposure to a carcinogenic chemical or radiation. Additionally, many external influences, such as environmental exposures or nutrition, can alter gene expression without changing the DNA sequence.[7] These alterations, referred to as epigenetic changes, can promote alterations in the expression of genes important for controlling cell growth and division.[8,9] Because the initiation of carcinogenesis is a multistep process, multiple factors are thought to contribute to the development of cancer.[9] Newer research suggests that childhood cancer may be caused by a combination of genetic predisposition and environmental exposure.[10-16]

Different types of cancer affect children at different ages. This pattern may reflect the different types of exposures and windows of vulnerability experienced by children as they grow older, and the time between the initiation of cancer and its clinical presentation. Children can be affected by exposures that occur during different developmental stages, such as during infancy and early childhood. Scientific evidence suggests that early childhood cancers may be related to exposure in the womb, or even to parents' exposures prior to conception.[17-21] Furthermore, recent studies suggest that susceptibility to some cancers that arise later in adulthood also may be determined while in the womb.[7]

Leukemia is the most common form of cancer in children. According to the Centers for Disease Control and Prevention, adults and children who undergo chemotherapy and radiation therapy for cancer treatment; take immune suppressing drugs; or have certain genetic conditions, such as Down syndrome; are at a higher risk of developing acute leukemia.[22] Multiple review articles have concluded that ionizing radiation from sources such as x-rays is associated with an increased risk of leukemia.[23-25] CT scans are also an increasing source of ionizing radiation exposure to children,[26] and may be associated with an increased risk of childhood leukemia.[27] Further, studies have consistently shown an approximately 40% increased risk of childhood leukemia after maternal exposure to ionizing radiation during pregnancy.[18,23-25] These confirmed risk factors, however, explain less than 10% of the incidence of childhood leukemia, meaning that the cause is unknown in at least 90% of leukemia cases.[18]

Associations between proximity to extremely low frequency electromagnetic radiation, such as radiation from electrical power lines, and childhood leukemia have been investigated for many years.[5] Some studies suggest an effect on cancer risk, while others do not.[28,29] At this time, a variety of national and international organizations have concluded that the link between exposure to extremely low frequency electromagnetic fields and cancer is controversial or weak.[4,5] Radon is a naturally occurring radioactive element that has been associated with lung cancer; some studies have also found an association between childhood leukemia and radon while other studies have not.[4,30-32] A recent study also reported an association between naturally occurring gamma radiation and childhood leukemia.[33]

Pesticides, solvents, hazardous air pollutants, motor vehicle exhaust, and environmental tobacco smoke have been studied for a potential role in childhood leukemia. Numerous studies have examined the link between parents' (parental), prenatal, and childhood exposures to pesticides and childhood leukemia, and several meta-analyses of these studies have found associations between pesticide exposure and childhood leukemia in both residential and occupational settings.[20,34-46] Recent literature has also suggested an association between childhood exposures to multiple hazardous air pollutants and leukemia.[47-49] A study exploring the relationship between childhood leukemia and hazardous air pollutants (HAPs) in outdoor air found an increased risk for childhood leukemia in census tracts with the highest concentrations of a group of 25 potentially carcinogenic HAPs, including several solvents.[48] Several other studies have found associations between leukemia and surrogate measures of exposure to motor vehicle exhaust, including residential proximity to traffic and gas stations.[18,50-53] However, other studies conducted in California and Denmark did not find an association between these proxy measures of motor vehicle exhaust and childhood leukemia,[54-57] and review studies have concluded that the overall evidence for a relationship is inconclusive.[18,58] According to the U.S. Surgeon General, there is also suggestive evidence that prenatal and postnatal exposure to environmental tobacco smoke can lead to leukemia in children.[59]

Cancers of the nervous system, including brain tumors, are the second most common form of cancer in children. Known risk factors for childhood brain tumors include radiation therapy and certain genetic syndromes, although these factors explain only a small portion of cases.[6] Some studies have also reported an association between prenatal exposure to ionizing radiation and brain tumors while a few smaller studies have not.[25,60,61] Other research reports that head CT scans may be associated with an increased risk of brain tumors in children.[27] Research also suggests that parental, prenatal, and childhood exposure to pesticides may lead to brain tumors in children.[43,45,46] There is suggestive evidence linking prenatal and postnatal exposure to environmental tobacco smoke and childhood brain tumors, according to the U.S. Surgeon General.[59] Many studies have examined whether there is an association between cellular phone use and brain cancer. Some of these studies have found an

association between cellular phone use and some types of brain cancer, while other studies have found no association.[62-69] Because the use of cellular phones by children has only recently become more common, no long-term epidemiological studies of cancer related to cellular phone use by children are available.[5]

Lymphomas, which affect a child's lymph system, are another common form of childhood cancer. The cause of most cases of childhood lymphoma is unknown, but it is clear that children with compromised immune systems are at a greater risk of developing lymphomas.[6] Extensive review studies have found suggestive associations between parental, prenatal, and childhood exposure to pesticides and childhood lymphomas.[43,46] The U.S. Surgeon General has concluded that there is also suggestive evidence linking prenatal and postnatal exposure to environmental tobacco smoke and childhood lymphomas.[59]

Other childhood cancers that have been associated with environmental exposures include thyroid cancer, Wilms' tumor (a type of kidney cancer), Ewing's sarcoma (a cancer of the bone or soft tissue), and melanoma. Some research has reported an increased risk of thyroid cancer in childhood or early adulthood from exposure to ionizing radiation.[70-72] Much of the evidence for this association comes from studies of individuals in areas with high ionizing radiation exposure due to the Chernobyl accident in eastern Europe. While the only known causal factors for Wilms' tumor and Ewing's sarcoma are certain birth defects and genetic conditions, there is limited research indicating that exposure to pesticides may also be a causal factor in the development of Wilms' tumor and Ewing's sarcoma in children.[36,46,73] Although childhood melanoma is rare, the incidence of melanoma is increasing in children, especially in adolescents. Environmental factors associated with melanoma include sunburns, especially in childhood, and increased exposure to ultraviolet (UV) radiation.[74-76] Depletion of the ozone layer causes more ultraviolet radiation to reach the earth's surface. Even though the use of ozone depleting compounds has been largely phased out and the ozone layer will eventually be restored, higher levels of ultraviolet radiation reaching the earth's surface will persist for many years to come.[77,78] Finally, the increased rates of melanoma in adolescent girls and young women may reflect increased UV exposure from sunbathing or from the widespread practice of indoor tanning.[79,80]

The two indicators that follow provide the best nationally representative data available on cancer incidence and mortality among U.S. children over time. Indicator H4 presents cancer incidence and mortality for children ages 0 to 19 years for the period 1992–2009. Indicator H5 presents cancer incidence, by cancer type, for children ages 0 to 19 years for the period 1992–2006. Changes in childhood cancer mortality are most likely reflective of changes in treatment options, rather than environmental exposures. However, showing childhood cancer mortality rates in conjunction with childhood cancer incidence rates highlights the magnitude and severity of childhood cancer and indicates the proportion of children that survive.

Indicator H4 provides an indication of broad trends in childhood cancer over time, while Indicator H5 provides more detailed information about the incidence of specific types of cancer in children.

Indicator H4: Cancer incidence and mortality for children ages 0 to 19 years, 1992–2009

Indicator H5: Cancer incidence for children ages 0 to 19 years by type, 1992–2006

About the Indicators: Indicators H4 and H5 present information about the number of new childhood cancer cases and the number of deaths caused by childhood cancer. The childhood cancer case data come from a program that collects information from tumor registries located in specific geographic regions around the country each year. The childhood cancer death data come from a national database of vital statistics that collects data on numbers and causes of all deaths each year. Indicator H4 shows how the rates of all new childhood cancers and all childhood cancer deaths have changed over time, and Indicator H5 shows how the rates of specific types of childhood cancers have changed over time.

SEER

The National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) Program has provided data on cancer incidence, survival, and prevalence since 1973. SEER obtains its cancer case data from tumor registries in various locations throughout the United States and its cancer mortality data from a national database of vital statistics that collects data on numbers and causes of all deaths each year. Each of the tumor registries collects information for all tumors within a specified geographic region. The sample population covered by the SEER tumor registries is comparable to the general U.S. population in terms of poverty and education. However, the population covered by the SEER tumor registries tends to be more urban and has a higher proportion of foreign-born persons compared with the general U.S. population.[81]

Since its initiation in 1973, the SEER program has expanded to include a greater number of tumor registries. Currently, the SEER program includes data from 18 tumor registries, but complete data from all 18 registries are only available beginning with the year 2000. SEER data are available from 13 different tumor registries that provide data starting in 1992, and represent geographic areas containing 13.8% of the total U.S. population.[82] The registries include the Alaska Native, Atlanta, Connecticut, Detroit, Hawaii, Iowa, Los Angeles, New Mexico, Rural Georgia, San Francisco-Oakland, San Jose-Monterey, Seattle-Puget Sound, and Utah tumor registries.

Data Presented in the Indicators

Childhood cancer incidence refers to the number of new childhood cancer cases reported for a specified period of time. Childhood cancer incidence is shown in Indicator H4 and Indicator H5 as the number of childhood cancer cases reported per million children for one year. The incidence rate is age-adjusted, meaning that each year's incidence calculation uses the age distribution of children from the year 2000. For example, 25.3% of all U.S. children were between the ages of 5 and 9 years in 2000, and this percentage is assumed to be the same for each year from 1992 to 2009. This age adjustment ensures that differences in cancer rates over time are not simply due to changes in the age composition of the population. Indicator H4 also shows childhood cancer mortality as the number of deaths per million children for each year.

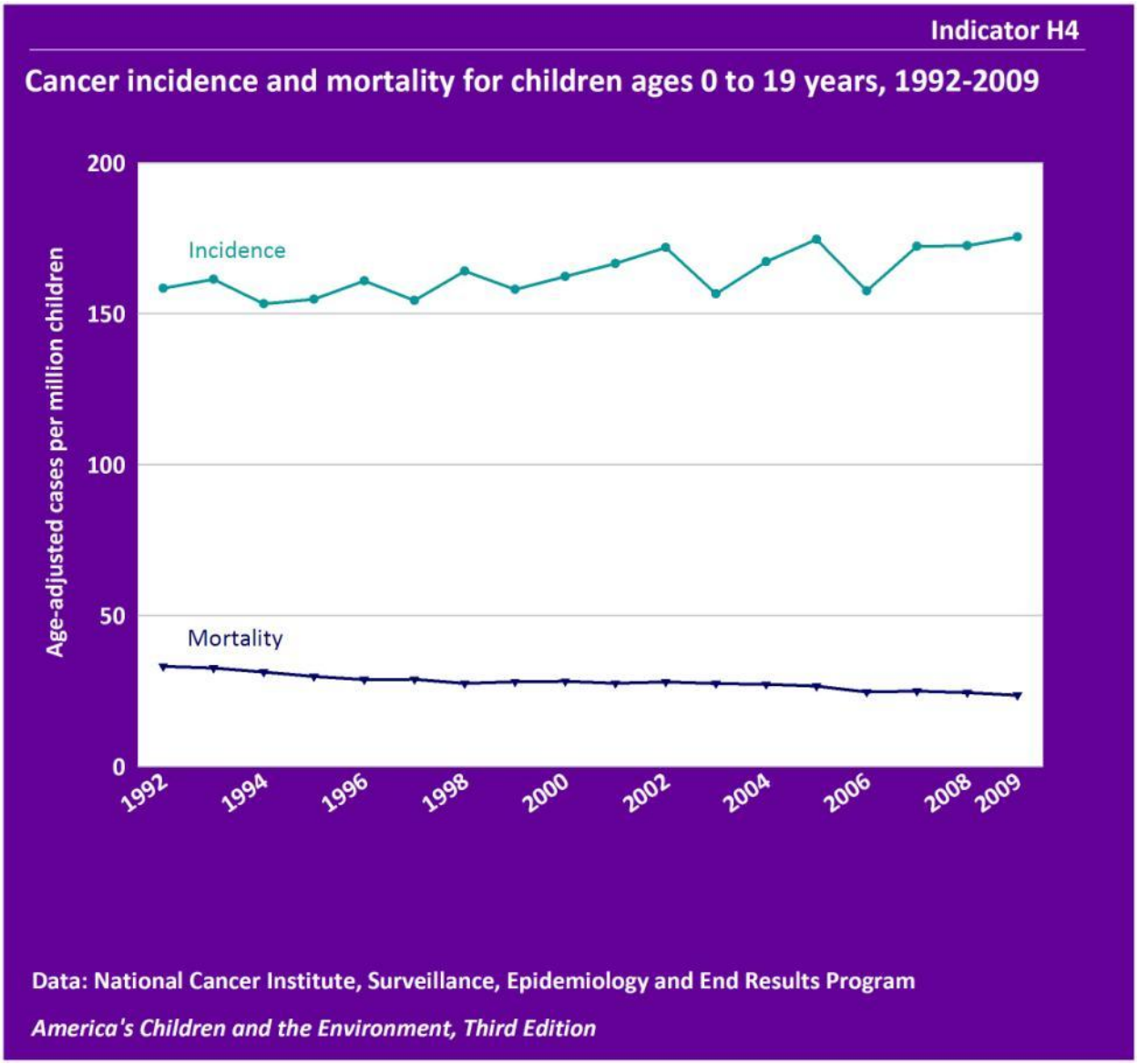
SEER reports the incidence data by single year of age, but reports mortality data in five age groups for children under the age of 20: under 1 year, 1–4, 5–9, 10–14, and 15–19 years. For this reason, both indicators use data for all children 0 to 19 years of age, in contrast to the other indicators in this report that define children as younger than age 18 years.

Trends in the total incidence of childhood cancer, as shown by Indicator H4, are useful for assessing the overall burden of cancer among children. However, broad trends mask changes in the frequency of specific types of cancers that often have patterns that diverge from the overall trend. Moreover, environmental factors may be more likely to contribute to some childhood cancers than to others. Indicator H5 shows trends in incidence for specific types of childhood cancers.

Some types of childhood cancers are very rare, and as such the yearly incidence is particularly low and variable. Due to this fact, Indicator H5 shows the incidence of individual childhood cancers in groupings of three years. Each bar in the graph represents the annual number of cases of that specific cancer diagnosed per million children, calculated as the average number of cases per year divided by the average population of children (in millions) per year for each three-year period.

The SEER cancer incidence data for the 13 longer-established registries, instead of all 18, were used to develop the H4 and H5 indicators because this allowed for more comprehensive trend analysis while still covering a substantial portion of the population. Indicator H4 begins with the earliest available SEER13 incidence data from 1992 and ends with 2009. Childhood cancer mortality data for 1992 to 2009 are also used for indicator H4. Indicator H5 presents data for the series of three-year periods beginning in 1992 and ending in 2006. In addition to the data shown in the Indicator H4 graph, supplemental tables show childhood cancer incidence and mortality by race/ethnicity and sex, as well as childhood cancer incidence by age group. These data tables use data from the three most current years shown in Indicator H4, which are 2007–2009. Combining three years of data allows for more statistically reliable estimates by race/ethnicity, sex, and age group. Five race/ethnicity groups are used in the supplemental tables for Indicator H4: White non-Hispanic, Black non-Hispanic, American Indian/Alaska Native non-Hispanic, Asian or Pacific Islander non-Hispanic, and Hispanic. In addition to the data shown in the Indicator H5 graph, a supplemental table shows childhood cancer incidence by cancer type and age group.

Please see the Introduction to the Health section for discussion of statistical significance testing applied to these indicators.

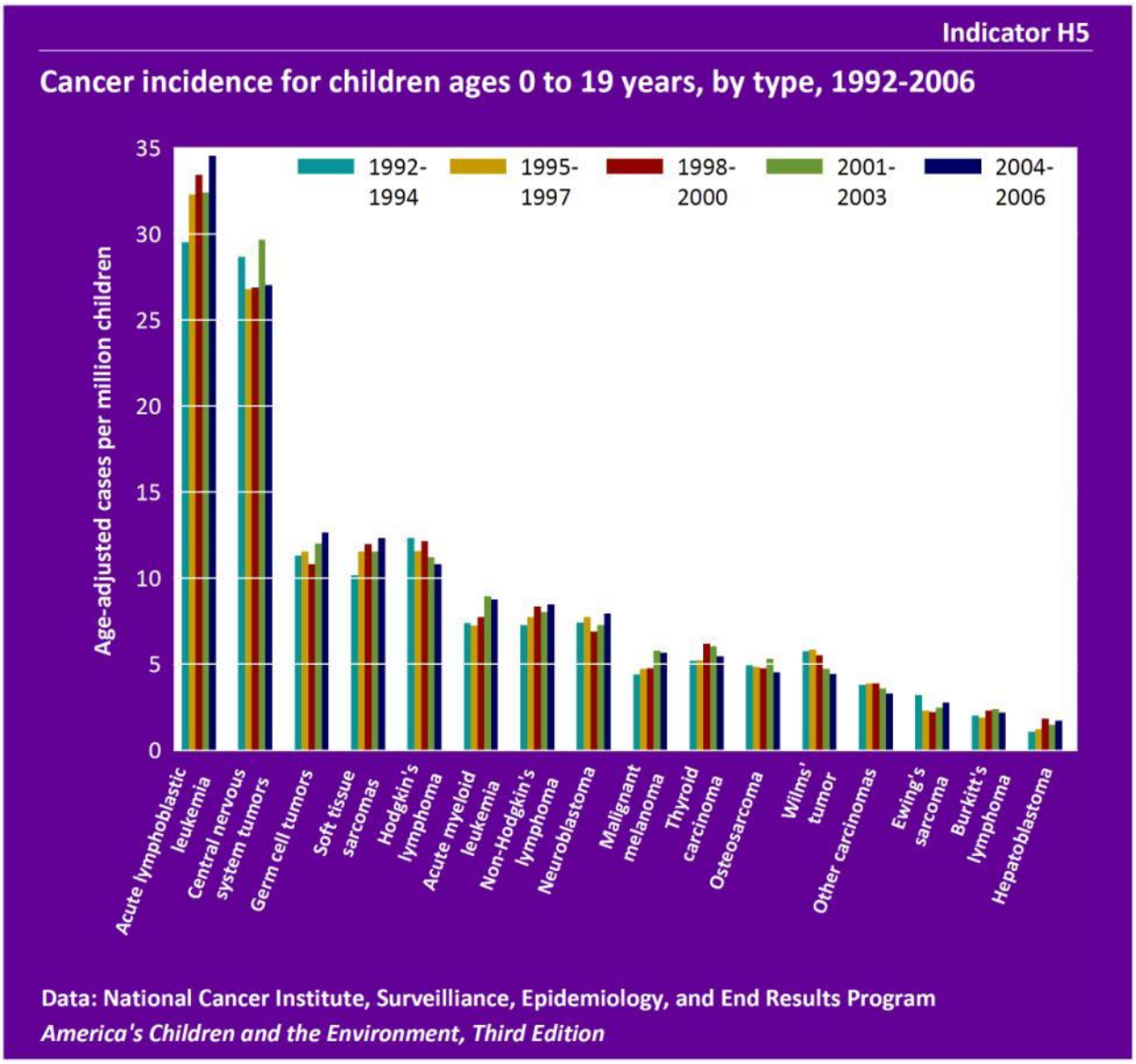


Data characterization

- Cancer incidence data for this indicator are obtained from a database of 13 regional tumor registries located throughout the country, maintained by the National Cancer Institute.
- The population covered by the 13 registries is comparable to the general U.S. population regarding poverty and education, but is more urban and has more foreign-born persons.
- Cancer mortality data for this indicator are obtained from a database of all death certificates in the United States; cause of death is recorded on the death certificates.

- The age-adjusted annual incidence of cancer ranged from 153 to 161 cases per million children between 1992 and 1994 and from 172 to 175 cases per million children between 2007 and 2009. This increasing trend from 1992–2009 was statistically significant.

- Childhood cancer mortality decreased from 33 deaths per million children in 1992 to 24 deaths per million children in 2009, a statistically significant decreasing trend.
- Childhood cancer incidence and mortality rates were generally higher for boys than for girls. In 2007–2009, rates of cancer incidence and mortality for boys were 183 cases per million and 26 deaths per million, compared with 163 cases per million and 22 deaths per million for girls. These differences by sex were statistically significant for cancer incidence (after adjustment for age and race/ethnicity) and cancer mortality. (See Tables H4a and H4b.)
- In 2007–2009, the difference in cancer incidence between boys and girls was not consistent for all races/ethnicities. No statistically significant difference in cancer incidence by sex was seen among Black non-Hispanic children or Asian or Pacific Islander non-Hispanic children. Among American Indian and Alaska Native non-Hispanic children, cancer incidence was greater for girls than for boys, although this difference was not statistically significant. Cancer incidence was greater for boys than for girls and statistically significant (after adjustment for age) among White non-Hispanic children and Hispanic children. (See Table H4a.)
- In 2007–2009, childhood cancer incidence was highest among White non-Hispanic children at 188 cases per million. Hispanic children had an incidence rate of 169 cases per million, Asian and Pacific Islander non-Hispanic children had an incidence rate of 152 cases per million, American Indian and Alaska Native non-Hispanic children had an incidence rate of 137 cases per million, and Black non-Hispanic children had an incidence rate of 133 cases per million. (See Table H4a.)
  - The cancer incidence rate for White non-Hispanic children was statistically significantly higher than the rates of each of the other race/ethnicity categories after accounting for differences by age and sex. The cancer incidence rate for Black non-Hispanic children was also statistically significantly lower than the rates for Hispanic children and Asian and Pacific Islander non-Hispanic children after adjustment for differences by age and sex. The cancer incidence rate for Asian and Pacific Islander non-Hispanic was also statistically significantly lower than the rate for Hispanic children after adjustment for differences by age and sex. The remaining differences between race/ethnicity groups were not statistically significant.
- Childhood cancer incidence rates vary by age. In 2007–2009, children under 5 and those of ages 15 to 19 years experienced the highest incidence rates of cancer at approximately 208 and 232 cases per million, respectively. Children ages 5 to 9 years and 10 to 14 years had lower incidence rates at 117 and 139 cases per million, respectively. These differences among age groups were statistically significant. (See Table H4c.)



