

Third National Report on Human Exposure to Environmental Chemicals

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**Department of Health and Human Services
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The Centers for Disease Control and Prevention (CDC) protects people's health and safety by preventing and controlling diseases and injuries; enhances health decisions by providing credible information on critical health issues; and promotes healthy living through strong partnerships with local, national, and international organizations.

About CDC's Environmental Health Laboratory



Using advanced laboratory science and innovative techniques, scientists in CDC's Environmental Health Laboratory at the National Center of Environmental Health (NCEH) have helped change the face of environmental public health in this country. By recognizing chemicals that enter the body from environmental exposure, by responding to terrorism and public health emergencies involving chemicals, and by improving laboratory methods to measure chemical exposure, the laboratory has greatly enhanced the understanding of chemical exposure and related health effects for the nation and around the world.

For more than three decades, laboratory scientists at NCEH have been determining which environmental chemicals enter people's bodies, how much of those chemicals are actually present, and how the amounts of those chemicals may be related to health effects. The highly trained scientists measure levels of chemicals directly in people's blood or urine. Rather than predicting how much of a substance gets into people from estimates based on measurements in food, water, air, and other media, NCEH's laboratory scientists have taken out the guesswork by measuring low levels of chemicals that are actually in people's bodies. And they do so with precision, speed, and accuracy, measuring many chemicals in a very small amount—often a teaspoon or less—of blood or urine.

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Introduction

The *National Report on Human Exposure to Environmental Chemicals* provides an ongoing assessment of the exposure of the U.S. population to environmental chemicals using biomonitoring. The *Second National Report on Human Exposure to Environmental Chemicals (Second Report)* was released in 2003 and presented biomonitoring exposure data for 116 environmental chemicals for the civilian, noninstitutionalized U.S. population over the 2-year period 1999-2000. This *Third Report* presents similar exposure data for the U.S. population for 148 environmental chemicals over the period 2001-2002. The *Third Report* also includes the data from the *Second Report*.

Chemicals or their metabolites were measured in blood and urine samples from a random sample of participants from the National Health and Nutrition Examination Survey (NHANES) conducted by CDC's National Center for Health Statistics. NHANES is a series of surveys designed to collect data on the health and nutritional status of the U.S. population.

For this *Report*, an environmental chemical means a chemical compound or chemical element present in air, water, food, soil, dust or other environmental media (e.g., consumer products). Biomonitoring is the assessment of

human exposure to chemicals by measuring the chemicals or their metabolites in human specimens such as blood or urine. A metabolite is chemical alteration of the original compound produced by body tissues. Blood and urine levels reflect the amount of the chemical that actually gets into the body from the environment.

Table 1 lists the chemicals measured in the *Second* and *Third Reports* and the years these chemicals were measured.

The new chemicals for the *Third Report* are—

- Pyrethroid insecticides.
- Additional polycyclic aromatic hydrocarbons (including benzo-[a]-pyrene).
- Aldrin, endrin, dieldrin.
- Additional phthalate metabolites.
- Additional pesticides and herbicides.
- Additional dioxins, furans, and polychlorinated biphenyls (PCBs).

We have not performed any analyses for differences in results between the 1999-2000 and 2001-2002 survey periods. As additional *Reports* are released every 2 years, it will become possible to analyze trends. Details on data analysis are presented in the section titled "Data Sources and Analysis."

Table 1. Chemicals Measured in the *Second* and *Third Reports*

Chemical	1999-2000	2001-2002
Metals		
Antimony	•	•
Barium	•	•
Beryllium	•	•
Cadmium	•	•
Cesium	•	•
Cobalt	•	•
Lead	•	•
Mercury	•	•
Molybdenum	•	•
Platinum	•	•
Tungsten	•	•
Thallium	•	•
Uranium	•	•
Tobacco Smoke		
Cotinine	•	•
Phytoestrogens		
Daidzein	•	•
Enterodiol	•	•
Enterolactone	•	•
Equol	•	•
Genistein	•	•
O-Desmethylangolensin	•	•