Data characterization

- Data for this indicator are obtained from a database of 13 regional tumor registries located throughout the country, maintained by the National Cancer Institute.
- The population covered by the 13 registries is comparable to the general U.S. population regarding poverty and education, but is more urban and has more foreign-born persons.

- Leukemia, which includes acute lymphoblastic leukemia and acute myeloid leukemia, was the most common cancer diagnosis for children from 2004–2006, representing 28% of total cancer cases. Incidence of acute lymphoblastic (lymphocytic) leukemia was 30 cases per million in 1992–1994 and 35 cases per million in 2004–2006. The rate of acute myeloid (myelogenous) leukemia was 7 cases per million in 1992–1994 and 9 cases per million in 2004–2006.
  - The increasing trend for incidence of acute lymphoblastic leukemia was statistically significant after accounting for differences by age, sex, and race/ethnicity. The trend for acute myeloid leukemia was not statistically significant.
- Central nervous system tumors represented 18% of childhood cancers in 2004–2006. The incidence of central nervous system tumors was 27 cases per million in 2004–2006, with no statistically significant trend for 1992–2006.
- Lymphomas, which include Hodgkin's lymphoma, non-Hodgkin's lymphoma, and Burkitt's lymphoma, represented 14% of childhood cancers in 2004–2006. Incidence of Hodgkin's lymphoma was 12 cases per million in 1992–1994 and 11 per million in 2004–2006. There were approximately 7 cases of non-Hodgkin's lymphoma per million children in 1992–1994 and 9 per million in 2004–2006. Incidence of Burkitt's lymphoma remained constant from 1992–2006 (2 cases per million children). The increasing trend in the incidence rate of non-Hodgkin's lymphoma was statistically significant, while there was no statistically significant trend in the incidence rate of Hodgkin's lymphoma or Burkitt's lymphoma.
- Between the years 1992 and 2006, increasing trends in the incidence of soft tissue sarcomas, malignant melanomas, and hepatoblastomas were statistically significant, as was the decreasing trend in the incidence of Wilms' tumor (tumors of the kidney). There was no statistically significant trend in the incidence rate of thyroid carcinomas, other and unspecified carcinomas, germ cell tumors, osteosarcomas, Ewing's sarcomas, or neuroblastomas.
  - The increasing trend in the incidence rate of hepatoblastomas was statistically significant after accounting for differences by age, sex, and race/ethnicity.
- Different types of cancer affect children at different ages. The incidence of neuroblastomas and Wilms' tumor (tumors of the kidney) was highest for young children (ages 0 to 4 years). Leukemias occur in all age groups, but the incidence is highest among 0- to 4-year-olds. The incidence of Hodgkin's and non-Hodgkin's lymphomas, thyroid carcinomas, malignant melanomas, other and unspecified carcinomas, germ cell tumors, and osteosarcomas was higher in those 15 to 19 years old. Differences among age groups were statistically significant for each of these cancer types. (See Table H5a.)

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Neurodevelopmental Disorders

Neurodevelopmental disorders are disabilities associated primarily with the functioning of the neurological system and brain. Examples of neurodevelopmental disorders in children include attention-deficit/hyperactivity disorder (ADHD), autism, learning disabilities, intellectual disability (also known as mental retardation), conduct disorders, cerebral palsy, and impairments in vision and hearing. Children with neurodevelopmental disorders can experience difficulties with language and speech, motor skills, behavior, memory, learning, or other neurological functions. While the symptoms and behaviors of neurodevelopmental disabilities often change or evolve as a child grows older, some disabilities are permanent. Diagnosis and treatment of these disorders can be difficult; treatment often involves a combination of professional therapy, pharmaceuticals, and home- and school-based programs.

Based on parental responses to survey questions, approximately 15% of children in the United States ages 3 to 17 years were affected by neurodevelopmental disorders, including ADHD, learning disabilities, intellectual disability, cerebral palsy, autism, seizures, stuttering or stammering, moderate to profound hearing loss, blindness, and other developmental delays, in 2006–2008.[1] Among these conditions, ADHD and learning disabilities had the greatest prevalence. Many children affected by neurodevelopmental disorders have more than one of these conditions: for example, about 4% of U.S. children have both ADHD and a learning disability.[2] Some researchers have stated that the prevalence of certain neurodevelopmental disorders, specifically autism and ADHD, has been increasing over the last four decades.[3-7] Long-term trends in these conditions are difficult to detect with certainty, due to a lack of data to track prevalence over many years as well as changes in awareness and diagnostic criteria. However, some detailed reviews of historical data have concluded that the actual prevalence of autism seems to be rising.[4,8-10] Surveys of educators and pediatricians have reported a rise in the number of children seen in classrooms and exam rooms with behavioral and learning disorders.[11-13]

Genetics can play an important role in many neurodevelopmental disorders, and some cases of certain conditions such as intellectual disability are associated with specific genes. However, most neurodevelopmental disorders have complex and multiple contributors rather than any one clear cause. These disorders likely result from a combination of genetic, biological, psychosocial and environmental risk factors. A broad range of environmental risk factors may affect neurodevelopment, including (but not limited to) maternal use of alcohol, tobacco, or illicit drugs during pregnancy; lower socioeconomic status; preterm birth; low birthweight; the physical environment; and prenatal or childhood exposure to certain environmental contaminants.[14-21]

Lead, methylmercury, and PCBs are widespread environmental contaminants associated with adverse effects on a child's developing brain and nervous system in multiple studies. The National Toxicology Program (NTP) has concluded that childhood lead exposure is associated with reduced cognitive function, including lower intelligence quotient (IQ) and reduced academic achievement.[22] The NTP has also concluded that childhood lead exposure is associated with attention-related behavioral problems (including inattention, hyperactivity, and diagnosed attention-deficit/hyperactivity disorder) and increased incidence of problem behaviors (including delinquent, criminal, or antisocial behavior).[22]

EPA has determined that methylmercury is known to have neurotoxic and developmental effects in humans.[23] Extreme cases of such effects were seen in people prenatally exposed during two high-dose

mercury poisoning events in Japan and Iraq, who experienced severe adverse health effects such as cerebral palsy, mental retardation, deafness, and blindness.[24-26] Prospective cohort studies have been conducted in island populations where frequent fish consumption leads to methylmercury exposure in pregnant women at levels much lower than in the poisoning incidents but much greater than those typically observed in the United States. Results from such studies in New Zealand and the Faroe Islands suggest that increased prenatal mercury exposure due to maternal fish consumption was associated with adverse effects on intelligence and decreased functioning in the areas of language, attention, and memory.[26-32] These associations were not seen in initial results reported from a similar study in the Seychelles Islands.[33] However, further studies in the Seychelles found associations between prenatal mercury exposure and some neurodevelopmental deficits after researchers had accounted for the developmental benefits of fish consumption.[34-36] More recent studies conducted in the United States have found associations between neurodevelopmental effects and blood mercury levels within the range typical for U.S. women, after accounting for the beneficial effects of fish consumption during pregnancy.[32,37,38]

Several studies of children who were prenatally exposed to elevated levels of polychlorinated biphenyls (PCBs) have suggested linkages between these contaminants and neurodevelopmental effects, including lowered intelligence and behavioral deficits such as inattention and impulsive behavior.[39-44] Studies have also reported associations between PCB exposure and deficits in learning and memory.[39,45] Most of these studies found that the effects are associated with exposure in the womb resulting from the mother having eaten food contaminated with PCBs,[46-51] although some studies have reported relationships between adverse effects and PCB exposure during infancy and childhood.[45,51-53] Although there is some inconsistency in the epidemiological literature, several reviews of the literature have found that the overall evidence supports a concern for effects of PCBs on children's neurological development.[52,54-58] The Agency for Toxic Substances and Disease Registry has determined that "Substantial data suggest that PCBs play a role in neurobehavioral alterations observed in newborns and young children of women with PCB burdens near background levels."[59] In addition, adverse effects on intelligence and behavior have been found in children of women who were highly exposed to mixtures of PCBs, chlorinated dibenzofurans, and other pollutants prior to conception.[60-63]

A wide variety of other environmental chemicals have been identified as potential concerns for childhood neurological development, but have not been as well studied for these effects as lead, mercury, and PCBs. Concerns for these additional chemicals are based on both laboratory animal studies and human epidemiological research; in most cases, the epidemiological studies are relatively new and the literature is just beginning to develop. Among the chemicals being studied for potential effects on childhood neurological development are organophosphate pesticides, polybrominated diphenyl ether flame retardants (PBDEs), phthalates, bisphenol A (BPA), polycyclic aromatic hydrocarbons (PAHs), arsenic, and perchlorate. Exposure to all of these chemicals is widespread in the United States for both children and adults.[64]

Organophosphate pesticides can interfere with the proper function of the nervous system when exposure is sufficiently high.[65] Many children may have low capacity to detoxify organophosphate pesticides through age 7 years.[66] In addition, recent studies have reported an association between prenatal organophosphate exposure and childhood ADHD in a U.S. community with relatively high exposures to organophosphate pesticides,[67] as well as with exposures found within the general U.S. population.[68] Other recent studies have described associations between prenatal organophosphate

pesticide exposures and a variety of neurodevelopmental deficits in childhood, including reduced IQ, perceptual reasoning, and memory.[69-71]

Studies of certain PBDEs have found adverse effects on behavior, learning, and memory in laboratory animals.[72-74] A recent epidemiological study in New York City reported significant associations between children's prenatal exposure to PBDEs and reduced performance on IQ tests and other tests of neurological development in 6-year-old children.[75] Another study in the Netherlands reported significant associations between children's prenatal exposure to PBDEs and reduced performance on some neurodevelopmental tests in 5- and 6-year-old children, while associations with improved performance were observed for other tests.[76]

Two studies of a group of New York City children ages 4 to 9 years reported associations between prenatal exposure to certain phthalates and behavioral deficits, including effects on attention, conduct, and social behaviors.[77,78] Some of the behavioral deficits observed in these studies are similar to those commonly displayed in children with ADHD and conduct disorder. Studies conducted in South Korea of children ages 8 to 11 years reported that children with higher levels of certain phthalate metabolites in their urine were more inattentive and hyperactive, displayed more symptoms of ADHD, and had lower IQ compared with those who had lower levels.[79,80] The exposure levels in these studies are comparable to typical exposures in the U.S. population.

In 2008, the NTP concluded that there is "some concern" for effects of early-life (including prenatal) BPA exposure on brain development and behavior, based on findings of animal studies conducted at relatively low doses.[81] An epidemiological study conducted in Ohio reported an association between prenatal exposure to BPA and effects on children's behavior (increased hyperactivity and aggression) at age 2 years.[82] Another study of prenatal BPA exposure in New York City reported no association between prenatal BPA exposure and social behavior deficits in testing conducted at ages 7 to 9 years.[78]

A series of recent studies conducted in New York City has reported that children of women who were exposed to increased levels of polycyclic aromatic hydrocarbons (PAHs, produced when gasoline and other materials are burned) during pregnancy are more likely to have experienced adverse effects on neurological development (for example, reduced IQ and behavioral problems).[83,84]

Early-life exposure to arsenic has been associated with measures of reduced cognitive function, including lower scores on tests that measure neurobehavioral and intellectual development, in four studies conducted in Asia; however there are some inconsistencies in the findings of these studies.[85] These findings are from countries where arsenic levels in drinking water are generally much higher than in the United States due to high levels of naturally occurring arsenic in groundwater.[86]

Perchlorate is a naturally occurring and man-made chemical that has been found in drinking water[87] and foods[88,89] in the United States. Exposure to elevated levels of perchlorate inhibits iodide uptake into the thyroid gland, thus possibly disrupting the function of the thyroid and potentially leading to a reduction in the production of thyroid hormone.[90,91] Moderate deficits in maternal thyroid hormone levels during early pregnancy have been linked to reduced childhood IQ scores and other neurodevelopmental effects.[92-94]

Interactions of environmental contaminants and other environmental factors may combine to increase the risk of neurodevelopmental disorders. For example, exposure to lead may have stronger effects on neurodevelopment among children with lower socioeconomic status.[21,95]

A child's brain and nervous system are vulnerable to adverse impacts from pollutants because they go through a long developmental process beginning shortly after conception and continuing through adolescence.[96,97] This complex developmental process requires the precise coordination of cell growth and movement, and may be disrupted by even short-term exposures to environmental contaminants if they occur at critical stages of development. This disruption can lead to neurodevelopmental deficits that may have an effect on the child's achievements and behavior even when they do not result in a diagnosable disorder.

Attention Deficit/Hyperactivity Disorder (ADHD)

Attention-deficit/hyperactivity disorder (ADHD) is a disruptive behavior disorder characterized by symptoms of inattention and/or hyperactivity-impulsivity, occurring in several settings and more frequently and severely than is typical for other individuals in the same stage of development.[98] ADHD can make family and peer relationships difficult, diminish academic performance, and reduce vocational achievement.

As the medical profession has developed a greater understanding of ADHD through the years, the name of this condition has changed. The American Psychiatric Association adopted the name "attention deficit disorder" in the early 1980s and revised it to "attention-deficit/hyperactivity disorder" in 1987.[99] Many children with ADHD have a mix of inattention and hyperactivity/impulsivity behaviors, while some may display primarily hyperactive behavior traits, and others display primarily inattentive traits. It is possible for an individual's primary symptoms of ADHD to change over time.[20] Children with ADHD frequently have other disorders, with parents reporting that about half of children with ADHD have a learning disability and about one in four have a conduct disorder.[2,100]

Other disorders, including anxiety disorders, depression, and learning disabilities, can be expressed with signs and symptoms that resemble those of ADHD. A diagnosis of ADHD requires a certain amount of judgment on the part of a doctor, similar to diagnosis of other mental disorders. Despite the variability among children diagnosed with the disorder and the challenges involved in diagnosis, ADHD has good clinical validity, meaning that impaired children share similarities, exhibit symptoms, respond to treatment, and are recognized with general consistency across clinicians.[20]

A great deal of research on ADHD has focused on aspects of brain functioning that are related to the behaviors associated with ADHD. Although this research is not definitive, it has found that children with ADHD generally have trouble with certain skills involved in problem-solving (referred to collectively as executive function). These skills include working memory (keeping information in mind while briefly doing something else), planning (organizing a sequence of activities to complete a task), response inhibition (suppressing immediate responses when they are inappropriate), and cognitive flexibility (changing an approach when a situation changes). Children with ADHD also generally have problems in maintaining sustained attention to a task (referred to as vigilance), and/or maintaining readiness to respond to new information (referred to as alertness).[20,101,102]

While uncertainties remain, findings to date indicate that ADHD is caused by combinations of genetic and environmental factors. [20,103-106] Much of the research on environmental factors has focused on the fetal environment. Maternal smoking during pregnancy has been associated with increased risk of ADHD in the child in numerous studies, however, this continues to be an active area of research as scientists consider whether other factors related to smoking (e.g., genetic factors, maternal mental health, stress, alcohol use, and low birth weight) may be responsible for associations attributed to smoking.[17,19,107] Findings regarding ADHD and maternal consumption of alcohol during pregnancy are considered more limited and inconsistent.[19,20] Preterm birth and low birth weight have also been found to increase the likelihood that a child will have ADHD.[16,18,20] Psychosocial adversity (representing factors such as low socioeconomic status and in-home conflict) in childhood may also play a role in ADHD.[108]

The potential role of environmental contaminants in contributing to ADHD, either alone or in conjunction with certain genetic susceptibilities or other environmental factors, is becoming better understood as a growing number of studies look explicitly at the relationship between ADHD and exposures to environmental contaminants.

Among environmental contaminants known or suspected to be developmental neurotoxicants, lead has the most extensive evidence of a potential contribution to ADHD. A number of recent epidemiological studies (all published since 2006, with data gathered beginning in 1999 or more recently) conducted in the United States and Asia have reported relationships between increased levels of lead in a child's blood and increased likelihood of ADHD.[55,109-115] In most of these studies, blood lead levels were comparable to levels observed currently in the United States. The potential contribution of childhood lead exposure to the risk of ADHD may be amplified in children of women who smoked cigarettes during pregnancy.[110] In addition, several studies have reported relationships between blood lead levels and the aspects of brain functioning that are most affected in children with ADHD, including sustained attention, alertness, and problem-solving skills (executive functions, specifically cognitive flexibility, working memory, planning, and response inhibition).[22,44,55,116-119] Similar results have been observed in laboratory animal studies.[55,96,120-122] The NTP has concluded that childhood lead exposure is "associated with increased diagnosis of attention-related behavioral problems."[22]

Although no studies evaluating a potential association between PCBs and ADHD itself have been published, a study in Massachusetts reported a relationship between levels of PCBs measured in cord blood and increased ADHD-like behaviors observed by teachers in children at ages 7 to 11 years. PCB levels in this study were generally lower than those measured in other epidemiological studies of PCBs and childhood neurological development.[40] Other research findings also suggest that PCBs may play a role in contributing to ADHD. Several studies in U.S. and European populations, most having elevated exposure to PCBs through the diet, have found generally consistent associations with aspects of brain function that are most affected in children with ADHD, including alertness and problem-solving skills (executive functions, specifically response inhibition, working memory, cognitive flexibility, and planning).[54,55] Studies in laboratory animals have similar findings regarding the mental functions affected by PCB exposure.[55,96]

Studies of other environmental chemicals reporting associations with ADHD or related outcomes have been published in recent years, but findings tend to be much more limited than for lead and PCBs. Findings for phthalates and organophosphate pesticides were noted above. In addition, three studies have reported associations between ADHD or impulsivity and concentrations of certain perfluorinated

chemicals measured in the blood of children.[123-125] Studies of mercury have produced generally mixed findings of associations with ADHD or related symptoms and mental functions.[29,111,118,126-128]

Learning Disability

Learning disability (or learning disorder) is a general term for a neurological disorder that affects the way in which a child's brain can receive, process, retain, and respond to information. A child with a learning disability may have trouble learning and using certain skills, including reading, writing, listening, speaking, reasoning, and doing math, although learning disabilities vary from child to child. Children with learning disabilities usually have average or above-average intelligence, but there are differences in the way their brains process information.[129]

As with many other neurodevelopmental disorders, the causes of learning disabilities are not well understood. Often learning disabilities run in the family, suggesting that heredity may play a role in their development. Problems during pregnancy and birth, such as drug or alcohol use during pregnancy, low birth weight, lack of oxygen, or premature or prolonged labor, may also lead to learning disabilities.[130]

As is the case with other neurodevelopmental outcomes, there are generally many more studies of lead exposure that are relevant to learning disabilities than for other environmental contaminants. Several studies have found associations between lead exposure and learning disabilities or reduced classroom performance that are independent of IQ.[119,120,131-133] Exposures to lead have been associated with impaired memory and difficulties or impairments in rule learning, following directions, planning, verbal abilities, speech processing, and classroom performance in children.[22,119,131,134-137] Other findings that may indicate contributions from environmental contaminants to learning disabilities include a study that found associations of both maternal smoking during pregnancy and childhood exposure to environmental tobacco smoke with parent report of a child with a learning disability diagnosis;[138] associations of prenatal mercury exposure with dysfunctions in children's language abilities and memory,[29,30] and associations of prenatal PCB exposure with poorer concentration and memory deficits compared with unexposed children.[39,45]

Autism Spectrum Disorders

Autism spectrum disorders (ASDs) are a group of developmental disabilities defined by significant social, communication, and behavioral impairments. The term "spectrum disorders" refers to the fact that although people with ASDs share some common symptoms, ASDs affect different people in different ways, with some experiencing very mild symptoms and others experiencing severe symptoms. ASDs encompass autistic disorder and the generally less severe forms, Asperger's syndrome and pervasive developmental disorder-not otherwise specified (PDD-NOS). Children with ASDs may lack interest in other people, have trouble showing or talking about feelings, and avoid or resist physical contact. A range of communication problems are seen in children with ASDs: some speak very well, while many children with an ASD do not speak at all. Another hallmark characteristic of ASDs is the demonstration of restrictive or repetitive interests or behaviors, such as lining up toys, flapping hands, rocking his or her body, or spinning in circles.[139]

To date, no single risk factor sufficient to cause ASD has been identified; rather each case is likely to be caused by the combination of multiple genetic and environmental risk factors.[140-142] Several ASD research findings and hypotheses may imply an important role for environmental contaminants. First, there has been a sharp upward trend in reported prevalence that cannot be fully explained by factors such as younger ages at diagnosis, migration patterns, changes in diagnostic criteria, inclusion of milder cases, or increased parental age.[8,9,143-146] Also, the neurological signaling systems that are impaired in children with ASDs can be affected by certain environmental chemicals. For example, several pesticides are known to interfere with acetylcholine (Ach) and γ-aminobutyric acid (GABA) neurotransmission, chemical messenger systems that have been altered in certain subsets of autistic individuals.[147] Some studies have reported associations between certain pharmaceuticals taken by pregnant women and increased incidence of autism, which may suggest that there are biological pathways by which other chemical exposures during pregnancy could increase the risk of autism.[148]

Furthermore, some of the identified genetic risk factors for autism are *de novo* mutations, meaning that the genetic defect is not present in either of the parents' genes, yet can be found in the genes of the child when a new genetic mutation forms in a parent's germ cells (egg or sperm), potentially from exposure to contaminants.[140,142,149,150] Many environmental contaminants have been identified as agents capable of causing mutations in DNA, by leading to oxidative DNA damage and by inhibiting the body's normal ability to repair DNA damage.[151] Some children with autism have been shown to display markers of increased oxidative stress, which may strengthen this line of reasoning.[152-154] Many studies have linked increasing paternal and maternal age with increased risk of ASDs.[144,146,155-157] The role of parental age in increased autism risk might be explained by evidence that shows advanced parental age can contribute significantly to the frequency of *de novo* mutations in a parent's germ cells.[151,158,159] Advanced parental age signifies a longer period of time when environmental exposures may act on germ cells and cause DNA damage and *de novo* mutations. Finally, a recent study concluded that the role of genetic factors in ASDs has been overestimated, and that environmental factors play a greater role than genetic factors in contributing to autism.[141] This study did not evaluate the role of any particular environmental factors, and in this context "environmental factors" are defined broadly to include any influence that is not genetic.