Chemical	1999-2000	2001-2002
Polycyclic Aromatic Hydrocarbons		
1-Hydroxybenz[a]anthracene	•	•
3-Hydroxybenz[a]anthracene and 9-Hydroxybenz[a]anthracene	•	•
1-Hydroxybenzo[c]phenanthrene	•	•
2-Hydroxybenzo[c]phenanthrene	•	•
3-Hydroxybenzo[c]phenanthrene	•	•
1-Hydroxychrysene		•
2-Hydroxychrysene		•
3-Hydroxychrysene	•	•
4-Hydroxychrysene		•
6-Hydroxychrysene	•	•
3-Hydroxyfluoranthene	•	
2-Hydroxyfluorene	•	•
3-Hydroxyfluorene	•	•
9-Hydroxyfluorene		•
1-Hydroxyphenanthrene	•	•
2-Hydroxyphenanthrene	•	•
3-Hydroxyphenanthrene	•	•
4-Hydroxyphenanthrene		•
9-Hydroxyphenanthrene		•
1-Hydroxypyrene	•	•
3-Hydroxybenzo[a]pyrene		•
1-Hydroxynaphthalene		•
2-Hydroxynaphthalene		•

Chemical	1999-2000	2001-2002
Polychlorinated Dibenzo-p-dioxins, Dibenzofurans, Coplanar and Mono-Ortho-Substituted Biphenyls		
1,2,3,4,6,7,8,9-Octachlorodibenzo-p-dioxin (OCDD)	•	•
1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin (HpCDD)	•	•
1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin (HxCDD)	•	•
1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin (HxCDD)	•	•
1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin (HxCDD)	•	•
1,2,3,7,8-Pentachlorodibenzo-p-dioxin (PeCDD)	•	•
2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD)	•	•
1,2,3,4,6,7,8,9-Octachlorodibenzofuran (OCDF)	•	•
1,2,3,4,6,7,8-Heptachlorodibenzofuran (HpCDF)	•	•
1,2,3,4,7,8,9-Heptachlorodibenzofuran (HpCDF)	•	•
1,2,3,4,7,8-Hexachlorodibenzofuran (HxCDF)	•	•
1,2,3,6,7,8-Hexachlorodibenzofuran (HxCDF)	•	•
1,2,3,7,8,9-Hexachlorodibenzofuran (HxCDF)	•	•
1,2,3,7,8-Pentachlorodibenzofuran (PeCDF)	•	•
2,3,4,6,7,8-Hexachlorodibenzofuran (HxCDF)	•	•
2,3,4,7,8-Pentachlorodibenzofuran (PeCDF)	•	•
2,3,7,8-Tetrachlorodibenzofuran (TCDF)	•	•
2,4,4'-Trichlorobiphenyl (PCB 28)	•	•
2,3',4,4'-Tetrachlorobiphenyl (PCB 66)	•	•
2,4,4',5-Tetrachlorobiphenyl (PCB 74)	•	•
3,4,4',5-Tetrachlorobiphenyl (PCB 81)	•	•
2,3,3',4,4'-Pentachlorobiphenyl (PCB 105)	•	•
2,3',4,4',5-Pentachlorobiphenyl (PCB 118)	•	•
3,3',4,4',5-Pentachlorobiphenyl (PCB 126)	•	•
2,3,3',4,4',5-Hexachlorobiphenyl (PCB 156)	•	•
2,3,3',4,4',5'-Hexachlorobiphenyl (PCB 157)	•	•
2,3',4,4',5,5'-Hexachlorobiphenyl (PCB 167)	•	•
3,3',4,4',5,5'-Hexachlorobiphenyl (PCB 169)	•	•
2,3,3',4,4',5,5'-Heptachlorobiphenyl (PCB 189)	•	•
Non-dioxin-like Polychlorinated Biphenyls		
2,2',5,5'-Tetrachlorobiphenyl (PCB 52)	•	•
2,2',3,4,5'-Pentachlorobiphenyl (PCB 87)	•	•
2,2',4,4',5-Pentachlorobiphenyl (PCB 99)	•	•
2,2',4,5,5'-Pentachlorobiphenyl (PCB 101)	•	•
2,3,3',4',6-Pentachlorobiphenyl (PCB 110)	•	•
2,2',3,3',4,4'-Hexachlorobiphenyl (PCB 128)	•	•
2,2',3,4,4',5' and 2,3,3',4,4',6-Hexachlorobiphenyl (PCB 138&158)	•	•
2,2',3,4',5,5'-Hexachlorobiphenyl (PCB 146)	•	•
2,2',3,4',5,6'-Hexachlorobiphenyl (PCB 149)	•	•
2,2',3,5,5',6-Hexachlorobiphenyl (PCB 151)	•	•
2,2',4,4',5,5'-Hexachlorobiphenyl (PCB 153)	•	•
2,2',3,3',4,4',5-Heptachlorobiphenyl (PCB 170)	•	•
2,2',3,3',4,5,5'-Heptachlorobiphenyl (PCB 172)	•	•
2,2',3,3',4,5,6'-Heptachlorobiphenyl (PCB 177)	•	•
2,2',3,3',5,5',6-Heptachlorobiphenyl (PCB 178)	•	•
2,2',3,4,4',5,5'-Heptachlorobiphenyl (PCB 180)	•	•
2,2',3,4,4',5,6-Heptachlorobiphenyl (PCB 183)	•	•
2,2',3,4',5,5',6-Heptachlorobiphenyl (PCB 187)	•	•
2,2',3,3',4,4',5,5'-Octachlorobiphenyl (PCB 194)	•	•
2,2',3,3',4,4',5,5',6-Octachlorobiphenyl (PCB 195)	•	•
2,2',3,3',4,4',5,6' and 2,2',3,4,4',5,5',6-Octachlorobiphenyl (PCB196&203)	•	•
2,2',3,3',4,5,5',6-Octachlorobiphenyl (PCB 199)	•	•
2,2',3,3',4,4',5,5',6'-Nonachlorobiphenyl (PCB 206)	•	•
Other Pesticides		
N,N-Diethyl-3-methylbenzamide	•	•
ortho-Phenylphenol	•	•
2,5-Dichlorophenol	•	•
Carbamate Pesticides		
2-Isopropoxyphenol	•	•
Carbofuranphenol	•	•

Chemical	1999-2000	2001-2002
Phthalates		
Mono-methyl phthalate		•
Mono-ethyl phthalate	•	•
Mono-n-butyl phthalate	•	•
Mono-isobutyl phthalate		•
Mono-benzyl phthalate	•	•
Mono-cyclohexyl phthalate	•	•
Mono-2-ethylhexyl phthalate	•	•
Mono-(2-ethyl-5-oxohexyl) phthalate		•
Mono-(2-ethyl-5-hydroxyhexyl) phthalate		•
Mono-n-octyl phthalate	•	•
Mono-(3-carboxypropyl) phthalate		•
Mono-isononyl phthalate	•	•
Organochlorine Pesticides		
Hexachlorobenzene	•	•
Beta-hexachlorocyclohexane	•	•
Gamma-hexachlorocyclohexane	•	•
Pentachlorophenol	•	•
2,4,5-Trichlorophenol	•	•
2,4,6-Trichlorophenol	•	•
p,p'-DDT	•	•
p,p'-DDE	•	•
o,p'-DDT	•	•
Oxychlorane	•	•
trans-Nonachlor	•	•
Heptachlor epoxide	•	•
Mirex	•	•
Aldrin		•
Dieldrin		•
Endrin		•
Organophosphate Insecticides: Dialkyl Phosphate Metabolites		
Dimethylphosphate	•	•
Dimethylthiophosphate	•	•
Dimethyldithiophosphate	•	•
Diethylphosphate	•	•
Diethylthiophosphate	•	•
Diethyldithiophosphate	•	•
Organophosphate Insecticides: Specific Metabolites		
Malathion dicarboxylic acid	•	•
para-Nitrophenol	•	•
3,5,6-Trichloro-2-pyridinol	•	•
2-Isopropyl-4-methyl-6-hydroxypyrimidine	•	•
2-(Diethylamino)-6-methylpyrimidin-4-ol/one		•
3-Chloro-7-hydroxy-4-methyl-2H-chromen-2-one/ol		•
Herbicides		
2,4,5-Trichlorophenoxyacetic acid	•	•
2,4-Dichlorophenoxyacetic acid	•	•
2,4-Dichlorophenol	•	•
Alachlor mercapturate	•	•
Atrazine mercapturate	•	•
Acetochlor mercapturate		•
Metolachlor mercapturate		•
Pyrethroid Pesticides		
4-Fluoro-3-phenoxybenzoic acid		•
cis-3-(2,2-Dichlorovinyl)-2,2-dimethylcyclopropane carboxylic acid		•
trans-3-(2,2-Dichlorovinyl)-2,2-dimethylcyclopropane carboxylic acid		•
cis-3-(2,2-Dibromovinyl)-2,2-dimethylcyclopropane carboxylic acid		•
3-Phenoxybenzoic acid		•