Studies, limited in number and often limited in research design, have examined the possible role that certain environmental contaminants may play in the development of ASDs. A number of these studies have focused on mercury exposures. Earlier studies reported higher levels of mercury in the blood, baby teeth, and urine of children with ASDs compared with control children;[160-162] however, another more recent study reported no difference in the blood mercury levels of children with autism and typically developing children.[163] Proximity to industrial and power plant sources of environmental mercury was reported to be associated with increased autism prevalence in a study conducted in Texas.[164]

Thimerosal is a mercury-containing preservative that is used in some vaccines to prevent contamination and growth of harmful bacteria in vaccine vials. Since 2001, thimerosal has not been used in routinely administered childhood vaccines, with the exception of some influenza vaccines.[165] The Institute of Medicine has rejected the hypothesis of a causal relationship between thimerosal-containing vaccines and autism.[166]

Some studies have also considered air pollutants as possible contributors to autism. A study conducted in the San Francisco Bay Area reported an association between the amount of certain airborne pollutants at a child's place of birth (mercury, cadmium, nickel, trichloroethylene, and vinyl chloride) and

the risk for autism, but a similar study in North Carolina and West Virginia did not find such a relationship.[167,168] Another study in California reported that mothers who lived near a freeway at the time of delivery were more likely to have children diagnosed with autism, suggesting that exposure to traffic-related air pollutants may play a role in contributing to ASDs.[169]

Finally, a study in Sweden reported an increased risk of ASDs in children born to families living in homes with polyvinyl chloride (PVC) flooring, which is a source of certain phthalates in indoor environments.[170]

Intellectual Disability (Mental Retardation)

The most commonly used definitions of intellectual disability (also referred to as mental retardation) emphasize subaverage intellectual functioning before the age of 18, usually defined as an IQ less than 70 and impairments in life skills such as communication, self-care, home living, and social or interpersonal skills. Different severity categories, ranging from mild to severe retardation, are defined on the basis of IQ scores.[171,172]

"Intellectual disability" is used as the preferred term for this condition in the disabilities sector, but the term "mental retardation" continues to be used in the contexts of law and public policy when designating eligibility for state and federal programs.[171]

Researchers have identified some causes of intellectual disability, including genetic disorders, traumatic injuries, and prenatal events such as maternal infection or exposure to alcohol.[172,173] However, the causes of intellectual disability are unknown in 30–50% of all cases.[173] The causes are more frequently identified for cases of severe retardation (IQ less than 50), whereas the cause of mild retardation (IQ between 50 and 70) is unknown in more than 75% of cases.[174,175] Exposures to environmental contaminants could be a contributing factor to the cases of mild retardation where the cause is unknown. Exposure to high levels of lead and mercury have been associated with intellectual disability.[23,176-178] Furthermore, lead, mercury, and PCBs all have been found to have adverse effects on intelligence and cognitive functioning in children,[22,26,43,52,179] and recent studies have reported associations of a number of other environmental contaminants with childhood IQ deficits, including organophosphate pesticides,[69-71] PBDEs,[75] phthalates,[79] and PAHs.[83,180] Exposure to environmental contaminants that reduce IQ has the potential to increase the proportion of the population with IQ less than 70, thus increasing the incidence of intellectual disability in an exposed population.[181-183]

Indicators in this Section

The four indicators that follow provide the best nationally representative data available on the prevalence of neurodevelopmental disorders among U.S. children over time. The indicators present the number of children ages 5 to 17 years reported to have ever been diagnosed with ADHD (Indicator H6), learning disabilities (Indicator H7), autism (Indicator H8), and intellectual disability (Indicator H9). These four conditions are examples of neurodevelopmental disorders that may be influenced by exposures to environmental contaminants. Intellectual disability and learning disabilities are disorders in which a child's cognitive or intellectual development is affected, and ADHD is a disorder in which a child's behavioral development is affected. Autism spectrum disorders are disorders in which a child's behavior, communication, and social skills are affected.

Indicator H6: Percentage of children ages 5 to 17 years reported to have attention-deficit/hyperactivity disorder, by sex, 1997–2010

Indicator H7: Percentage of children ages 5 to 17 years reported to have a learning disability, by sex, 1997–2010

Indicator H8: Percentage of children ages 5 to 17 years reported to have autism, 1997–2010

Indicator H9: Percentage of children ages 5 to 17 years reported to have intellectual disability (mental retardation), 1997–2010

About the Indicators: Indicators H6, H7, H8, and H9 present information about the number of children who are reported to have ever been diagnosed with four different neurodevelopmental disorders: attention-deficit/hyperactivity disorder (ADHD), learning disabilities, autism, and intellectual disability. The data come from a national survey that collects health information from a representative sample of the population each year. The four indicators show how the prevalence of children's neurodevelopmental disorders has changed over time, and, when possible, how the prevalence differs between boys and girls.

National Health Interview Survey

The National Health Interview Survey (NHIS) provides nationally representative data on the prevalence of ADHD, learning disabilities, autism, and intellectual disability (mental retardation) in the United States each year. NHIS is a large-scale household interview survey of a representative sample of the civilian noninstitutionalized U.S. population, conducted by the National Center for Health Statistics (NCHS). The interviews are conducted in person at the participants' homes. From 1997–2005, interviews were conducted for approximately 12,000–14,000 children annually. From 2006–2008, interviews were conducted for approximately 9,000–10,000 children per year. In 2009 and 2010, interviews were conducted for approximately 11,000 children per year. The data are obtained by asking a parent or other knowledgeable household adult questions regarding the child's health status. NHIS asks "Has a doctor or health professional ever told you that <child's name> had Attention Deficit/Hyperactivity Disorder (ADHD) or Attention Deficit Disorder (ADD)? Autism? Mental Retardation?" Another question on the NHIS survey asks "Has a representative from a school or a health professional ever told you that <child's name> had a learning disability?"

Data Presented in the Indicators

The following indicators display the prevalence of ADHD, learning disabilities, autism, and intellectual disability among U.S. children, for the years 1997–2010. Diagnosing neurodevelopmental disorders in young children can be difficult: many affected children may not receive a diagnosis until they enter preschool or kindergarten. For this reason, the indicators here show children ages 5 to 17 years. Where data are sufficiently reliable, the indicators provide separate prevalence estimates for boys and girls.

Although the NHIS provides national-level data on the prevalence of neurodevelopmental disorders over a span of many years, NHIS data could underestimate the prevalence of neurodevelopmental disorders. Reasons for underestimation may include late identification of affected children and the exclusion of institutionalized children from the NHIS survey population. A diagnosis of a neurodevelopmental disorder depends not only on the presence of particular symptoms and behaviors in a child, but on concerns being raised by a parent or teacher about the child's behavior, as well as the child's access to a doctor and the accuracy of the doctor's diagnosis. Further, the NHIS relies on parents reporting that their child has been diagnosed with a neurodevelopmental disorder, and the accuracy of parental responses could be affected by cultural and other factors.

Long-term trends in these conditions are difficult to detect with certainty due to a lack of data to track prevalence over many years, as well as changes in awareness and diagnostic criteria, which could explain at least part of the observed increasing trends.[184-186] The NHIS questions also do not assess whether a child currently has a disorder; instead, they provide data on whether a child has ever been diagnosed with a disorder, regardless of their current status.

Survey responses for learning disabilities may be more uncertain than for the other three disorders presented. Whereas survey respondents are asked whether the child has been diagnosed with ADHD, autism, or intellectual disability (mental retardation) by a health professional, for learning disabilities an affirmative response may also include a school representative. It is possible that some parents may respond "yes" to the question regarding learning disabilities based on informal comments made at school, rather than a formal evaluation to determine whether the child has any specific learning disability; similarly, they may give a "yes" answer for children with diagnosed disorders that are not learning disabilities. For example, parents of children with intellectual disability might also respond "yes" to the learning disability question, thinking that any learning problems may apply, even though intellectual disability and learning disabilities are distinct conditions.[2]

Because autism is the only autism spectrum disorder (ASD) referred to in the survey, it is not clear how parents of children with other ASDs, i.e., Asperger's syndrome and PDD-NOS, may have responded. The estimates shown by Indicator H8 could represent underestimates of ASD prevalence if parents of children with Asperger's syndrome and PDD-NOS did not answer yes to the NHIS questions about autism.

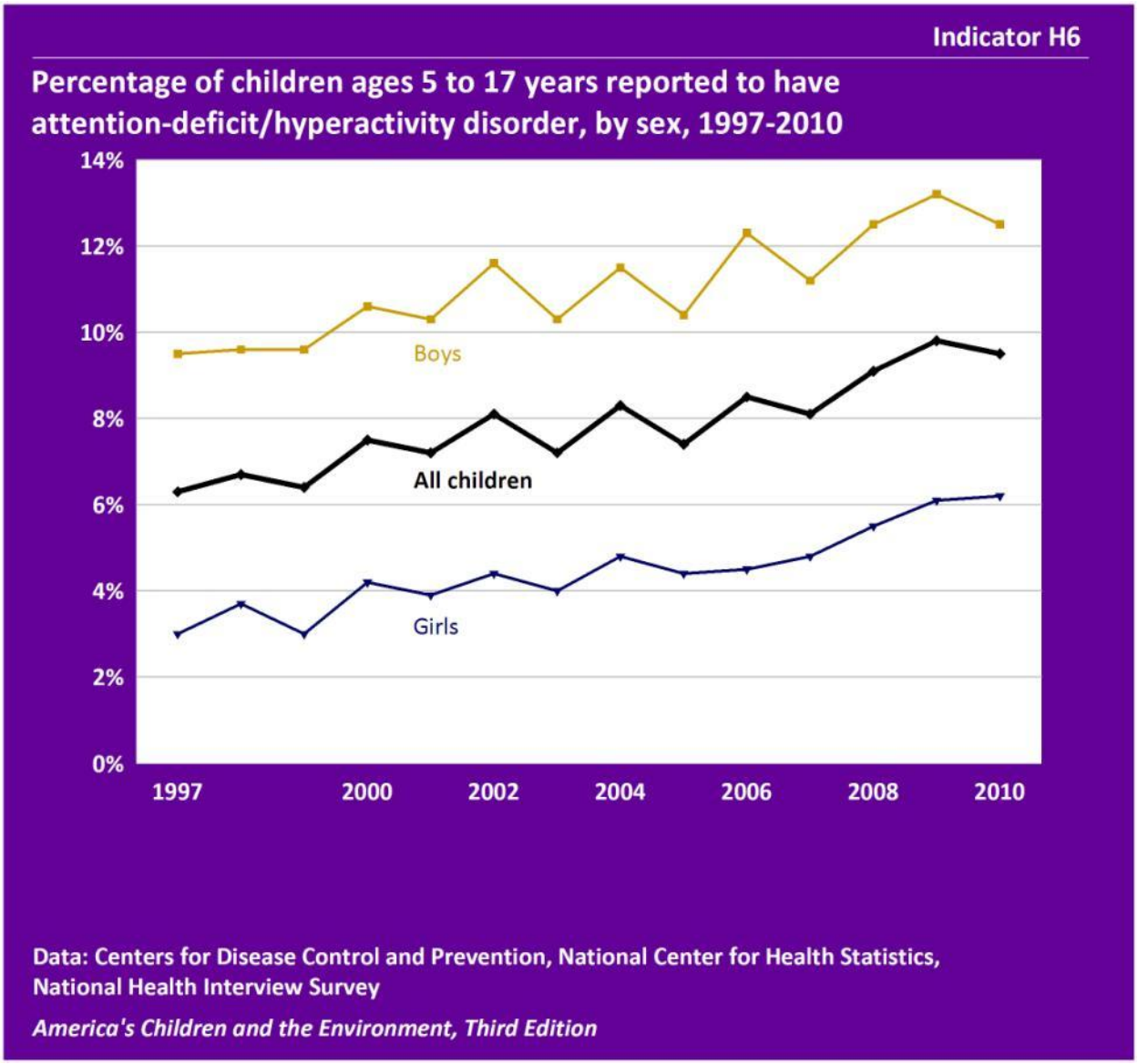
In addition to the data shown in the indicator graphs, supplemental tables provide information regarding the prevalence of neurodevelopmental disorders for different age groups and prevalence by race/ethnicity, sex, and family income. These comparisons use the most current four years of data available. The data from four years are combined to increase the statistical reliability of the estimates for each race/ethnicity, sex, and family income group. The tables include prevalence estimates for the following race/ethnicity groups: White non-Hispanic, Black non-Hispanic, Asian non-Hispanic, Hispanic, and "All Other Races." The "All Others Races" category includes all other races not specified, together with those individuals who report more than one race. The limits of the sample design and sample size often prevent statistically reliable estimates for smaller race/ethnicity groups. The data are also tabulated for three income groups: all incomes, income below the poverty level, and greater than or equal to the poverty level.

Please see the Introduction to the Health section for discussion of statistical significance testing applied to these indicators.

Other Estimates of ADHD and Autism Prevalence

In addition to NHIS, other NCHS studies provide data on prevalence of ADHD and ASDs among children. The National Survey of Children's Health (NSCH), conducted in 2003 by NCHS, found that 7.8% of children ages 4 to 17 years had ever been diagnosed with ADHD. The same survey, when conducted again in 2007, found that 9.5% of children ages 4 to 17 years had ever been diagnosed with ADHD.[7] Both estimates are somewhat higher than the ADHD prevalence estimates from the NHIS for those years. The 2007 NSCH also estimates that 7.2% of children ages 4 to 17 years currently have ADHD. The 2007 NSCH also provides information at the state level: North Carolina had the highest rate, with 15.6% of children ages 4 to 17 years having ever been diagnosed with ADHD; the rate was lowest in Nevada, at 5.6%.[7]

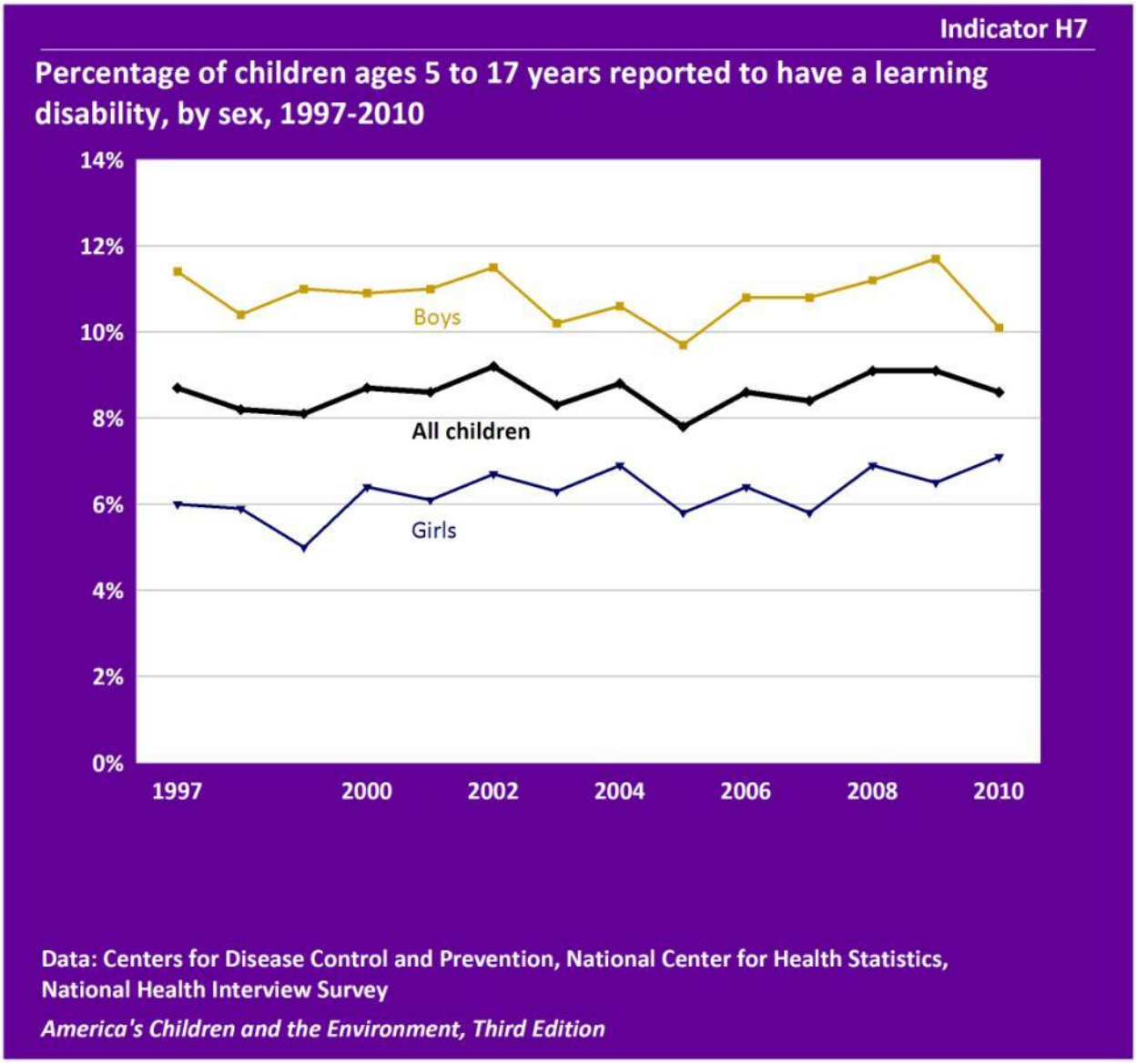
In 2002 and 2006, the Centers for Disease Control and Prevention performed thorough data gathering in selected areas to examine the prevalence of ASDs in eight-year-old children. The ASD prevalence estimate for 2002 was 0.66%, or 1 in 152 eight-year-old children, and the estimate for 2006 was 0.9%, or 1 in 110 eight-year-old children.[8,187] The 2007 NSCH also provides an estimate of 1.1% of children ages 3 to 17 years reported to have ASDs, or about 1 in 90.[188]



Data characterization

- Data for this indicator are obtained from an ongoing annual survey conducted by the National Center for Health Statistics.
- Survey data are representative of the U.S. civilian noninstitutionalized population.
- A parent or other knowledgeable adult in each sampled household is asked questions regarding the child's health status, including if they have ever been told the child has Attention Deficit/Hyperactivity Disorder (ADHD).

- From 1997 to 2010, the proportion of children ages 5 to 17 years reported to have ever been diagnosed with attention-deficit/hyperactivity disorder (ADHD) increased from 6.3% to 9.5%.
  - The increasing trend was statistically significant for children overall, and for both boys and girls considered separately.
- For the years 2007–2010, the percentage of boys reported to have ADHD (12.4%) was higher than the rate for girls (5.7%). This difference was statistically significant. (See Table H6a.)
- In 2007–2010, 11.6% of children of "All Other Races," 10.7% of White non-Hispanic children, 10.2% of Black non-Hispanic children, 4.8% of Hispanic children, and 1.7% of Asian non-Hispanic children were reported to have ADHD. (See Table H6b.)
  - These differences were statistically significant, after accounting for the influence of other demographic differences (i.e., differences in age, sex, and family income), with two exceptions: there was no statistically significant difference between children of "All Other Races" and White non-Hispanic children, or between children of "All Other Races" and Black non-Hispanic children.
- In 2007–2010, 11.3% of children from families living below the poverty level were reported to have ADHD compared with 8.6% of children from families living at or above the poverty level. This difference was statistically significant. (See Table H6b.)

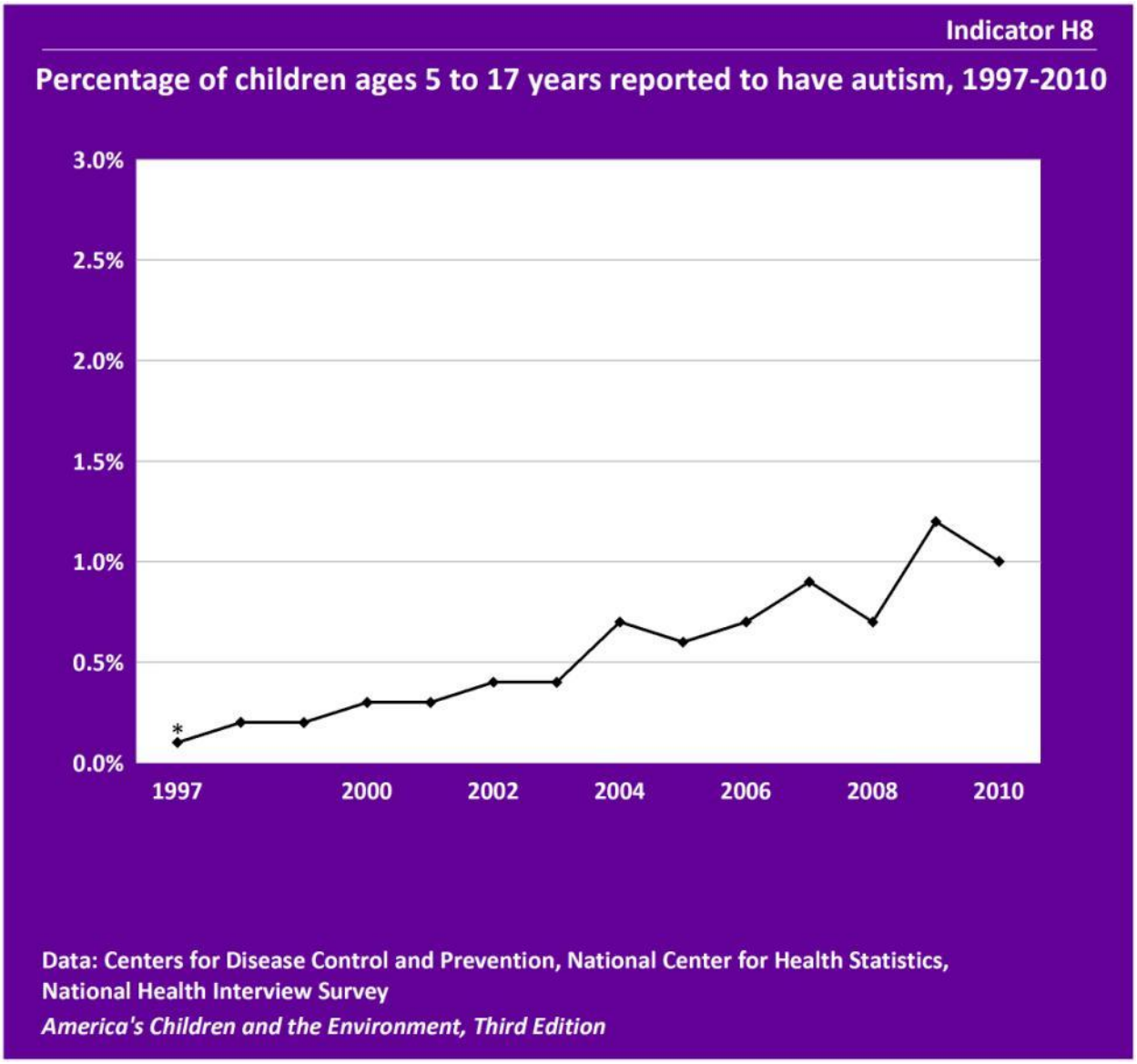


Data characterization

- Data for this indicator are obtained from an ongoing annual survey conducted by the National Center for Health Statistics.
- Survey data are representative of the U.S. civilian noninstitutionalized population.
- A parent or other knowledgeable adult in each sampled household is asked questions regarding the child's health status, including if they have ever been told the child has a learning disability.

- In 2010, 8.6% of children ages 5 to 17 years had ever been diagnosed with a learning disability. There was little change in this percentage between 1997 and 2010.
- For the years 2007–2010, the percentage of boys reported to have a learning disability (10.9%) was higher than for girls (6.6%). This difference was statistically significant. (See Table H7a.)

- The reported prevalence of learning disability varies by race and ethnicity. The highest percentages of learning disability are reported for children of "All Other Races" (11.2%), Black non-Hispanic children (10.2%), and White non-Hispanic children (9.3%). By comparison, 7.2% of Hispanic children are reported to have a learning disability, and Asian non-Hispanic children have the lowest prevalence of learning disability, at 2.7%. (See Table H7b.)
  - The prevalence of learning disability reported for Hispanic children and for Asian non-Hispanic children were lower than for the remaining race/ethnicity groups, and these differences were statistically significant. The difference in prevalence between Hispanic and Asian non-Hispanic children was also statistically significant.
- For the years 2007–2010, the percentage of children reported to have a learning disability was higher for children living below the poverty level (12.6%) compared with those living at or above the poverty level (7.9%), a statistically significant difference. (See Table H7b.)



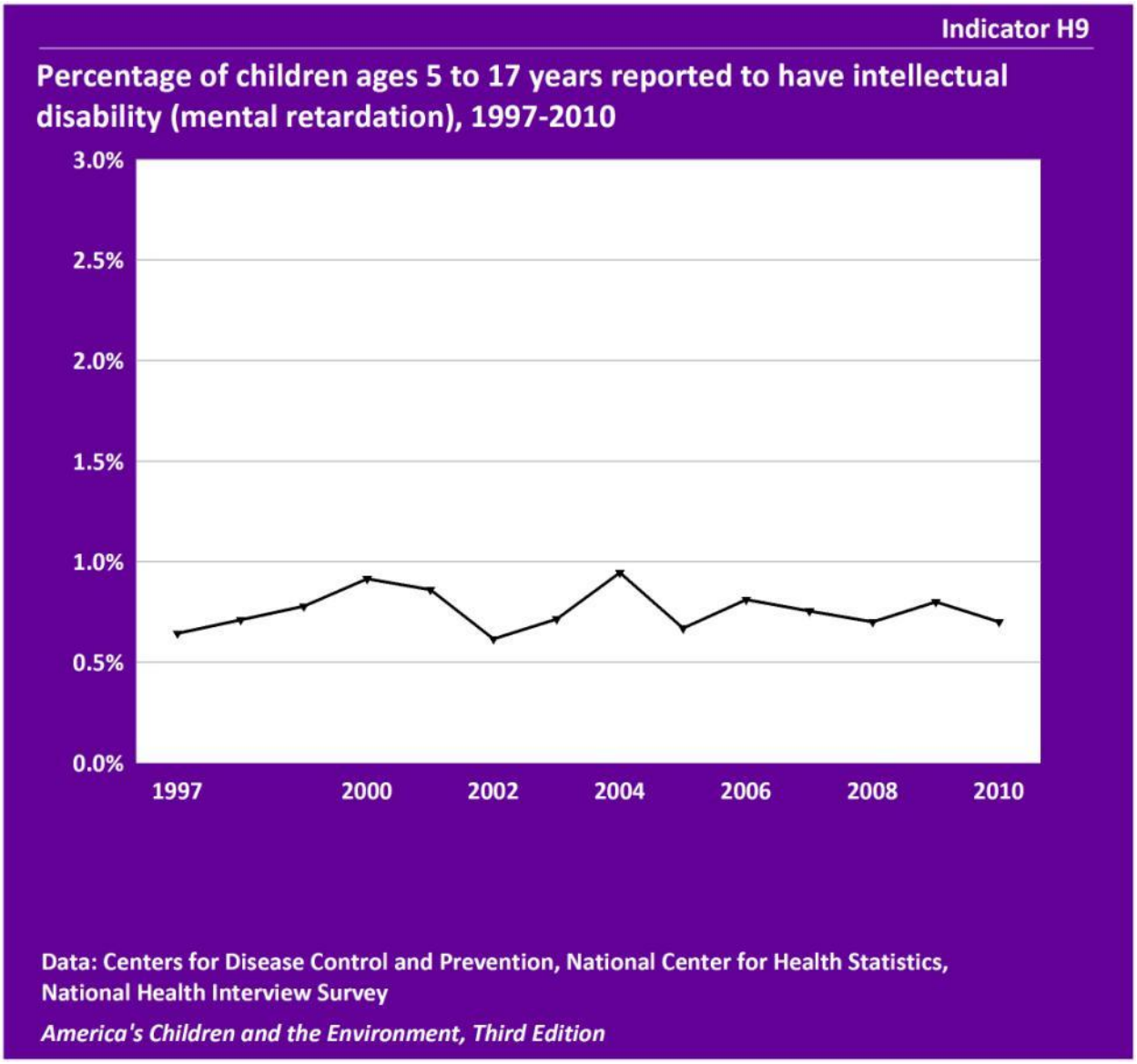
* The estimate should be interpreted with caution because the standard error of the estimate is relatively large: the relative standard error, RSE, is at least 30% but is less than 40% (RSE = standard error divided by the estimate).

Data characterization

- Data for this indicator are obtained from an ongoing annual survey conducted by the National Center for Health Statistics.
- Survey data are representative of the U.S. civilian noninstitutionalized population.
- A parent or other knowledgeable adult in each sampled household is asked questions regarding the child's health status, including if they have ever been told the child has autism.

- The percentage of children ages 5 to 17 years reported to have ever been diagnosed with autism rose from 0.1% in 1997 to 1.0% in 2010.This increasing trend was statistically significant.

- For the years 2007–2010, the rate of reported autism was more than three times higher in boys than in girls, 1.5% and 0.4%, respectively. This difference was statistically significant. (See Table H8a.)
- The reported prevalence of autism varies by race/ethnicity. The highest prevalence of autism is for children of "All Other Races" (1.7%) and White non-Hispanic children (1.1%). Autism prevalence was lower among Asian non-Hispanic children (0.8%), Black non-Hispanic children (0.7%), and Hispanic children (0.6%). (See Table H8b.)
  - The prevalence of autism for both White non-Hispanic children and children of "All Other Races" was statistically significantly different from the prevalence for both Black non-Hispanic children and Hispanic children.
- For the years 2007–2010, the prevalence of autism was similar for children living below the poverty level and those living at or above the poverty level. (See Table H8b.)



Data characterization

- Data for this indicator are obtained from an ongoing annual survey conducted by the National Center for Health Statistics.
- Survey data are representative of the U.S. civilian noninstitutionalized population.
- A parent or other knowledgeable adult in each sampled household is asked questions regarding the child's health status, including if they have ever been told the child has mental retardation.

- In 2010, 0.7% of children ages 5 to 17 years were reported to have ever been diagnosed with intellectual disability (mental retardation). There was little change in this percentage between 1997 and 2010.

- In 2007–2010, the percentage of boys reported to have intellectual disability (0.9%) was higher than for girls (0.6%). This difference was statistically significant. (See Table H9a.)
- In 2007–2010, there was little difference by race/ethnicity in the reported prevalence of intellectual disability. (See Table H9b.)
- In 2007–2010, 1.2% of children from families with incomes below the poverty level were reported to have intellectual disability, compared with 0.7% of children from families at or above the poverty level, a statistically significant difference. (See Table H9b.)

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Obesity

Obesity is the term used to indicate the high range of weight for an individual of given height that is associated with adverse health effects.[1] Definitions of overweight and obesity for adults are based on set cutoff points directly related to an individual's body mass index (BMI, weight in kilograms divided by the square of height in meters). Essential to this definition is that a high degree of body weight be associated with a large amount of body fat. The BMI is correlated to body fat, but BMI varies with age and sex in children more than it does in adults. Thus the designation of a child or adolescent (ages 2 to 19 years) as either overweight or obese is based on comparing his or her BMI to a sex- and age-specific reference population (the CDC growth charts). Children and adolescents between the 85th and 94th percentiles of BMI-for-age are considered overweight; those greater than or equal to the 95th percentile are considered obese. The percentiles used to identify children as overweight or obese are fixed, and based on data collected from 1963–1980 (or, for children ages 2 to 6 years, data from 1963–1994).[1-3]

The prevalence of excessive body weight in the United States population has been increasing for several decades, though it has stabilized over the last several years.[4-7] BMI is the most common screening measure used to determine whether an individual may be overweight or obese. The BMI does not measure body fat directly, but is used as a surrogate measure since it correlates with direct measures of body fat, especially at high BMI levels, and is inexpensive and easy to obtain in a clinical setting. The significance of a child being overweight is complicated by the BMI's inability to distinguish between differences in mass due to muscle or due to the unhealthy accumulation of fatty tissue. A recent study found that less than half of "overweight" children had excess body fat, and that there are differences among race/ethnicity groups in the amount of body fat for a given BMI in children.[8] Among children with an elevated BMI, some may have excess body fat, and others may be incorrectly identified as overweight because they have a higher amount of mass attributed to nonfatty tissue. Despite the limitations imposed by measuring the BMI, a rise in the prevalence of overweight children is cause for concern, since overweight children are more likely to become overweight or obese adults.[9-11]

Obesity has rapidly become a serious public health concern in the United States, and is associated with several adverse health effects in childhood and later in life, including cardiovascular disease risk factors (which includes hypertension and altered lipid levels),[12-22] cancer,[15,23,24] psychological stress,[25-28] asthma,[29-31] and diabetes.[32-37] Some studies have found a relationship between obesity and early onset of puberty and early menarche in girls,[38-40] although other research has found differences in the timing of puberty even after controlling for BMI in the population.[41] As such, the extent to which the obesity epidemic may contribute to early puberty is unclear.

An emerging body of research suggests there may be common biological mechanisms underlying a cluster of adverse health effects (obesity, hypertension, altered lipid levels, and other metabolic abnormalities) referred to as metabolic syndrome. While the clinical utility of a diagnosis of metabolic syndrome is debated in the medical literature,[42-44] the term describes an area of active research, and prospective data demonstrate the relevance of metabolic syndrome in obese children for both type 2 diabetes[45] and cardiovascular disease.[46] Metabolic syndrome has been identified in obese children and adolescents, and studies suggest a developmental origin of the condition.[47-49] The consideration of obesity and metabolic effects as a group is supported by findings in laboratory animals, where early-life exposure to certain organophosphate pesticides can disrupt adult lipid metabolism, induce weight gain, and cause other metabolic responses that mimic those seen in diabetes and obesity.[50-52] Given these relationships, obesity and other health conditions related to metabolism are discussed below.

Obesity is due primarily to an imbalance between caloric intake and activity. Increased caloric intake and reduced physical activity are likely the major drivers of obesity in children. Researchers are also investigating whether exposures to certain environmental chemical exposures may play a contributing role in childhood obesity.[53,54] These chemicals, which are referred to as obesogens, are thought to be capable of disrupting the human body's regulation of metabolism and the accumulation of fatty tissue.[55] Studies have also reported associations between exposure to certain chemicals and diabetes in adults. Diabetes (Type 2) results from the body's inability to regulate blood sugar levels with insulin in response to dietary intake, and is positively associated with the increasing rates of obesity seen in the U.S. population.[56] Excess body weight is a risk factor for Type 2 diabetes. In the past, Type 2 diabetes has been diagnosed almost exclusively in adult populations, but it is now being diagnosed in youth—although with low prevalence (0.25%).[56-59] However, the clinical designation of prediabetes (elevated blood glucose levels that do not meet the diagnostic criteria for diabetes) is prevalent in obese youth.[35]

While the possible contribution of chemical exposures to obesity is not clear, a number of animal and cellular studies provide some evidence that environmental chemical exposures may contribute to obesity and diabetes. Studies finding associations between chemical exposures and obesity in children are limited. A recent study reported that prenatal exposure to high levels of hexachlorobenzene was associated with increased BMI and weight in children at 6.5 years.[60] Another recent study in Belgium, at relatively high exposure levels within the general population, reported an association between prenatal exposure to DDE (the primary metabolite of the pesticide DDT) and BMI, as well as an association between exposure to polychlorinated biphenyls (PCBs) and increased BMI during early childhood.[61] In adults, associations have been reported between diabetes and both PCBs and dioxins at levels of exposure seen in the U.S. population.[62,63] A study of adult occupational exposures to organochlorine and organophosphate pesticides reported an increased risk of diabetes in exposed workers.[64] However, other studies have reported no association between these exposures and markers of obesity or diabetes.[65-68] Several animal and cellular studies suggest that endocrine-disrupting chemicals (including bisphenol A, diethylstilbestrol, and tributyltin) may contribute to increased weight and diabetes.[69-73] After reviewing these findings, scientists at a National Toxicology Program-sponsored workshop concluded that existing research provides evidence of plausibility (varying from "suggestive" to "strong" evidence) that several environmental chemicals could contribute to obesity and/or diabetes.[74] For example, scientists concluded that the available data support the biological plausibility that exposure to a number of classes of pesticides may affect risk factors for obesity and diabetes. The National Institutes of Health Strategic Plan for Obesity Research and the White House Task Force on Childhood Obesity Report to the President also acknowledge a potential relationship between environmental exposures and obesity and cite the need for further research.[75,76]

Research has also considered a role for air pollution in childhood obesity and diabetes. In one recent study, adult mice fed a high-fat diet and exposed to concentrated particulate air pollution ($PM_{2.5}$) experienced an increase in blood glucose levels and insulin resistance, which are precursors of diabetes.[77] Other studies in animals and children have reported that obesity may result in greater susceptibility to the adverse effects of airborne pollutants such as $PM_{2.5}$ and ozone, including airway inflammation, cardiovascular effects, and increased deposition of particles in the lungs.[30,78,79] Air pollution may contribute to childhood obesity by limiting the number of days when air quality is appropriate for outdoor recreational activity, particularly in children with pre-existing respiratory conditions such as wheeze and asthma.[80] Animal studies further suggest that diet-induced obesity may increase susceptibility to the effects of environmental toxicants such as PCBs, dioxins, and acrylamide.[81-83]

Other environmental factors are thought to contribute to the increasing rates of overweight and obesity seen in the U.S. population. The term "built environment" is used to describe the physical elements of the environment for a population.[84,85] Multiple reviews of the literature have concluded that several properties of the built environment, including the extent of urban sprawl, housing density, access to food outlets, and access to recreational facilities, may be associated with overweight and obesity and/or levels of physical activity in children.[84-90] The relationship between characteristics of the built environment and obesity is likely more significant in children than adults, because children are less able to leave their local environment without the help of an adult.[91,92] Built environments that promote exercise through the inclusion of nearby recreational areas and walkable communities, and those that provide healthy eating options through reducing the number of fast food restaurants while providing access to fresh produce, are thought to reduce the frequency of obesity in children.[84,85,93] "Green" environments that contain a greater number of natural environments and features such as parks, trees, and nature trails, may contribute to increased levels of physical activity in children that can reduce rates of obesity.[94]

Socioeconomically disadvantaged populations are more likely to be located in built environments with characteristics that promote lifestyles that increase rates of obesity in children.[95-97] However, a child living in a suburban community with a higher socioeconomic status may spend greater amounts of time commuting in a car rather than walking, which may also contribute to a sedentary lifestyle that promotes obesity.[98,99] Factors contributing to the prevalence of obesity may differ among environments. Previous research had suggested that differences in obesity rates in rural or urban environments were small.[100,101] However, other recent studies have identified a higher prevalence of obesity in rural compared to urban environments.[87,102] The complex interplay of behavioral, environmental, and physiological factors and the disparities in pediatric obesity observed in the population add to the difficulty in identifying effective interventions.

The following indicators present the best nationally representative data on obesity in the U.S. child population. The first indicator shows the prevalence of obesity among children ages 2 to 17 years from 1976–2008. The second indicator presents the current prevalence of obesity by race/ethnicity and family income, using data from 2005–2008. Together these indicators highlight basic trends and current status in prevalence of childhood obesity in the United States.

Indicator H10: Percentage of children ages 2 to 17 years who were obese, 1976–2008

Indicator H11: Percentage of children ages 2 to 17 years who were obese, by race/ethnicity and family income, 2005–2008

About the Indicators: Indicators H10 and H11 present the prevalence of obesity in U.S. children ages 2 to 17 years. The data are from a national survey that measures weight and height in a representative sample of the U.S. population every two years. Indicator H10 shows the trend in obesity prevalence from 1976–2008. Indicator H11 presents comparisons of current obesity rates in children of different race/ethnicities and income levels, using data for 2005–2008.

NHANES

The National Health and Nutrition Examination Survey (NHANES) provides data on childhood obesity in the United States. NHANES is a nationally representative survey of the health and nutritional status of the civilian noninstitutionalized U.S. population, conducted by the National Center for Health Statistics. Interviews and physical examinations are conducted with approximately 10,000 people in each two-year year survey cycle. Height and weight are measured for survey participants of all ages.

Obesity and BMI

Determination of obesity in children is based on the calculation of body mass index (BMI), which is correlated with body fat.[103] First, the BMI is calculated by dividing an individual's weight in kilograms by the square of his or her height in meters. For children and teenagers in the United States, the BMI number is then compared with an age- and sex-specific reference population based on the 2000 CDC growth charts. These charts are based on national data collected from 1963–1994 for children 2 through 6 years of age and from 1963–1980 for children ages 7 years and older.[2] These growth charts apply to all racial and ethnic groups, and were obtained from nationally representative surveys. Children and teenagers with BMIs at or above the 95th percentile on the growth charts are classified as obese.[1,3]

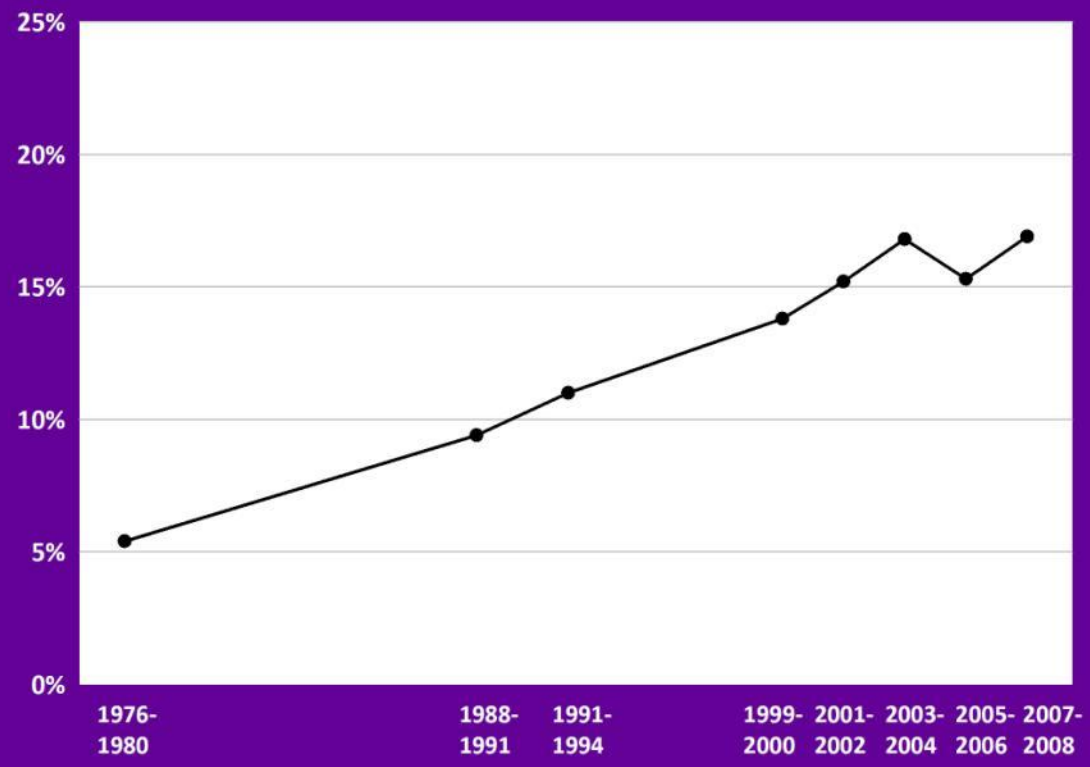
Data Presented in the Indicators

Indicator H10 presents the percentage of children ages 2 to 17 years who were obese from NHANES surveys conducted from 1976 through 2008. Indicator H11 presents the current prevalence of childhood obesity by race/ethnicity and family income using the 2005–2006 and 2007–2008 surveys combined. The data from two NHANES cycles are combined to increase the statistical reliability of the estimates for each race/ethnicity and income group, and to reduce any possible influence of geographic variability that may occur in two-year NHANES data. Four race/ethnicity groups are presented in Indicator H11: White non-Hispanic, Black non-Hispanic, Mexican-American, and "All Other Races/Ethnicities." The "All Other Races/Ethnicities" category includes all other races and ethnicities not specified, together with those individuals who report more than one race. The limits of the sample design and sample size often prevent statistically reliable estimates for smaller race/ethnicity groups. The data are also tabulated across three income categories: all incomes, below the poverty level, and greater than or equal to the poverty level.