Public Health Uses of the Report

The overall purpose of the *Report* is to provide unique exposure information to scientists, physicians, and health officials to help prevent disease that results from exposure to environmental chemicals. Specific public health uses of the exposure information in the *Third Report* are—

- To determine which chemicals get into Americans and at what concentrations.
- For chemicals with a known toxicity level, to determine the prevalence of people with levels above those toxicity levels.
- To establish reference ranges that can be used by physicians and scientists to determine whether a person or group has an unusually high exposure.
- To assess the effectiveness of public health efforts to reduce exposure of Americans to specific chemicals.
- To determine whether exposure levels are higher among minorities, children, women of childbearing age, or other potentially vulnerable groups.
- To track, over time, trends in levels of exposure of the population.
- To set priorities for research on human health effects.

Data Presented for Each Environmental Chemical

The Report presents tables of descriptive statistics on the distribution of blood or urine levels for each environmental chemical. Statistics include unadjusted geometric means and percentiles with confidence intervals.

Geometric means are calculated by taking the log of each concentration, then calculating the mean of those log values, and finally, taking the antilog of that mean (the calculation can be done using any log base, such as 10 or e). A geometric mean provides a better estimate of central tendency for data that are distributed with a long tail at the upper end of the distribution. This type of distribution is common when measuring environmental chemicals in blood or urine. The geometric mean is influenced less by high values than is the arithmetic mean.

Percentiles (50th, 75th, 90th, and 95th) are given to provide additional information about the shape of the distribution. In the *Third Report*, 10th and 25th percentiles are no longer included in order to provide adequate space in the tables to cover multiple years of data. Many of the tables in the *Report* are heavily filled with data. For tables with data entered for the 50th, 90th, and 95th

percentiles, figures showing these percentiles have also been included to help readers visualize the analytical results. Vertical lines above and below the point estimate of the percentile in these graphs represent the 95% confidence interval, which gives an estimate of uncertainty for that percentile. Percentile estimates for both survey periods are plotted.

For urine measurements, data are shown for the both the concentration in urine and the concentration corrected for urine-creatinine level. Serum measurements for chemicals that concentrate in lipid (e.g., dioxins, furans, PCBs, organochlorine pesticides) are presented per gram of total lipid in the serum and also per whole weight of serum.

General information is provided for each chemical that also aids the interpretation of levels.

A brief overview of information about each chemical is provided in the text to address common uses, sources of human exposure, disposition in the body, and known human health effects or major consistent effects in animals. Additionally, studies from other populations where blood and urine levels are available are presented for comparison.

The text also discusses briefly differences among demographic groups obtained by comparing the geometric means adjusted for the demographic covariates of age, gender, race/ethnicity, and when applicable, urinary creatinine, serum cotinine, or a lipid level. These adjusted geometric means are not shown in the tables. See the section titled “Data Sources and Data Analysis” for more details.

Interpreting Report Exposure Data: Important Factors

The survey design provides estimates for the U.S. population.

NHANES is designed to provide estimates for the civilian, noninstitutionalized U.S. population. The NHANES design does not select or exclude participants on the basis of their potential for low or high exposure to a chemical. The current design does not permit examination of exposure levels by locality, state, or region; seasons of the year; proximity to sources of exposure; or use of particular products. For example, it is not possible to extract a subset of the data and examine levels of blood lead that represent levels in a particular state’s population.

Data from earlier Reports are included in the Third Report.

The *Third Report* includes data from the *First* and *Second Reports* in the tables and charts. One exception is that 10th and 25th percentiles are no longer included in the *Report* because of space limitations in the tables. Each chemical has 50th, 75th, 90th, and 95th percentiles included in the tables along with the unadjusted geometric means and sample sizes for the survey periods (i.e., 1999-2000 and 2001-2002) for which that chemical was analyzed. Current plans are to release future *Reports* of the exposure of the U.S. population to cover 2-year periods (e.g., 2003-2004, 2005-2006, 2007-2008).

Statistical tests for significance of trends over time should await additional data from future Reports. More detailed research analyses of the data in the Report is encouraged.