Please see the Introduction to the Health section for discussion of statistical significance testing applied to these indicators.

Indicator H10

Percentage of children ages 2 to 17 years who were obese, 1976-2008



Data: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health and Nutrition Examination Survey

America's Children and the Environment, Third Edition

Data characterization

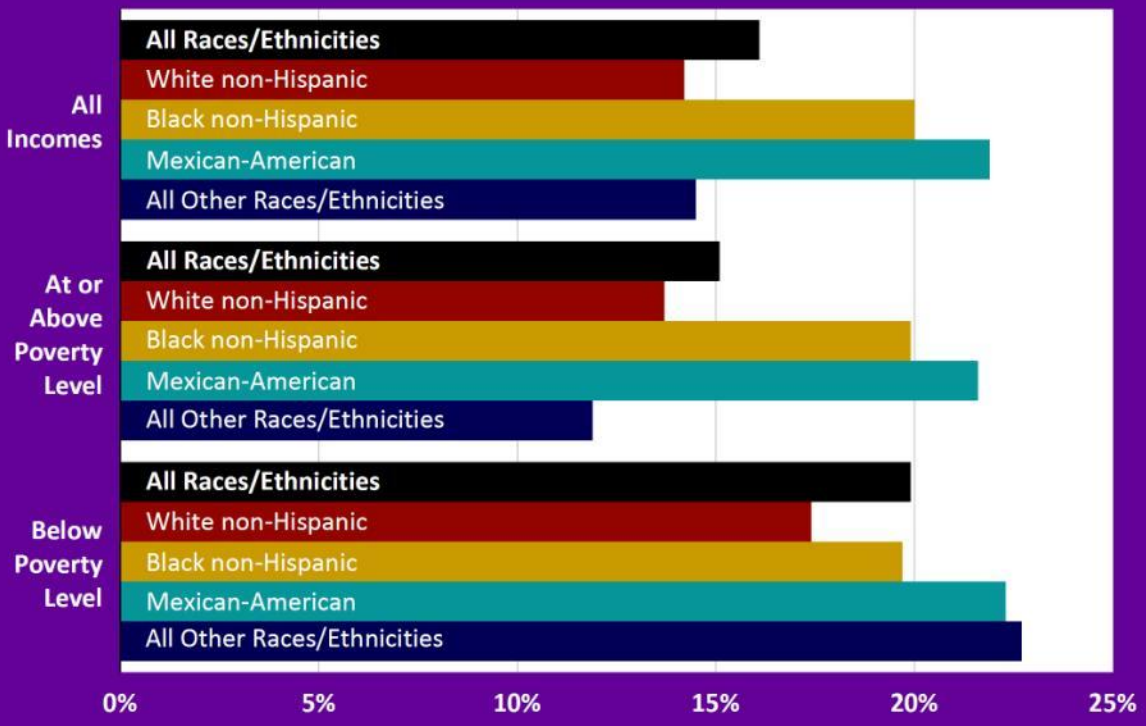
- Data for this indicator are obtained from an ongoing continuous survey conducted by the National Center for Health Statistics.
- Survey data are representative of the U.S. civilian noninstitutionalized population.
- Height and weight are measured in individual survey participants.

- Between 1976–1980 and 2007–2008, the percentage of children identified as obese showed an increasing trend. In 1976–1980, 5% of children ages 2 to 17 years were obese. This percentage reached a high of 17% in 2007–2008. Between 1999–2000 and 2007–2008, the percentage of children identified as obese remained between 15% and 17%.
  - From 1976–2008, the increasing trend in prevalence of obese children was statistically significant for children overall, and for children of each race/ethnicity (See Table H10)

and age group (Table H10a). From 1999–2008, the trends were not statistically significant for each of these groups.

Indicator H11

Percentage of children ages 2 to 17 years who were obese, by race/ethnicity and family income, 2005-2008



Data: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health and Nutrition Examination Survey

America's Children and the Environment, Third Edition

Data characterization

- Data for this indicator are obtained from an ongoing continuous survey conducted by the National Center for Health Statistics.
- Survey data are representative of the U.S. civilian noninstitutionalized population.
- Height and weight are measured in individual survey participants.

- In 2005–2008, 16% of children ages 2 to 17 years were classified as obese.
- In 2005–2008, a higher percentage of Mexican-American and Black non-Hispanic children were obese at 22% and 20%, respectively, compared with14% of White non-Hispanic children and 14% of children of "All Other Races/Ethnicities."

  - The greater prevalence of obesity among Mexican-American and Black non-Hispanic children, compared with the lower prevalence among White non-Hispanic children and children of "All Other Races/Ethnicities," was statistically significant.
- Among children overall, the prevalence of obesity was greater in children with family incomes below poverty level than in those above poverty level. However, when accounting for differences by race/ethnicity as well as poverty status, children of "All Other Races/Ethnicities" were the only group to have a statistically significant association between low family income and higher prevalence of obesity.

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Adverse Birth Outcomes

The period of gestation is a crucial determinant of an infant's health and survival for years to come. Two measures that may be used to understand the quality of an infant's gestation are 1) length of gestation (pregnancy length) and 2) birth weight. Normal term pregnancies last between 37 and 41 completed weeks, allowing for more complete development of an infant's organs and systems.[1] Preterm birth is defined as a live birth before 37 completed weeks of gestation.[1] Birth weight is determined by two factors: length of gestation and fetal growth (the rate at which an infant develops and increases in size). Low birth weight infants are defined as weighing less than 2,500 grams (about 5 pounds, 8 ounces).[2] Infants may be born with a low birth weight because they were born early, because their growth while in utero has been restricted, or both. Because they have had sufficient time to develop, infants born at term with low birth weight are usually considered growth restricted. Because birth weight alone does not always indicate whether an infant's fetal growth has been restricted, other measurements such as birth length, head circumference, and abdominal circumference are also used.

Other adverse birth outcomes that are not discussed here include post-term birth, high birth weight, neonatal mortality, and birth defects, a specific group of adverse birth outcomes that include structural and functional abnormalities.

Preterm and low birth weight infants are at greater risk for mortality and a variety of health and developmental problems. As a result, the birth of a preterm or low birth weight infant can have significant emotional and economic effects on the infant's family.[3] Conditions related to preterm birth and low birth weight are the second leading cause of infant death in the United States (after birth defects).[4] The infant mortality rate for low birth weight infants is about 25 times that of the infant mortality rate for normal weight babies. Likewise, the infant mortality rate for late preterm babies (34–36 weeks of gestation) is about three times the infant mortality rate for term babies, and the infant mortality rate for very preterm babies (less than 32 weeks of gestation) is 75 times that of term babies.[4] Preterm infants may experience complications such as acute respiratory, gastrointestinal, immunologic, and central nervous system problems. Longer-term effects of preterm birth, including motor, cognitive, visual, hearing, behavioral, social-emotional, health, and growth problems, may not become apparent for years and may persist throughout a child's life into adulthood. It is important to recognize that not all infants born before 37 completed weeks have the same risk of adverse health outcomes. As gestational age decreases, the risk of morbidity and mortality increases greatly. Also, recent research suggests that even early term births, those at 37 or 38 weeks, are at increased risk of respiratory and other adverse neonatal outcomes.[5-7]

Because many of the effects of low birth weight are due to being born immature and unprepared for life outside the womb, morbidities associated with low birth weight often overlap with those of preterm birth. Low birth weight infants are more likely to have underdeveloped lungs and breathing problems; heart problems (which can lead to heart failure); immature and improperly functioning livers; too many or too few red blood cells (polycythemia or anemia); inadequate body fat, leading to trouble maintaining a normal body temperature; feeding problems; and increased risk of infection.[2] Furthermore, the process of growth restriction may exert its own negative effects aside from often producing low birth weight infants. Data suggest that fetuses with a declining growth rate may make adaptations, such as preserving brain growth, in order to survive adverse intrauterine conditions. Such adaptations can have physiological costs, and may have effects on fetal brain development, cardiac and renal function, and adult health.[8] The theory of fetal origins of adult disease postulates that certain types of chemical,

nutritional, or stress-related exposures in utero can alter the programming of fetal cells in ways that are not apparent at birth, but are predictive of disease risk later in life. Birth weight and measures of growth restriction are used as proxies for these changes and have been associated with diseases in adulthood, including cardiovascular disease, obesity, metabolic disorders, and cancer.[9]

For many years, the rates of both preterm birth and low birth weight have been increasing;[10] however, starting in 2006 this pattern seems to be partially reversing as the rate of preterm birth is now declining. A number of factors may contribute to increasing rates of preterm birth and low birth weight, including increases in maternal age, rates of multiple births (e.g., twins, triplets), use of early Cesarean sections and labor inductions, changes in neonatal technology, and use of assisted reproductive technologies (e.g., in vitro fertilization).[3] Multiple births run a higher risk of preterm birth and low birth weight, and the rates of multiple births have increased in recent decades. The rate of twin births increased 70% from 1980–2004, but has been essentially stable since that time. The rate of triplet and higher-order births increased 400% from 1980 to 1998, but since that time has been trending downward.[11] Advances in medical technology that allow for resuscitation of infants born at increasingly early gestational ages may also contribute to the increase in percentage of births that are preterm, since many of those infants would not have survived previously and thus would have be characterized as fetal deaths. Other factors linked to preterm birth and low birth weight include birth defects; chronic maternal health problems (e.g., high blood pressure); maternal use of tobacco, alcohol, and illicit drugs; maternal and fetal infections; placental problems; inadequate maternal weight gain; and socioeconomic factors (e.g., low income and poor education).[12-16]

Rates of low birth weight and preterm birth can vary greatly by maternal race/ethnicity. Black women have consistently had higher rates of preterm and low birth weight babies.[17] While it has been suggested that race is a proxy for differences in socioeconomic status (SES), most studies that have controlled for differences in SES continue to find persistent birth outcomes differences between Black and White women.[17-20] Similarly, studies that have adjusted for other risk factors, such as risky behavior during pregnancy and use of prenatal care, have found these persistent Black-White differences in birth outcomes as well.[4,21,22]

While maternal characteristics and obstetric practices play an important role in preterm birth and low birth weight, other factors—including environmental contaminants—may also contribute to adverse birth outcomes.[23] A growing number of studies have examined the possible role that exposure to environmental contaminants may play in the causation of preterm birth and low birth weight. The evidence is particularly strong for environmental tobacco smoke (ETS) and lead. The Surgeon General has determined that exposure of pregnant women to ETS causes a small reduction in mean birth weight, and that the evidence is suggestive (but not sufficient to infer causation) of a relationship between maternal exposure to environmental tobacco smoke during pregnancy and preterm delivery.[24] The National Toxicology Program has concluded that maternal exposure to lead is known to cause reduced fetal growth, and that there is limited evidence of an association with preterm birth.[25]

In recent years, the potential effects of common air pollutants on adverse birth outcomes have received more attention. A number of large epidemiological studies (many with 10,000+ participants) from several countries have identified potential links between elevated levels of exposure to particulate matter (PM), sulfur dioxide (SO_2), nitrogen dioxide (NO_2), and carbon monoxide (CO) exposure and outcomes such as decreased fetal growth, low birth weight, and preterm birth.[26-40] Several of these studies have identified such links to adverse birth outcomes even in regions with relatively low ambient

air pollution levels.[27,29,33,36] In such epidemiological studies, researchers make an effort, when data are available, to adjust for other factors that may also lead to an increased risk of low birth weight or preterm birth, such as mother's age, smoking status, race, and income.[41] Articles reviewing the findings from these studies have generally concluded that these air pollutants likely have an adverse effect on birth outcomes, although methodological inconsistencies across studies have made definitive conclusions difficult.[42-45] In addition, studies have reported associations between elevated levels of exposure to airborne polycyclic aromatic hydrocarbons (PAHs), generated largely by fossil fuel combustion, and reduced birth weight and fetal growth restriction, especially when in combination with ETS exposure.[46-49] Other studies have reported associations between living in proximity to traffic during pregnancy and increased risk of preterm birth and low birth weight, although an extensive review study concluded that there is inadequate and insufficient evidence to infer a causal relationship.[50-54]

In addition to air pollutants, several other environmental chemicals have been studied for possible roles in contributing to adverse birth outcomes. A handful of studies with typical population-level exposure levels have reported associations between prenatal exposure to some phthalates and preterm birth, shorter gestational length, and low birth weight; however, one study reported phthalate exposure to be associated with longer gestational length and increased risk of delivery by Cesarean section.[55-59]

A limited number of studies suggest that prenatal exposure to another class of chemicals, polychlorinated biphenyls (PCBs), may lead to preterm birth and low birth weight or otherwise restrict fetal growth.[60-63] One study examining women from the Danish National Birth Cohort reported that elevated exposure to PCBs from fatty fish consumption was associated with lower birth weight. The study found that infants born to highly exposed women weighed, on average, about 5.5 ounces less than infants born to women with relatively low PCB exposure.[64] Another study looked at a historical cohort of women who were pregnant prior to the 1979 ban of PCBs, and did not observe any relation between levels of PCB exposure and low birth weight or shorter pregnancy length.[65] Some human health studies have reported associations between prenatal exposure to perfluorinated compounds (PFCs)—particularly perfluorooctane sulfonic acid (PFOS) and perfluorooctanoic acid (PFOA)—and a range of adverse birth outcomes, such as low birth weight, decreased head circumference, reduced birth length, and smaller abdominal circumference.[66-70] However, there are inconsistencies in the results of these studies, and two other studies did not find an association between prenatal PFC exposure and birth weight.[71,72] The participants in all of these studies had PFC blood serum levels comparable to levels in the general population. Studies of disinfection byproducts in drinking water as possible causes of adverse birth outcomes are also conflicting, with recent evidence indicating that there may be no effect on preterm birth.[73-75] Studies of arsenic in drinking water and birth outcomes have produced similarly mixed results.[76-78] For the following environmental contaminants, there is some evidence from animal studies and a limited number of studies in humans of possible associations with adverse birth outcomes, particularly reduced fetal growth: benzene,[79] herbicides,[80] bisphenol A (BPA),[81] dioxins and dioxin-like chemicals,[82] and manganese.[83]

This section presents two indicators of adverse birth outcomes: Indicator H12 presents the rate of preterm birth, and Indicator H13 presents the rate of term low birth weight. These two indicators were chosen because for each there is a wealth of quality data available.

Indicator H12: Percentage of babies born preterm, by race/ethnicity, 1993–2008

Indicator H13: Percentage of babies born at term with low birth weight, by race/ethnicity, 1993–2008

About the Indicator: Indicator H12 shows the percentage of babies born preterm, and Indicator H13 shows the percentage of babies who are born at term with low birth weight. Both graphs show separate lines for the different race/ethnicity groups. The data come from a national data system that collects data from birth certificates for virtually every baby born in the United States each year. Indicators H12 and H13 show the change in preterm and term low birth weight over time.

The National Vital Statistics System

The National Vital Statistics System (NVSS), operated by the National Center for Health Statistics (NCHS), provides national data on gestational ages and birth weights. The NVSS data are provided through contracts between the NCHS and vital registration systems operated in each state, which are legally responsible for the registration of vital events including births, deaths, marriages, divorces, and fetal deaths. The collection and publication of this information is mandated by federal law. Together NCHS and the states have developed standard forms and procedures to use for the data collection. The NVSS captures virtually all of the births occurring in the United States. The most current NVSS data available are for 2008.

Birth certificates provide information on characteristics of both the infant and his/her parents, including the weight of the infant and the length of gestation. Length of gestation is recorded in completed weeks, so for example a pregnancy of 36 weeks and 6 days would be recorded as 36 weeks, and would therefore be considered preterm.[3] Pregnancy duration is most often estimated from the date of a woman's last menstrual period. Many factors, including age, levels of physical activity, and body mass, can cause variation in menstrual cycle timing, making this method of estimating gestational length subject to some error.[3] NVSS data report pregnancy duration based on a clinical estimation, often determined using ultrasound, if information on last menstrual period is unavailable or is inconsistent with the reported birth weight. Because ultrasound measurements tend to give lower gestational age estimates than last menstrual period,[3] the slight increase in use of ultrasound data in recent years could contribute to any increase in the rate of preterm birth.

Data Presented in the Indicators

Indicator H12 displays the trend in the percentage of preterm births for all births (singletons, as well as multiples), with a separate line for each maternal race/ethnicity group and a single line for all maternal races and ethnicities combined for the years 1993–2008.

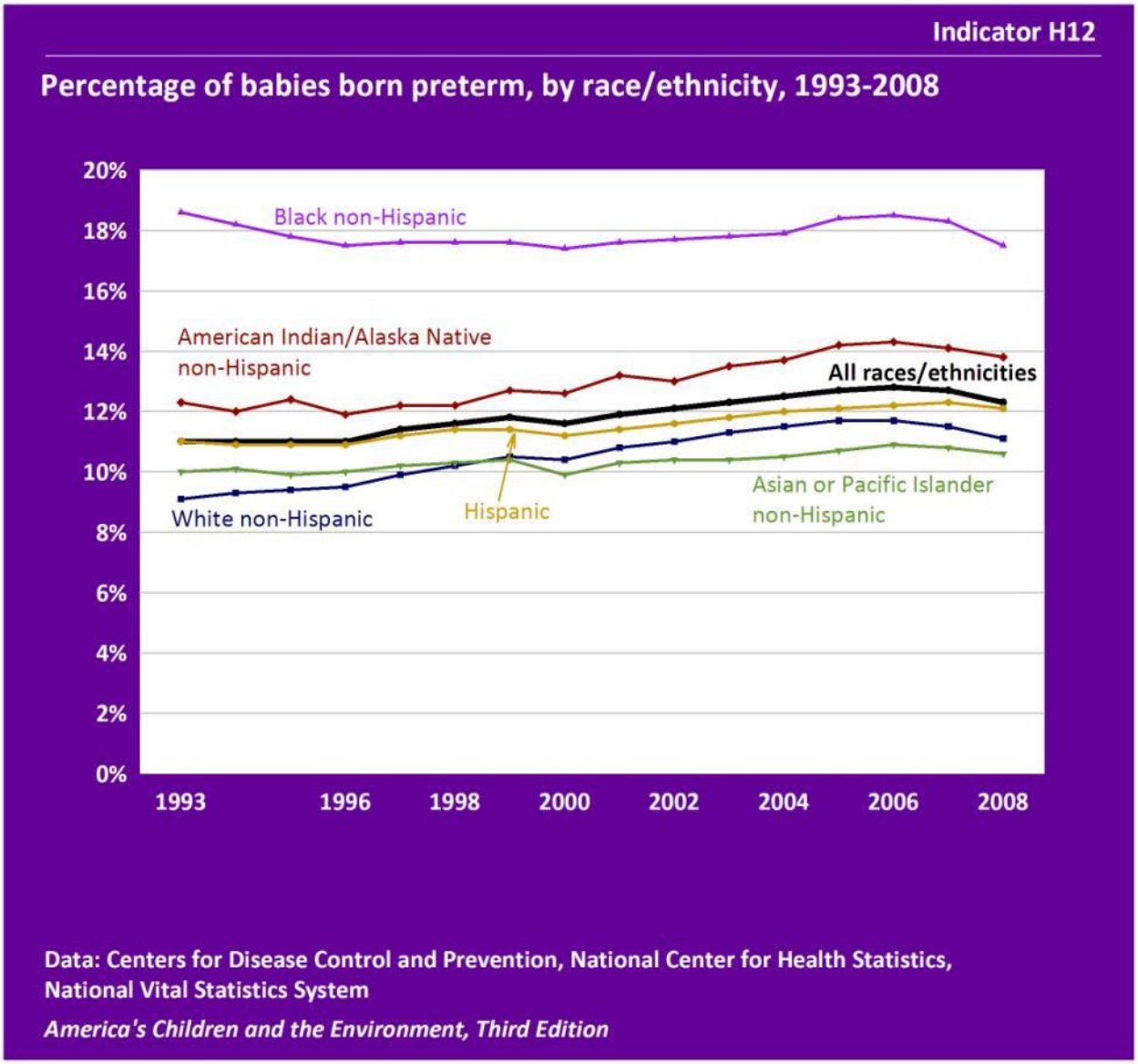
Indicator H13 displays the trend in the percentage of low birth weight births at term among all births (singletons, as well as multiples), with a separate line for each maternal race/ethnicity group and a single line for all maternal races and ethnicities combined for the years 1993–2008. Presentation of low birth weight data for only term births (babies with a gestational age of 37 completed weeks or more) is intended to identify trends in growth restriction separate from trends in gestational duration. This

indicator does not include all infants with low birth weight, nor does it include all infants who are growth-restricted; therefore, it is designed as a surveillance tool and not as a way to identify a group of infants that are particularly at risk for adverse health effects.

Five maternal race/ethnicity groups are presented in these indicators: White non-Hispanic, Black non-Hispanic, Hispanic, American Indian/Alaska Native non-Hispanic, and Asian Pacific Islander non-Hispanic. Prior to the year 1993, not all states recorded Hispanic origin on birth certificates; for this reason, both Indicator H12 and H13 begin with data from 1993. Birth certificates do not include information on family or maternal income, so it is not possible to examine differences or trends by income level.

The indicator graphs show data for all births, singletons and multiples combined. The rates for singletons and multiples are provided in supplemental data tables. Additional supplemental tables highlight differences in rates of preterm birth and term low birth weight by age of the mother.

Please see the Introduction to the Health section for discussion of statistical significance testing applied to these indicators. The NVSS records virtually all births in the United States—approximately 4 million per year. Because of this very large sample size, differences in birth outcomes that appear to be small in magnitude may be found to be statistically significant. Extensive research has been conducted with NVSS data to assess the presence of statistically significant trends and demographic differences, including analyses with much more detail than the one conducted here.[23,84,85]



Data characterization

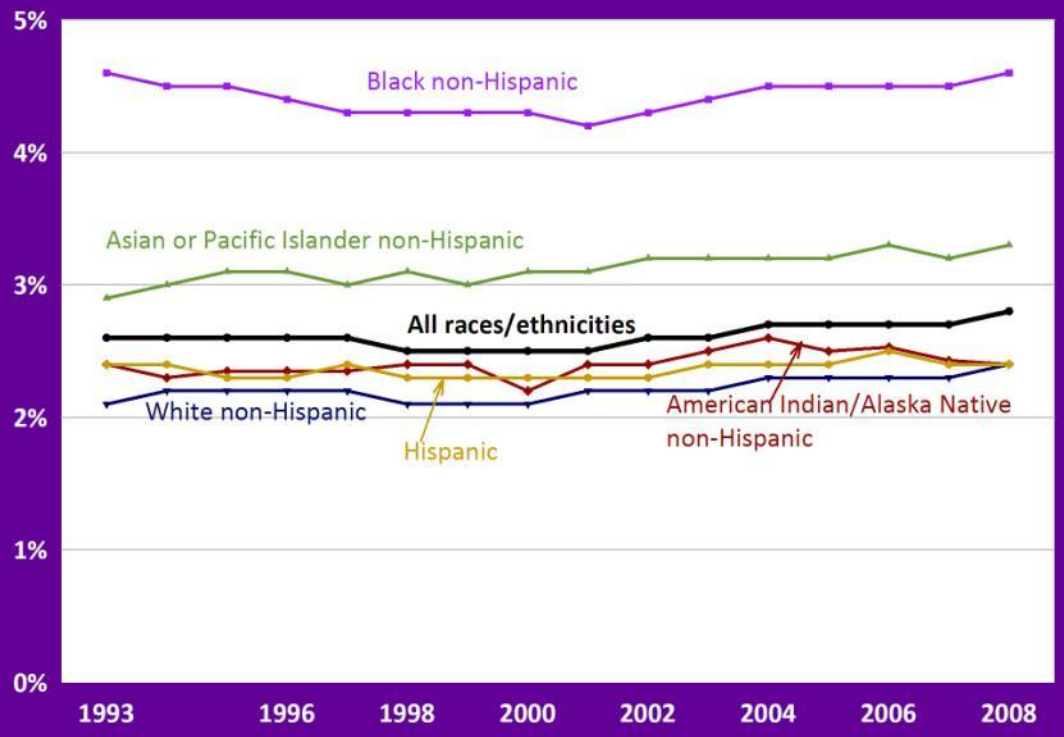
- Data from this indicator are obtained from a database maintained by the National Center for Health Statistics.
- The database collects information from birth certificates for virtually all births in the United States.
- Length of gestation is recorded on each birth certificate.

- Between 1993 and 2008, the rate of preterm birth showed an increasing trend, ranging from 11.0% in 1993 to its highest value of 12.8% in 2006. This increasing trend was statistically significant.
- In 2008, Black non-Hispanic women had the highest rate of preterm birth, compared with women of other races/ethnicities. More than 1 in 6 infants born to Black non-Hispanic women were born prematurely in that year.

  - The difference between the rate of preterm birth for Black non-Hispanic women and the rates for the other race/ethnicity groups was statistically significant.
- Between 1993 and 2008, the preterm birth rate showed an increasing trend for each race/ethnicity group except Black non-Hispanic women. The preterm birth rate for Black non-Hispanic women stayed relatively constant, ranging between 17% and 19%.
  - The increasing trend in the rate of preterm birth was statistically significant for White non-Hispanic, Hispanic, American Indian/Alaska Native non-Hispanic, and Asian or Pacific Islander non-Hispanic women.
- The preterm birth rate varies depending on the age of the mother. Women ages 20 to 39 years have the lowest rate of preterm birth, compared with women under 20 years and women 40 years and older. The rates of preterm birth for women ages 20 to 39 years and women 40 years and older showed an increasing trend between 1993 and 2008; however, the increase for women ages 20 to 39 years was smaller. (See Table H12a.)
  - The differences between the preterm birth rates for the different age groups were statistically significant. The increasing trends in the rate of preterm birth for women ages 20 to 39 years and women 40 years and older were statistically significant as well.
- Twins, triplets, and other higher-order multiple birth babies are more than 5 times as likely to be born preterm compared with singleton babies (60.4% vs. 10.6% in 2008). The preterm birth rates for both singletons and multiples showed an increasing trend from 1993 to 2008; however, the increase for multiples was larger than for singletons. (See Table H12b.)
  - The increasing trend for both singleton and multiple births was statistically significant.

Indicator H13

Percentage of babies born at term with low birth weight, by race/ethnicity, 1993-2008



Data: Centers for Disease Control and Prevention, National Center for Health Statistics, National Vital Statistics System

America's Children and the Environment, Third Edition

Data characterization

- Data from this indicator are obtained from a database maintained by the National Center for Health Statistics.
- The database collects information from birth certificates for virtually all births in the United States.
- Birth weight and length of gestation are recorded on each birth certificate.

- Between 1993 and 2008, the rate of term low birth weight for all races/ethnicities stayed relatively constant, ranging between 2.5% and 2.8%. The rates of term low birth weight for infants born to White non-Hispanic mothers and Asian or Pacific Islander non-Hispanic mothers showed increasing trends between 1993 and 2008, while the rates of term low birth weight for infants born to mothers of the other race/ethnicity groups stayed relatively constant.

- The rate of term low birth weight varies by race/ethnicity. In 2008, the rate was highest for infants born to Black non-Hispanic mothers, and next highest for infants born to Asian or Pacific Islander non-Hispanic mothers. The rate of term low birth weight is lowest for infants born to White non-Hispanic mothers, Hispanic mothers, and American Indian/Alaska Native non-Hispanic mothers.
  - The rate of term low birth weight for Black non-Hispanic women was statistically significantly higher than for all other race/ethnicity groups. The rate of term low birth weight for Asian or Pacific Islander non-Hispanic women was significantly lower than for Black non-Hispanic women but significantly higher than the other race/ethnicity groups.
- Term low birth weight rates vary by the age of the mother. In 2008, women ages 20 to 39 years had the lowest rate of term low birth weight infants, while women under 20 years had the highest rate of term low birth weight infants. (See Table H13a.) These differences were statistically significant.
- Between 1993 and 2008, the rate of term low birth weight for women 40 years and older showed an increasing trend, ranging from 2.9% to 3.4%. This increasing trend was statistically significant. (See Table H13a.)
- Twins, triplets, and other higher-order multiple birth babies are more than 5 times as likely to be born at term with low birth weight compared with singleton babies (12.6% vs. 2.4% in 2008). The rate of term low birth weight for singleton and multiple babies stayed relatively constant over the period of 1993–2008. (See Table H13b.)

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Introduction

The three main sections of *America's Children and the Environment, Third Edition* (ACE3) present environment and contaminant, biomonitoring, and health indicators derived from data sources of national interest, updated on a regular basis. Some topics of interest for children's environmental health do not have suitable national data sources. For some of these topics, ACE3 Supplementary Topics measures have been developed using data sets from a single state, or produced by one-time studies that have not been repeated. The data presentations in this section are referred to as "measures" rather than "indicators" because they are lacking in at least one key characteristic desired for ACE3 indicators.

Measures have been prepared for two Supplementary Topics in ACE3:

- Birth Defects
- Contaminants in Schools and Child Care Facilities

The birth defects topic includes a measure summarizing data from the Texas Birth Defects Registry. The contaminants in schools and child care facilities topic includes three measures of conditions in educational environments. Contaminants in child care facilities are represented by measures drawn from EPA's Children's Total Exposure to Persistent Pesticides and Other Persistent Organic Pollutants (CTEPP) Study, conducted in North Carolina and Ohio, and the First National Environmental Health Survey of Child Care Centers, a federal government study of a nationally representative sample of child care facilities. Contaminants in schools are represented by a measure calculated using a database on pesticide application in schools from the California Department of Pesticide Regulation.

For each Supplementary Topic, an introduction section explains the relevance of the topic to children's environmental health. The introduction section is followed by a description of the measures, including a summary of the data and information on how each measure was calculated. The measures are then presented in graphical form. Beneath each figure are explanatory bullet points describing dataset characteristics and key findings from the data presented in the figure, along with key data from any supplemental data tables. References are provided for each topic at the end of the report.[i]

Data tables are provided in Appendix A. The tables include all values depicted in the Supplementary Topics figures, along with additional data of interest not shown in the figures.

[i] In the current copy, references are provided at the end of each topic section. In the final printed report, references will appear, separated by topic, at the end of the report.

Metadata describing the data sources are provided in Appendix B. Documents providing details of how the measures were calculated are available on the ACE website (www.epa.gov/ace).[ii]

The topics presented in this section are addressed in Healthy People 2020, which provides science-based, 10-year national objectives for improving the health of all Americans. Appendix C provides examples of the alignment of the ACE3 Supplementary Topics with objectives in Healthy People 2020.

[ii] The Methods documents will be posted to the ACE website when the final ACE3 report is posted in early 2013. Detailed methods documents are not provided for Measures S2 and S3 (contaminants in child care facilities) because all values were taken directly from published sources.

Birth Defects

The term "birth defects" covers a range of structural and chromosomal abnormalities that occur while the baby is developing in the mother's body.[1,2] A birth defect may affect how the body looks, works, or both. Some birth defects can be detected before birth, others can be detected when the baby is born, and others may not be detected until some time has passed after birth.

Birth defects are the leading cause of infant death in the first year of life, accounting for about 20% of infant deaths in 2005.[3] Infants who do survive with a birth defect often have lifelong disabilities, such as intellectual disability, heart problems, or difficulty in performing everyday activities such as walking.

Some birth defects are inherited. Others have known risk factors that can be avoided such as prenatal exposure of the fetus to certain pharmaceuticals (such as Accutane® or Thalidomide); exposure to alcohol; maternal smoking, and insufficient folate in a woman's diet.[3-5] For example, birth defects resulting from fetal alcohol syndrome are prevented when a woman does not consume alcohol during pregnancy, and reported cases of neural tube defects such as spina bifida and anencephaly have been shown to decrease following mandatory folic acid fortification of cereal grain products.[6,7] About 60–70% of birth defects have unknown causes, but research suggests that some defects could be modified or caused by environmental factors, possibly in conjunction with genetic factors.[3,8-10] Several environmental contaminants cause birth defects when pregnant women are exposed to high concentrations. Mercury poisoning in Minamata, Japan resulted in birth defects such as deafness and blindness.[11] Prenatal exposures to high concentrations of polychlorinated biphenyls (PCBs) and related chemicals have resulted in skin alterations, including chloracne, a potentially serious inflammatory condition.[12] However, any possible relationship between exposures to lower concentrations of these or other environmental contaminants and birth defects is less clear.

A number of epidemiological studies have evaluated the relationship between environmental and occupational exposures to chemicals and birth defects. The majority of studies consider the relationship of birth defects to exposures to specific types of environmental contaminants, including solvents, pesticides, drinking water disinfection byproducts, endocrine disrupting chemicals, and air pollutants. Some studies consider other scenarios in which individuals may have elevated exposures without measuring or estimating exposure to any particular substances. These studies evaluate factors such as occupational category, or residence near a contaminated site or industrial facility.

Several studies have evaluated the relationship between maternal and paternal solvent exposure and birth defects. An extensive review of the literature concluded that the evidence linking neural tube defects to paternal exposures to solvents was suggestive of an association, although not strong enough to draw a conclusion regarding a causal relationship.[10] A meta-analysis that included multiple studies of women's occupational exposure to organic solvents

reported an increased risk for birth defects such as heart defects and oral cleft defects in children born to exposed women.[13] In a recent study conducted in Massachusetts, women who were exposed to drinking water contaminated with the solvent tetrachloroethylene around the time of conception were reported to have an increased risk of giving birth to a child with a birth defect.[14]

Multiple studies have suggested an association between maternal and paternal exposure to pesticides (both before and after conception) and increased risk of offspring having or dying from birth defects.[15-31] A subsequent review study that evaluated many of these individual studies together, however, concluded that the data are inadequate at this time to confirm an association between pesticide exposure and the risk of birth defects.[10]

Disinfection byproducts in drinking water have also been linked to birth defects in some epidemiological studies. Disinfection byproducts are formed when organic material found in source water reacts with chemicals (primarily chlorine) used in treatment of drinking water to control microbial contaminants. Some individual epidemiological studies have reported associations between the presence of disinfection byproducts in drinking water and increased risk of birth defects, especially neural tube defects and oral clefts; however, recent articles reviewing the body of literature determined that the evidence is too limited to make conclusions about a possible association between exposure to disinfection byproducts and birth defects.[10,32-35]

Some studies have also reported associations between exposure to endocrine disrupting chemicals and urogenital malformations in newborn boys, such as cryptorchidism (undescended testes) and hypospadias (abnormally placed urinary opening).[19,22,36-44] An analysis of a large national database showed a significant increase in the incidence of congenital penile anomalies, particularly hypospadias, from 1988–2000.[45] According to studies by the Centers for Disease Control and Prevention, the prevalence of hypospadias in the United States has doubled in recent decades.[46] This considerable increase, combined with evidence of an association between endocrine-disrupting contaminants and urogenital birth defects in animal studies, has led to the hypothesis that environmental exposures are a contributing factor.[47] However, a review study recently concluded that there is inadequate evidence at this time of associations between male genital birth defects and exposure to environmental contaminants such as pesticides, PCBs, wood preservatives, and phthalates.[10]

A limited number of studies have investigated the relationship between birth defects and prenatal exposure to air pollution, specifically carbon monoxide, ozone, particulate matter, nitrogen dioxide, and sulfur dioxide.[48-57] Most of these studies have focused on cardiac and oral cleft birth defects. A recent pooled analysis of these studies reported statistically significant associations between nitrogen dioxide, sulfur dioxide and particulate matter and certain cardiac birth defects.[58] No statistically significant associations were found between any of the pollutants and oral cleft defects.

Since the discovery of extensive environmental contamination in the Love Canal community in New York State in the 1970s, there has been increased awareness that contaminated sites can be associated with negative birth outcomes, including birth defects.[59,60] Multiple epidemiological studies conducted over the last 25 years have found possible associations between residence near contaminated sites and an increased risk of birth defects, particularly neural tube defects and congenital heart defects.[38,61-64] Studies have also reported associations between residence near hazardous waste sites or active industrial facilities and chromosomal birth defects.[65,66] The majority of these studies use maternal proximity to sites of interest in order to classify exposure and do not distinguish between specific types of contaminant exposures; however, a few studies have reported associations between birth defects and sites that emit heavy metals or solvents.[64,65] Some studies have suggested that the greatest impact may be for mothers residing within a half mile of a contaminated site.[61,67] Studies comparing Superfund sites undergoing assessment or remediation to active industrial facilities reporting toxic chemical releases reported no association between birth defect rates and proximity to Superfund sites, but did report significant associations with proximity to the active industrial sites.[65,68] A recent study of birth defect records for children born to mothers living with proximity to any of 154 Superfund cleanup sites reported an overall reduced incidence of birth defects.[69]

The process of fetal development is intensely complicated, requiring the precise coordination of cell division, growth, and movement. During the process of fetal development there are critical periods of susceptibility or vulnerability, at which point exposure to environmental contaminants may be especially damaging.[70] For example, two air pollution epidemiology studies found that the first two months of gestation are a particularly vulnerable period, during which exposure to air pollutants may cause birth defects of the heart and oral clefts.[52,56] Similarly, studies hypothesizing a role for pesticide exposure in birth defects have reported that conception during the spring is a risk factor for birth defects.[25,29,71] Agricultural use of certain pesticides is at its highest during spring, potentially leading to increased exposures that could contribute to the observed seasonal pattern in the incidence of birth defects.[25,29,71] These types of studies are useful for generating hypotheses for future research investigating the relationship between environmental exposures and the development of birth defects.

There is currently no unified national monitoring system for birth defects. Information on prevalence of birth defects comes from birth certificates and from state birth defects monitoring systems. Many birth defects can be observed shortly after delivery and are recorded on birth certificates. A national-scale indicator could be constructed using birth certificate data, but would miss any birth defect that is not immediately recognized and recorded at birth. Comparisons of birth defects recorded on birth certificates and birth defect registries have indicated that typically, less than half of birth defects are recorded on birth certificates.[72,73] Most states have some type of birth defects monitoring program, although the type of tracking varies widely among the states. As of 2008, 45 states had some type of existing birth defects monitoring program.[74] A small portion of these states have the most complete type of tracking system, which includes actively researching medical records for birth defects and following

children through at least the first year of life. The remaining states have some type of monitoring program, but do not have all the aspects of a complete surveillance system. The National Birth Defects Prevention Network has pooled data from several state registries to derive prevalence estimates for a subset of 21 selected birth defects for the years 1999–2001 and 2004–2006.[75]

The Texas monitoring program, which has monitored birth defects since 1995, is considered one of the most complete in the nation.[76] Data from the Texas registry for several categories of birth defects are presented in this section, as an example.

Measure S1: Birth defects in Texas, 1999–2007

About the Measure: Measure S1 presents information about the number of infants born with birth defects in Texas. The data come from a registry of birth defects for the state of Texas, which compiles data on any birth defects identified in the first year after each child is born. The Texas Registry staff routinely review medical records at all hospitals and birthing centers where babies are delivered or treated to identify birth defects. Measure S1 shows how the rates of different types of birth defects have changed over time. The rates of birth defects in Texas are not necessarily representative of those in other states.

The Texas Birth Defects Registry

The Texas Birth Defects Epidemiology and Surveillance Branch of the Texas Department of State Health Services provides information on birth defects in the state of Texas. The Texas program began monitoring the Houston/Galveston and South Texas areas in 1995 and expanded so that beginning in 1999, it covered the entire state. The Texas monitoring program covers approximately 380,000 births each year, which represents almost 10% of all births in the United States. In addition to live births, the Texas monitoring program also covers birth defects occurring in a fetal death or pregnancy termination. The Texas monitoring program reports a wide array of birth defects.

Although most states have a birth defects monitoring program in place, the comprehensiveness of these programs varies. Texas's birth defects monitoring program is one of the most complete in the nation, using high-quality active surveillance methods to examine a wide range of birth defects throughout a child's first year of life.[76] Specifically, the Texas Registry staff employ robust approaches to collecting, verifying, and ascertaining cases of birth defects such as routinely visiting all hospitals and birthing centers where babies are delivered or treated to individually review logs, discharge lists, and medical records.[77] As a result, a joint review by the Trust for America's Health and the National Birth Defects Prevention Network of the birth defects tracking activities in all 50 states assigned the Texas Registry their highest grade ranking, based on a number of criteria such as the ability to carry out tracking and the resources devoted to the task.[76] Although the Texas Registry data are of high quality, the rates and types of birth defects in Texas are not necessarily representative of those in other states.

Comparing the Texas Birth Defects Registry with Other Data Sources

To examine whether the rate of birth defects in Texas is similar to the rate for the country as a whole, it is useful to compare birth defect rates from birth certificates. Birth certificates record only those birth defects apparent at birth, and do not represent defects that become apparent after some time. Most states report birth defects on birth certificates using the standard birth certificate format recommended by the National Center for Health Statistics. The birth certificate reported rates of birth defects for Texas are generally similar to the nationwide rates.[78]

Comparing the Texas Birth Defects Registry data to the birth certificate data for Texas reveals that the active surveillance strategies detect a far greater number of birth defects than can be detected at an infant's birth. For specific birth defects that could be directly compared, the Texas monitoring program typically detects two to three times the number of birth defects reported on birth certificates, demonstrating the importance of tracking birth defects that are not observed at the time of delivery.[77,78] Texas birth certificates list potential birth defects for clinicians to choose from when recording the details of an infant's birth. An analysis by the Texas Birth Defects Registry found that birth certificates identify these listed birth defects only 15% of the time that they occur. Furthermore, of those birth defects listed on Texas birth certificates, the most obvious birth defects, such as spina bifida and cleft palate, are only identified 36-42% of the time.[73]

As mentioned previously, there is currently no unified national monitoring system for birth defects. However, CDC, in collaboration with the National Birth Defects Prevention Network, pools data from states with active and passive monitoring programs to estimate national prevalence rates for several selected birth defects. The pooled data set currently accounts for about 30% of births nationwide.[75]

Data Presented in the Measure

Measure S1 displays the number of birth defects per 10,000 live births for the state of Texas. Measure S1 shows data for 1999–2007 and groups birth defects by structural categories. A supplemental data table for this measure provides information showing how birth defect rates vary by race/ethnicity.[iii]

Trends in the rates of birth defects may be influenced by differences in clinical practice. For example, increasing trends in the prevalence of some birth defects could represent more accurate recording of birth defects and/or better diagnosis of subtle defects due to the use of more sensitive examinations and technology.[79-82] Trends for specific birth defects may also be masked when grouping birth defects by structural categories. For example, anencephaly is included in the structural category of central nervous system defects. Incidence of central nervous system birth defects overall in Texas increased from 1999–2007, but the incidence of anencephaly defects specifically appear to be decreasing in the same years.[83]

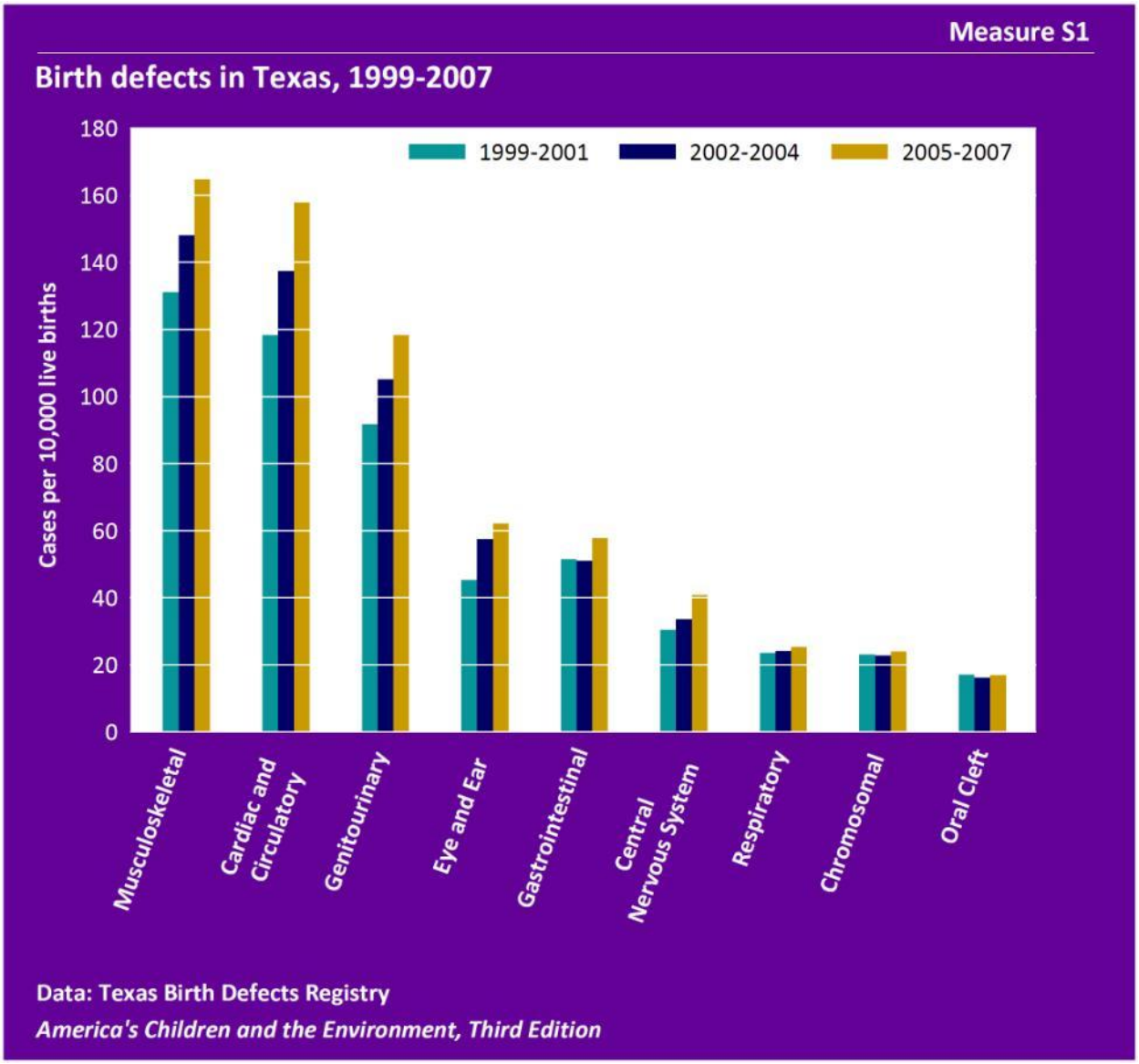
Statistical Testing

Statistical analysis has been applied to Measure S1 to evaluate trends over time or differences between demographic groups in the prevalence of birth defects. These analyses use a 5% significance level, meaning that a conclusion of statistical significance is made only when there

[iii] 95% confidence intervals for the birth defects rates are provided in a file available on the ACE website (www.epa.gov/ace). (This file will be posted to the ACE website when the final ACE3 report is posted in early 2013.)

is no more than a 5% probability that the observed trend or difference occurred by chance ($p \leq 0.05$). The statistical analysis of trends over time is dependent on how the values in the measure vary over time as well as on the number of time periods. For example, the statistical test is more likely to detect a trend when data have been obtained over a longer period. A finding of statistical significance for differences between demographic groups depends on the magnitude of the difference and the number of observations in each group. It should be noted that conducting statistical testing for multiple categories of birth defects increases the probability that some trends or differences identified as statistically significant may actually have occurred by chance.

A finding of statistical significance is useful for determining that an observed trend or difference was unlikely to have occurred by chance. However, a determination of statistical significance by itself does not convey information about the magnitude of the increase, decrease, or difference. Furthermore, a lack of statistical significance means only that occurrence by chance cannot be ruled out. Thus a conclusion about statistical significance is only part of the information that should be considered when determining the public health implications of trends or differences in the prevalence of birth defects.



Data characterization

- Data for this measure are obtained from the Texas Birth Defects Registry.
- The Registry employs robust surveillance methods to monitor all births in Texas and identify cases of birth defects.
- The Registry represents almost 10% of all births in the United States, but the rates and types of birth defects in Texas are not necessarily representative of those in other states.

- Musculoskeletal defects are the most common type of birth defect in Texas, with 165 cases per 10,000 live births for the years 2005–2007. The second most common type of birth defect in Texas is cardiac and circulatory, with 158 cases per 10,000 live births for the years 2005–2007.

- The rates for all categories of birth defects in Texas have increased or remained stable for the period of 1999–2007. Some of the biggest increases were seen for musculoskeletal defects, cardiac and circulatory defects, genitourinary defects, eye and ear defects, and central nervous system defects.
  - The increases were statistically significant for musculoskeletal defects, cardiac and circulatory defects, genitourinary defects, eye and ear defects, gastrointestinal defects, and central nervous system defects.
- The prevalence of birth defects varies by race/ethnicity for most of the anatomical categories examined. Compared with White non-Hispanics, Black non-Hispanics had lower rates of musculoskeletal, genitourinary, eye and ear, gastrointestinal, chromosomal, and oral cleft birth defects, and these differences were statistically significant. There were no statistically significant differences between Black non-Hispanics and White non-Hispanics in rates of cardiac and circulatory, central nervous system, and respiratory birth defects. (See Table S1a.)
- Compared with White non-Hispanics, Hispanics had higher rates of cardiac and circulatory, eye and ear, and respiratory defects, whereas rates of musculoskeletal and genitourinary birth defects were lower. These differences were statistically significant. There were no statistically significant differences between Hispanics and White non-Hispanics in rates of gastrointestinal, central nervous system, chromosomal, and oral cleft defects. (See Table S1a.)

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Contaminants in Schools and Child Care Facilities

The indoor and outdoor environmental quality of schools and child care facilities plays an important role in affecting children's health and academic performance. Depending on the type of facility and its particular characteristics (i.e., age, usage, and maintenance), children may be exposed to contaminants from a variety of indoor and outdoor sources. Potential indoor exposure sources include building materials and furnishings (such as paint, treated wood, furniture, carpet, and fabrics), products used for building maintenance (such as cleaning products and pesticides), and products used for hobbies, science projects, and arts and crafts projects or within the learning environment (such as paint, markers, and correction fluid). Potential outdoor exposure sources include air pollution from nearby traffic and industry. In addition to these specific exposures, children may also experience unsatisfactory environmental conditions such as inadequate lighting, ventilation, indoor air quality, or noise control.[1] These exposures potentially impact the comfort and health of students, which may adversely affect their academic performance and increase their risk of both short- and long-term health problems.[2-4]

These potential exposures are of particular concern because children generally spend most of their active, awake time at schools and child care facilities. Children are especially sensitive to contamination, for several reasons. First, children are biologically more vulnerable than adults since their bodies are still growing and developing.[5-7] Second, children's intake of air and food is proportionally greater than that of adults. For example, relative to body weight, a child may breathe up to twice as much air as adults do; this increases their sensitivity to indoor air pollutants.[8] In particular for younger children, the inhalation and ingestion of contaminated dust is a major route of exposure due to their frequent and extensive contact with floors, carpets, and other surfaces where dust gathers, such as windowsills, as well as their high rate of hand-to-mouth activity.[8] Lastly, children have many years of future life in which to develop disease associated with exposure.[7]

School and child care environments share many characteristics influencing children's exposure to indoor environmental contaminants, such as the sources and types of potential environmental contaminants. Both environments also tend to house a large number of occupants in a small confined space, so that without proper ventilation a large number of children can be at risk for potential exposure to indoor contaminants.[9] However, there are also a number of important differences between the two. Children in child care facilities are generally much younger than those in schools, sometimes as young as a few weeks old. The behaviors of very young children (e.g., crawling, hand-to-mouth activity) increase their exposure to contaminants in dust, on surfaces, or in toys and other objects.[6,10] Younger children may also spend more time in child care facilities, some as many as 10 hours per day, 5 days a week.[11,12] Also, compared with schools, child care facilities can be located in a much wider variety of settings, including office buildings, individual homes, and religious buildings. As a result, the indoor and outdoor environments can differ widely between child care facilities and may not be directly under the control of those running the child care itself. Furthermore, child

care facilities are more often operated independently, while schools are frequently part of a school district with centralized facilities management. This has important implications for strategies to address environmental issues in these facilities.

Building upkeep characteristics are extremely important, because the design, construction, and current condition of school and child care center facilities can contribute to children's exposure to environmental contaminants.[13-17] Age, level of deterioration, and ventilation efficiency are key characteristics that determine a building's indoor environmental quality. Many substances are released into the indoor environment as a result of deterioration of the building from old age, poor maintenance, or through improperly managed removal and renovation processes.[15,18]

Children may be exposed to a variety of contaminants in school and child care settings, such as lead, asbestos, polychlorinated biphenyls (PCBs), pesticides, brominated flame retardants, phthalates, and perfluorinated chemicals. Exposure to indoor contaminants can occur through multiple routes, such as dermal (through the skin), inhalation, and direct and indirect ingestion. These types of indoor environmental contaminants have been associated with a variety of adverse health outcomes, as well as outcomes related to educational performance for which impaired health is a suspected cause.[19,20] These adverse health effects may be short-term (headache, dizziness, nausea, allergy attacks, or respiratory problems) or longer-term and more serious (asthma, neurodevelopmental effects, or cancer).[21] Children exposed to indoor air pollution also miss more days of school due to illness.[14,22] A child's overall academic performance can suffer as a result of such an illness or absence.[23] For example, exposure to indoor air pollutants has been associated with decreased concentration and poor testing outcomes.[24-26]

There is evidence that many schools and child care facilities in the United States have significant and serious problems with indoor environmental contaminants,[27] and certain groups of children are especially susceptible to such exposures.[28] Children with allergies, asthma, and other respiratory problems are especially susceptible to the effects of indoor air pollution. Asthma attacks and allergies are often triggered by indoor allergens (pollen, dust, cockroaches), as well as by mold.[29]

Lead

Lead is a pervasive and serious environmental health threat for children in the United States.[30,31] The most common sources of lead exposure in schools and child care environments are lead-based paint, lead dust, and lead-contaminated soil in outdoor play areas.[32] This is a particular concern for young children, due to their frequent and extensive contact with floors, carpets, window areas, and other surfaces where dust gathers, as well as their frequent hand-to-mouth activity.[33] A nationally representative sample of licensed child care facilities in 2001 estimated that approximately 14% of these facilities in the United States have significant lead-based paint hazards. Most of these are facilities in older buildings: 26% of facilities located in a building built before 1960 were found to have lead-based paint hazards, compared with 4% in newer buildings. [11]

Additional sources of lead may include lead in drinking water, lead-contaminated products (such as toys, books, and jewelry), and outdoor air from nearby industry.[33-35] The ingestion and inhalation of lead-contaminated dust are the primary pathways of childhood exposure to lead.[36] The National Toxicology Program has concluded that childhood lead exposure is associated with reduced cognitive function, reduced academic achievement, and increased attention-related behavioral problems.[30] Studies have reported associations of childhood exposure to lead with behavioral problems such as attention-deficit/hyperactivity disorder,[37-44] increased likelihood of school absenteeism and of dropping out of high school,[45] increased risks of juvenile delinquency and antisocial behaviors,[46-49] higher total arrest rates, and arrest rates for violent crimes in early adulthood.[50,51]

Polychlorinated Biphenyls (PCBs)

PCBs are a family of industrial chemicals used primarily as cooling or insulating fluids for electrical equipment or as additives to paints, plastics, and rubber products.[20] While the manufacture of PCBs was banned in 1979, PCBs continue to be present in products and materials produced before the ban. Many schools in the United States have lighting systems containing PCBs. When contained in the lighting systems, PCBs pose very little health risk or environmental hazard.[52] However, lighting systems degrade as they age, increasing the risk of PCB leaks or even fires, which pose health and environmental hazards. In December 2010, EPA issued guidance recommending that schools take steps to reduce potential exposures to PCBs from these types of older lighting fixtures.[53] PCBs are also found in caulk and paint used in building structures before 1980,[54,55] which may mobilize into the surroundings from removal efforts, natural weathering, or deterioration over time, and contribute significantly to PCB levels in indoor air and dust in schools.[56,57] Although there is some inconsistency in the epidemiological literature, several reviews of the literature have concluded that the overall evidence supports a concern for adverse effects of PCBs on children's neurological development.[58-62]

Asbestos

Asbestos is a naturally occurring mineral fiber that has been used in building materials as an insulator and fire retardant.[63] The production and use of building materials containing asbestos is currently limited by law in the United States,[64] but many older schools and other buildings may have asbestos-containing materials that were previously allowed in construction. The Asbestos Hazard Emergency Response Act provides rules for the management of asbestos in schools.[65] Under this law, some asbestos-containing products are removed when found, but most often it is recommended that they are "managed-in-place"—i.e., maintaining and managing the contaminated material to reduce potential exposure. Properly managed asbestos that has not been disturbed poses little health risk to students. However, if asbestos-containing materials are disturbed or begin to deteriorate, they can release hazardous fibers into the air and water. Long-term exposure to these fibers can lead to lung cancer, asbestosis (lung scarring), or mesothelioma (cancer of the lung cavity lining).[66,67] These diseases require a long

time to develop following exposure, putting children at greater risk of disease development later in life.

Other Indoor Contaminants

Cleaning products and maintenance activities in schools and child care facilities are significant sources of exposure to chemical contaminants. Many conventional cleaning supplies contain harmful chemicals that have been associated with various health effects, including asthma and cancer.[68] Additionally, maintenance activities, from routine cleaning to renovation, can cause dust and particulate matter to become airborne, leading to increased opportunity for inhalation and ingestion of contaminated particles.[69]

Children also may be exposed to a variety of other hazardous chemicals in these environments, such as glues, paints, and other art supplies; mercury from older thermometers; a range of chemicals in chemistry labs; lead acid in batteries and other automotive and trade shop supplies; formaldehyde in pressed wood furniture, flooring, carpets, curtains, and cleaning products; volatile organic compounds (VOCs) in paints, aerosol sprays and fresheners, cleaning supplies, and building materials and furnishings; and the wide variety of toxic chemicals found in environmental tobacco smoke.[70] These and other chemicals commonly found in indoor air have been associated with a range of short-term effects, such as eye, lung, and skin irritation; headaches; nausea; fatigue; and a range of long-term health effects, from chronic lung irritation to cancer, depending on the specific chemical.

In addition to these direct sources of potential exposure, inefficient or malfunctioning heating, ventilation, and air conditioning (HVAC) systems may increase the risks of adverse health effects or even become an additional source for indoor contaminant exposures. First, failing to provide sufficient circulation and filtration of the indoor air mixed with fresh outdoor air can lead to an accumulation of existing air pollutants to dangerous levels.[9,71] This includes increased levels of the chemical contaminants already discussed, as well as other environmental contaminants such as particulate matter and allergens such as cockroach allergen, rodent dander, or pollen.[72] Second, failing to adequately control moisture and temperature levels can trigger the growth of dust mites and mold, which thrive in damp, warm environments.[19,72] Exposure to these are known to cause asthma or trigger asthma attacks.[73,74] Inefficient HVAC capabilities are of particular concern in temporary classroom structures, such as trailers and portable classrooms, which have been associated with poor indoor air quality due to a combination of inadequate ventilation along with use of toxic building materials. A state-wide survey of permanent and portable classrooms in California found that, on average, portable classrooms had worse indoor air quality than permanent ones did, including less efficient or improperly functioning HVAC systems; higher levels of indoor air formaldehyde, particulate matter, polycyclic aromatic hydrocarbons (PAHs), and humidity; and temperatures above and below thermal comfort standards during warm and cool seasons, respectively.[75]

School Siting

School siting (selecting a site, or location, for a new school) is a complex process that often requires assessment of several considerations, such as whether to renovate an old school or to build a new one, cost of land and location preparation, and the availability of infrastructure including roads and utilities. EPA has recently developed voluntary guidelines for school siting as a way to support states, tribes, communities, local officials, and the public in understanding and appropriately considering environmental and public health factors when making school siting decisions. These siting guidelines address issues such as the special vulnerabilities of children to hazardous substances or pollution exposures, modes of transportation available to students and staff, the efficient use of energy, and the potential use of the school as an emergency shelter.[17]

School locations may have underlying causes of potential exposure, such as site contamination, neighborhood emission sources, or indoor air quality problems.[17] Radon, a naturally occurring gas, can seep into buildings from soil. A nationwide survey of radon levels in schools estimates that nearly one in five schools has at least one schoolroom with a short-term radon level above the level at which EPA recommends that schools take action.[76] Additionally, children attending schools near highways or industrial sources may be exposed to various air pollutants such as ozone, particulate matter, carbon monoxide, VOCs, and lead. These potential exposures may pose either short-term or long-term health risks to children who utilize school facilities.[17]

Pesticides in Schools and Child Care Facilities

Pesticides are used in the indoor and outdoor environment to prevent, destroy, repel, or otherwise control pests such as rodents, insects, unwanted plants, and microbials (such as bacteria). They can be sold in many different forms, such as sprays, powders, crystals, or balls, and thus their application inside or outside of schools and child care facilities may lead to several potential routes of exposure for children. For example, application of pesticides in the indoor environment has been shown to contaminate untreated surfaces, including kitchen counters and toys,[77-83] indoor air,[77-79,83-87] and dust.[84,88-92]

Once applied, pesticide residues may take anywhere from a few hours to several months or years to completely break down (degrade). Pesticide residues in the indoor environment are less exposed to factors, such as sunlight, that enable their degradation, and therefore are more persistent than those pesticide residues in the outdoor environment.[82,93,94] This persistence means that pesticide exposures can remain a potential concern for a long period of time, even if the area is no longer being treated. For example, an assessment of pesticide residues in dust of inner city homes found a high prevalence of the pesticide chlorpyrifos two to three years after its indoor use was banned.[90] DDT also continues to be measured in indoor dust several decades after its use was banned in the United States.[91,92,95,96] Furthermore, the persistence of pesticides in the environment after application creates not only an opportunity for children to be exposed directly to the residues, but also the potential for residue migration, leading to

contamination of untreated areas.[82,97] As a result, exposures may occur long after application and through a variety of routes such as inhalation and indirect ingestion of dust.[77]

Outdoor pesticide applications on school property, as well as on nearby agricultural fields, lawns, or house perimeters, may contaminate nearby schools and child care facilities.[77] Several studies demonstrate increased levels of pesticides in indoor air[82,98] and dust[95,98] following pesticide applications in an adjacent outdoor area. This often occurs when outdoor air contaminated with pesticide residues mixes with the indoor air (through natural drifting into the building or being brought in through HVAC systems), or residue particles are tracked in on the shoes and clothing of people entering the building.[80,82,95,98,79]

Few studies have evaluated pesticide exposures in the school environment. Some states have conducted studies of pesticide occurrence in their schools. A comprehensive survey of public K–12 classrooms was conducted by the state of California between October 2001 and February 2002.[99] The California study found residues of both available and restricted-use pesticides in all floor dust samples, and concluded that pesticides enter classrooms either during application or by being tracked in on clothing or shoes from outdoors. Pesticides detected in more than 80% of the samples include *cis*- and *trans*-permethrin, chlorpyrifos, and piperonyl butoxide. The First National Environmental Health Survey of Child Care Centers evaluated potential pesticide exposures in child care facilities, and reported that 75% of licensed child care facilities had at least one pesticide application in the past year.[97] The study detected numerous organophosphate and pyrethroid pesticides in indoor floor wipe samples. Chlorpyrifos, diazinon, and permethrin were detected in more than 67% of the tested centers.[97]

Several studies have reported associations between exposure to pesticides in early life and adverse health effects such as cancer and neurodevelopmental disorders. Childhood leukemia in particular has been associated with childhood exposures to pesticides.[100-104] Permethrin and resmethrin, which both belong to the commonly used class of pesticides known as pyrethroids, were recently classified by EPA as "likely to be carcinogenic to humans."[104] Childhood exposures to organophosphate pesticides have been associated with various adverse neurodevelopmental effects.[105-107] Exposure to herbicides and/or other pesticides in the first year of life has been associated with higher risk of asthma.[108]

The short- and long-term health effects of exposure to pesticides in the school environment are largely unknown, due to a lack of data. Between 1993 and 1996, there were 2,300 pesticide-related exposures reported to poison control centers that involved individuals at schools, resulting in 329 people seen in health care facilities, 15 hospitalized, and 4 treated in intensive care units.[109] Data on the long-term effects of pesticide exposure in schools are not available.[109]

Currently, there is no federal law on pesticide use in the school environment. However, at least 35 states have adopted laws on pesticide use in schools.[110] The state laws are generally focused on the adoption of certain types of practices that eliminate or minimize the use of hazardous pesticides: adoption of Integrated Pest Management (IPM) programs, prohibiting when and

where pesticides can be applied, requiring signs before and after indoor and outdoor pesticide application, requiring prior written notification to parents and staff for pesticide use, and establishing restricted buffer zones to address chemicals drifting into school yards and buildings. Strategies such as restrictions on the use of pesticides and adoption of IPM have been shown to be effective at reducing human exposure.[87,111,112]

There is no national system for compiling data on the amount of pesticides used in schools.[109] Some states require reporting on pesticide use in schools. The state of Louisiana requires schools to submit a written record of "restricted use" pesticides used annually.[113] In the state of New York, commercial applicators are required by a 1996 law to report the amount of each specific pesticide used and the location where it was applied. Also, six states—Arizona, California, Connecticut, Massachusetts, New Hampshire, and New Mexico—require commercial applicators to report the amount of specific pesticides used.[109]

Measures in This Section

Data on school or child care environmental exposures are not systematically collected. Over the years, there have been few national and state-specific surveys or assessments to acquire information on environmental hazards in educational facilities. The following two measures provide data on the use or presence of pesticides and other chemicals of concern indoors in schools and child care facilities. Measures S2 and S3 present data on detectable levels of pesticides and other contaminants in a regional and national sample of child care centers. Measure S4 presents data on the amount of pesticides applied in schools in California.

Measure S2: Percentage of environmental and personal media samples with detectable pesticides in child care facilities, 2001

Measure S3: Percentage of environmental and personal media samples with detectable industrial chemicals in child care facilities, 2001

About the Measures: Measures S2 and S3 present information about the types of contaminants that were detected in child care facilities. The data come from two different studies. One study collected information from selected child care facilities in Ohio and North Carolina, while the other study collected information from child care facilities throughout the United States. The measures show how frequently different contaminants were detected in various media samples (e.g., indoor air, dust) taken at the testing locations.

CTEPP Study and the First National Environmental Health Survey of Child Care Centers

Measures S2 and S3 present data on the relative potential exposures of children to a variety of pesticides and other contaminants found in child care centers. The measures are based on data from two different federal studies: the Children's Total Exposure to Persistent Pesticides and Other Persistent Organic Pollutants (CTEPP) Study and the First National Environmental Health Survey of Child Care Centers. Data shown in these measures were obtained directly from these sources:

> Tulve, N.S., P.A. Jones, M.G. Nishioka, R.C. Fortmann, C.W. Croghan, J.Y. Zhou, A. Fraser, C. Cave, and W. Friedman. 2006. Pesticide Measurements from the First National Environmental Health Survey of Child Care Centers Using a Multi-Residue GC/MS Analysis Method. *Environmental Science and Technology* 40(20) 6269-6274.
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> Morgan, M.K., L.S. Sheldon, C.W. Croghan, J.C. Chuang, R.A. Lordo, N.K. Wilson, C. Lyu, M. Brinkman, N. Morse, Y.L. Chou, C. Hamilton, J.K. Finegold, K. Hand, and S.M. Gordon. 2004. A Pilot Study of Children's Total Exposure to Persistent Pesticides and Other Persistent Organic Pollutants (CTEPP), Appendix I and Appendix J. Research Triangle Park, NC: U.S. Environmental Protection Agency. http://www.epa.gov/heasd/ctepp/.

The CTEPP study investigated the potential exposures of 257 preschool children, ages 1.5 to 5 years, and their primary adult child care providers to more than 50 anthropogenic chemicals, including pesticides, PAHs, PCBs, phthalates, and phenols. This regional study was conducted by EPA at 29 child care centers in North Carolina and Ohio in 2000–2001. Environmental (indoor and outdoor air, carpet house dust, and soil) and personal (hand wipe, solid and liquid food, drinking water, and urine) samples were collected for each child in the study at home and at the child care center over a 48-hour period.[114]

The First National Environmental Health Survey of Child Care Centers was conducted by the U.S. Department of Housing and Urban Development, the Consumer Product Safety Commission, and EPA in 2001. Indoor and outdoor environmental media samples (surface wipes and soil samples) from a nationally representative sample of 168 child care centers were tested for lead, allergens, and pesticides. No personal samples were collected.

Data Presented in the Measures

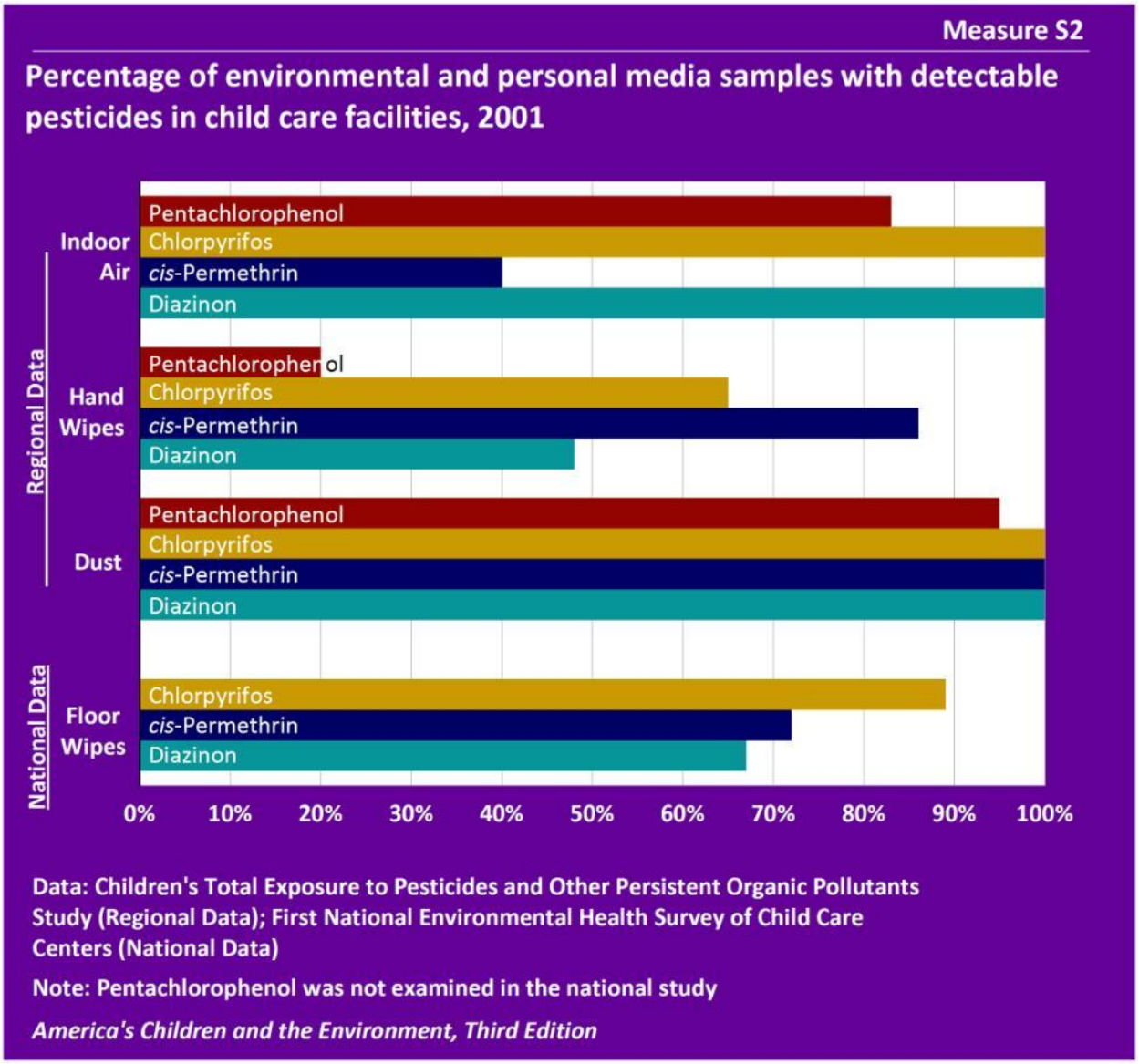
Measure S2 presents the percentage of environmental and personal media samples (indoor air, hand wipe, dust, and floor wipe samples) taken from selected regional and national child care facilities with detectable pesticides. Measure S3 presents the percentage of environmental and personal media samples (indoor air, hand wipe, and dust samples) taken from selected regional child care facilities with detectable industrial chemicals. The "Regional Data" in the first graph and all data in the second graph are derived from the CTEPP study, and reflect the percentage of media samples with detectable pesticides and chemical residues. The chemicals were each measured in 42–43 indoor air and dust samples collected from child care centers in Ohio and North Carolina, and the chemicals were measured in hand wipe samples collected from 60–61 children attending those child care centers. The "National Data" in the first graph are derived from The First National Environmental Health Survey of Child Care Centers, and reflect the percentage of 168 floor wipe samples with detectable chemical residues. The level that is detectable is determined by the capabilities of the sampling and testing equipment used in a study; therefore, it cannot be completely ruled out that contaminants are present at lower levels in samples classified as being below the detection limit. Both measures are based on whether the contaminant is detected or not detected, and thus provide an indication of potential for exposure, but they do not provide data on concentrations of the chemicals or levels of exposure.

The "indoor air" category reflects children's potential exposure to airborne chemicals through inhalation. The "hand wipes" category is based on sampling for the presence of chemicals on children's hands. Due to children's high levels of hand-to-mouth activity, hand wipe data indicate potential exposure via ingestion.[8] The "dust" category captures contaminants that accumulate in dust on various indoor surfaces, and reflects potential inhalation exposure to contaminants if dust is resuspended in the air, as well as indirect ingestion if dust contaminates items that children put in their mouths, such as food, toys, and their hands.

The specific pesticides shown in Measure S2 are pentachlorophenol, an organochlorine pesticide that has been used in the past in some paints, and in industrial and agricultural practices, but which is now limited to use in wood railroad ties and utility poles; chlorpyrifos, an organophosphate insecticide used previously indoors against cockroaches, fleas, and termites, and currently used on farms to control pests on animals and crops and in warehouses, factories, and food processing plants; *cis*-permethrin, a synthetic pyrethroid used to kill and repel domestic insects; and diazinon, an organophosphate pesticide with current agricultural uses and previous residential uses.

The industrial chemicals shown in Measure S3 are PCB-52, polycyclic aromatic hydrocarbons (PAHs, represented in the measure with data for the PAH benzo[b]fluoranthene), dibutyl phthalate, and bisphenol A. While the manufacture of PCBs was banned in 1979, PCBs continue to be present in electrical equipment and some building materials, such as caulk, produced before the ban. Several PCBs were measured in the CTEPP study; data for PCB-52 are displayed in the graph because it is one of the PCBs most frequently detected in the study, and thus gives an indication of potential for exposure to PCBs in general. Benzo[b]fluoranthene is one of several PAHs measured in the CTEPP study. Mixtures of PAHs are produced when carbon-based fuels are burned. Data for benzo[b]fluoranthene are displayed in the graph because it is one of the PAHs most frequently detected in the study, and thus gives an indication of potential for exposure to PAHs in general. Dibutyl phthalate is a chemical commonly used in adhesives, plastics, and personal care products. Bisphenol A is a high-volume industrial chemical used in the production of epoxy resins and polycarbonate plastics. Polycarbonate plastics may be encountered in many products, notably food and drink containers, while epoxy resins are frequently used as inner liners of metallic food and drink containers to prevent corrosion.

Many of these pesticides and industrial chemicals are no longer available or have highly restricted uses. Manufacture of PCBs and PCB-containing equipment and materials was banned in 1979, though equipment and materials manufactured with PCBs prior to the ban remain in use. Pentachlorophenol has not been used other than as a wood preservative since 1987. Indoor application of chlorpyrifos, and any use at schools, was restricted beginning in 2001. All indoor uses of diazinon were banned in 2001.

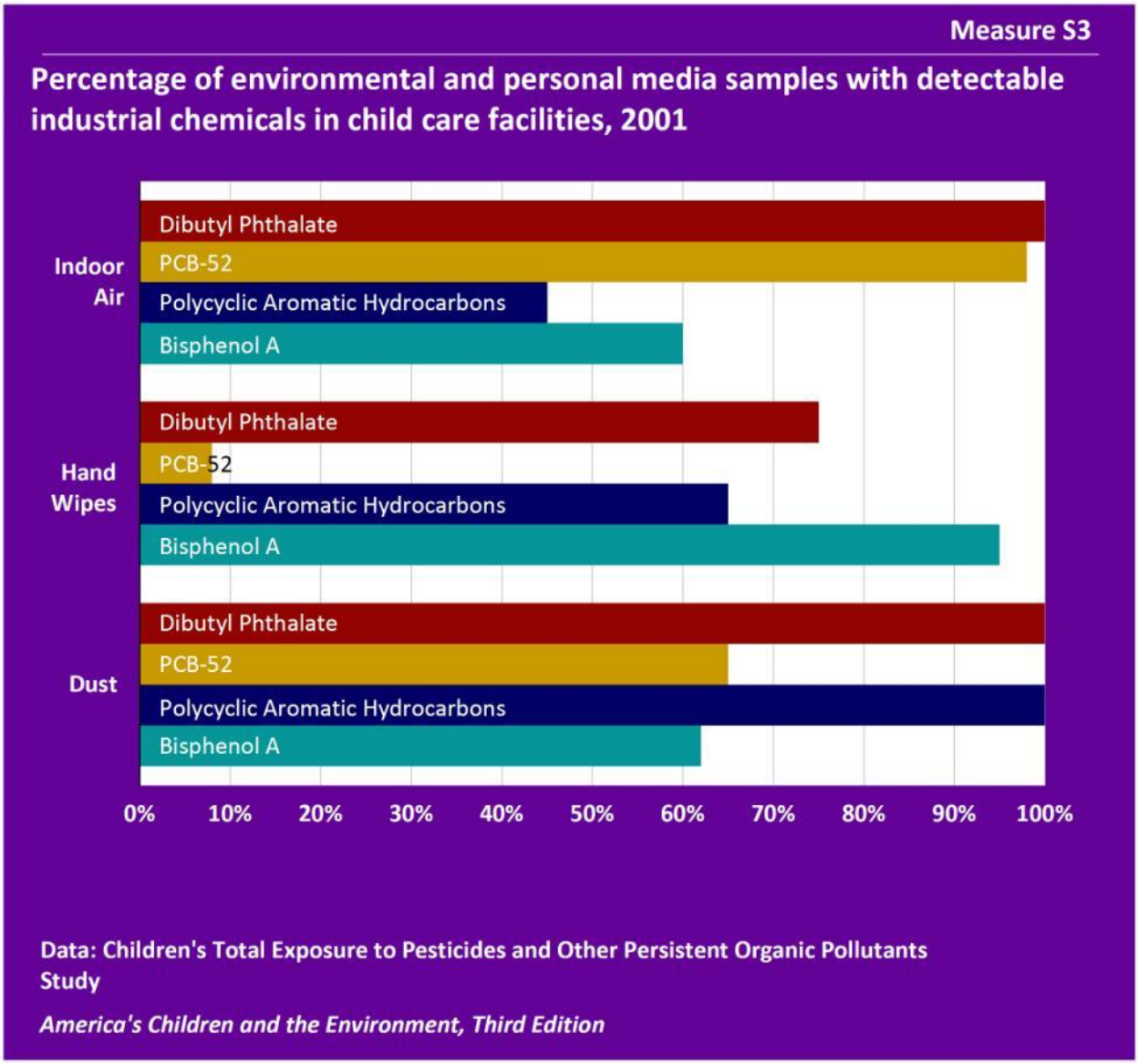


Data characterization

- National data for this measure were obtained from a federal government study of a nationally representative sample of 168 child care centers. Pesticides were measured in environmental samples collected from the child care centers.
- Regional data for this measure were obtained from an EPA study of 29 child care centers in Ohio and North Carolina. Pesticides were measured in environmental samples collected from the child care centers and from the hands of children in the centers.

- Chlorpyrifos, *cis*-permethrin, and diazinon were detected in all of the dust samples collected at Ohio and North Carolina child care centers included in the CTEPP study in 2000-2001. Chlorpyrifos and diazinon were also detected in all of the indoor air samples collected at these child care centers.

- Pesticide residues were detected least often in the hand wipe samples collected at the selected Ohio and North Carolina child care centers, but chlorpyrifos and *cis*-permethrin were detected in more than half of the hand wipe samples.
- The national level floor wipe sampling found chlorpyrifos most frequently, in 89% of samples. *Cis*-permethrin and diazinon were also detected frequently, in 72% and 67% of floor wipe samples, respectively. (Pentachlorophenol was not examined in the national study.)



Data characterization

- Data for this measure were obtained from an EPA study of 29 child care centers in Ohio and North Carolina.
- Chemicals were measured in environmental samples collected from the child care centers and from the hands of children in the centers.

- Of the chemicals shown in this measure, dibutyl phthalate was the most frequently detected in indoor air and dust samples collected at Ohio and North Carolina child care centers included in the CTEPP study in 2000–2001.
- Dibutyl phthalate and PAHs (represented by benzo[b]fluoranthene) were detected in 100% of the dust samples. PCB-52 and bisphenol A were detected in 65% and 62% or dust samples, respectively.

- Dibutyl phthalate, PAHs, and bisphenol A were detected in more than 60% of hand wipe samples, while PCB-52 was detected in less than 10% of these samples.
- Dibutyl phthalate was detected in all of the indoor air samples and PCB-52 was detected in almost all (98%) of the samples. PAHs were detected in slightly less than half of the indoor air samples, while bisphenol A was detected in slightly more than half of the indoor air samples.

Measure S4: Pesticides used inside California schools by commercial applicators, 2002–2007

About the Measure: Measure S4 presents information about pesticides used inside California schools. The data for this measure come from the California Department of Pesticide Regulation, which collects data on all commercial pesticide application in California schools. The measure shows how the application amounts of different pesticide categories have changed over the years.

California Schools Pesticide Use Reporting Database

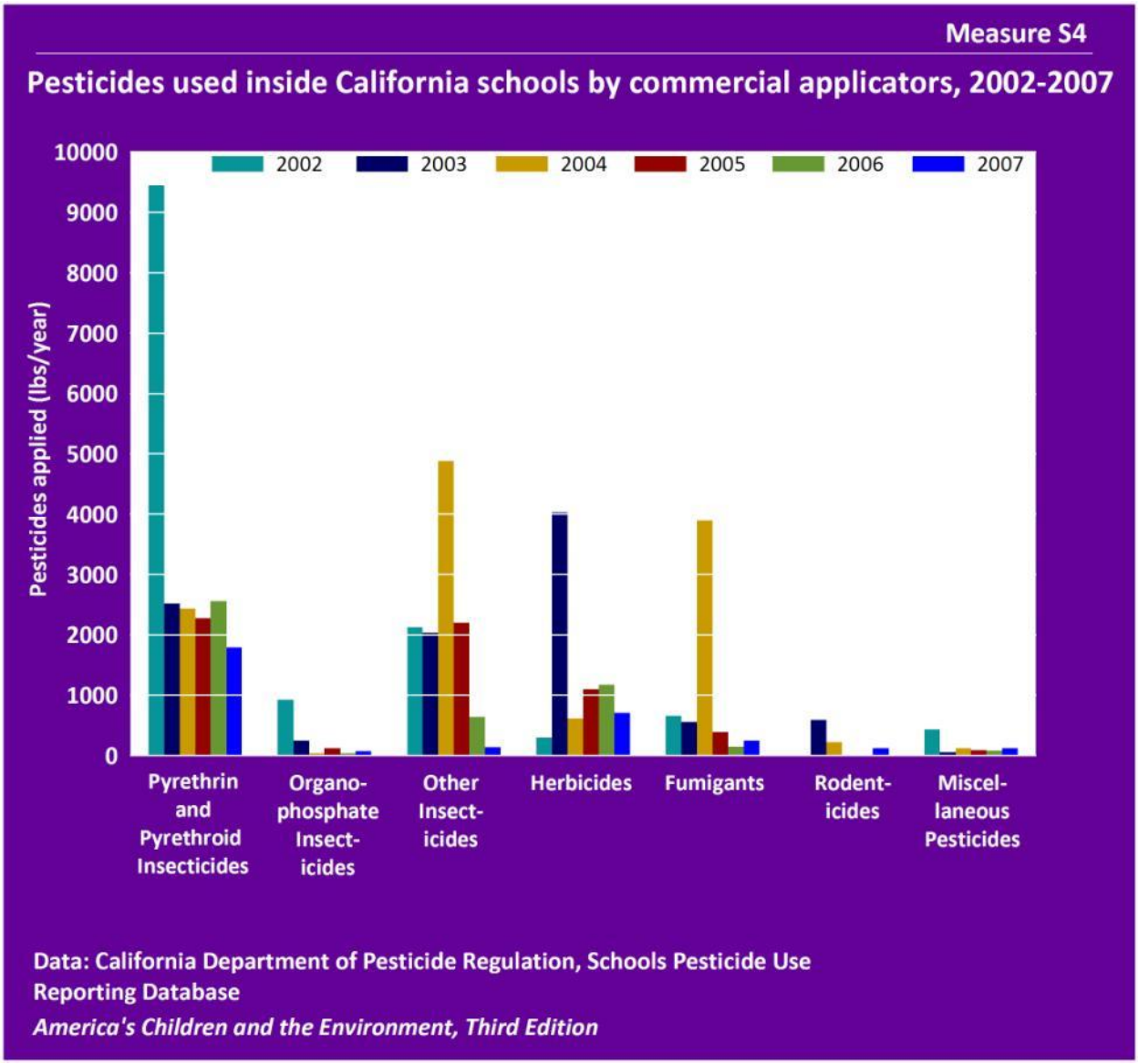
The California Department of Pesticide Regulation collects data on all commercial pesticide application in California schools. In the year 2000, California passed the Healthy Schools Act of 2000, which required all public child care facilities and school sites to report pesticide use on school sites by pest control businesses.[115] Schools are required to report pesticide use at least once per year, and all schools are required to maintain records of their reports on-site for four years. The California Healthy Schools Act requires reporting for application of pesticides to the buildings or structures (including attics and crawl spaces), playgrounds, athletic fields, school vehicles, or any other area of school property, indoors and outdoors, visited or used by pupils.[115] The law does not apply to products used as self-contained baits or traps; gels or pastes used as crack-and-crevice treatments; pesticides exempted from regulation by EPA; or antimicrobial pesticides, including sanitizers and disinfectants. All other pesticides must be reported.

Data Presented in the Measure

Measure S4 displays the annual amount (pounds per year) of pesticides used inside California schools and child care facilities by commercial applicators. The measure presents data for the indoor applications of pesticides for all years for which data are available: 2002–2007. Although the measure presents data for schools and child care facilities, nearly all of the data reported are from schools.

The measure presents the amount of pesticides applied in California schools and child care facilities, in pounds per year, with pesticides grouped into seven categories: pyrethrin and pyrethroid insecticides, organophosphate insecticides, other insecticides, herbicides, fumigants, rodenticides, and miscellaneous pesticides. Most use of the “other insecticides” category inside of California schools is accounted for by imidacloprid, which is marketed for indoor termite and cockroach control. Most of the “miscellaneous pesticides” category use inside of schools is accounted for by a borate compound used as a fungicide and insecticide.

Routinely collected pesticide use data can provide helpful information about the types of pesticides used and the extent of such use, including changes over time. However, these data do not indicate the extent of pesticide exposure experienced by children in California schools.



Data characterization

- Data for this measure are obtained from a reporting database maintained by the California Department of Pesticide Regulation.
- Reporting is required for all pesticide applications by pest control companies at all school and childcare facilities in California.
- Pesticide reports are submitted to the database at least annually and report all pesticide application on any area of school or childcare facility property visited or used by pupils.

- Pyrethrin and pyrethroid insecticides accounted for the greatest volume of pesticide use in California schools overall from 2002 to 2007, although there was greater use of herbicides in 2003, and of the "other" insecticides category and fumigants in 2004.

- The application of pyrethrin and pyrethroid insecticides, and organophosphate insecticides inside California schools has decreased since 2002.

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