We have not performed statistical tests for trends over time given that data are available only for the 1999-2000 and 2001-2002 survey periods. New data will be released for the U.S. population every 2 years, with the next release covering the survey period 2003-2004. With additional data points it will be possible to describe patterns over time and in some cases test for trends. We plan to investigate trends in future *Reports* for chemicals that have at least 3 survey periods

More in-depth statistical analysis, including additional covariates, interactions and predictive variables, are beyond the scope of this document. We hope that scientists will be stimulated to examine the data further through analysis of the raw data available at <http://www.cdc.gov/nchs/nhanes.htm>.

Research studies, separate from the Report, are required to determine which blood or urine levels are safe and which are associated with disease.

The measurement of an environmental chemical in a person's blood or urine does not by itself mean that the chemical causes disease. Advances in analytical methods allow us to measure low levels of environmental chemicals in people, but separate studies of varying exposure levels and health effects are needed to determine which blood or urine levels result in disease. These studies must also consider other factors such as duration of exposure. The *Third Report* does not present new data on health risks from different exposures.

For some environmental chemicals, such as lead, research studies have given us a good understanding of the health risks associated with different blood lead levels. However, for many environmental chemicals, we need more research to assess health risks from different blood or urine levels. The results shown in the *Third Report* should help prioritize and foster research on human health risks that result from exposure to environmental chemicals.

Not all the chemicals in the *Report* are measured in the same individuals. Therefore, it is not possible to determine how many of the 148 chemicals were found at detectable levels in a given person. As noted above, the presence of a chemical does not imply disease. The levels or concentrations of the chemical are more important determinants of the relation to disease, when established in appropriate research studies, than the detection or presence of a chemical.

For more information about exposure to environmental chemicals, see the section titled "Toxicology and Health-Risk Information," which includes Internet reference sites. Each environmental chemical can be searched in databases at these Web sites using its chemical name or the Chemical Abstract Service (CAS) number, which is provided in the *Third Report*. The Agency for Toxic Substances and Disease Registry's (ATSDR) Toxicological Profiles and ToxFAQs provide good summaries of toxicology information as well as answers to common questions about exposure and health effects.

Blood and urine levels of a chemical should not be confused with levels of the chemical in air, water, food, soil, or dust.

Concentrations of environmental chemicals in blood or urine are not the same as those in air, water, food, soil, or dust. For example, a chemical concentration of 10 µg/L in water does not produce a level of 10 µg/L in blood or urine. Blood or urine levels may reflect exposure from one or more sources, including air, water, food, soil, and dust.

Levels of a chemical in blood and urine are determined by how much of the chemical has entered the body through all routes of exposure, including ingestion, inhalation, or dermal absorption, and how the chemical is distributed in body tissues, transformed into metabolites, and eliminated from the body. Although the levels in the blood and urine are measures of the amount of a chemical that has entered the body by all routes of exposure, the blood or urine level alone does not determine which exposure source or which route of

exposure has occurred. Except for metals, most measurements in urine quantify chemical metabolites.

Biomonitoring Exposure Measurements

The blood and urine exposure measurements presented in the *Third Report* were made at CDC's Environmental Health Laboratory (Division of Laboratory Sciences, National Center for Environmental Health). The analytical methods used for measuring the environmental chemicals or their metabolites in blood and urine were based on isotope dilution mass spectrometry, inductively coupled plasma mass spectrometry, or graphite furnace atomic absorption spectrometry. References for the analytical methods used to measure the different chemicals are provided in Appendix B. Laboratory measurements undergo extensive quality control and quality assurance review, including tolerance limits for operational parameters, the measurement of quality control samples in each analytical run to detect unacceptable performance in accuracy or precision, and verification of traceable calibration materials.

For chemicals measured in urine, levels are presented two ways: per volume of urine and per gram of creatinine. Levels per gram of creatinine (i.e., creatinine-corrected) adjust for urine dilution. For example, if one person has consumed more fluids than another person, his or her urine output is likely higher and the urine more dilute than that of the latter person. Creatinine is excreted from the body at a relatively constant rate over time, so expressing the result per gram of creatinine helps adjust for the effects of urinary dilution. The range and mean of

creatinine levels were 2-650 mg/dL and 136.4 mg/dL in NHANES 1999-2000, and 5-774 mg/dL and 130.6 mg/dL in NHANES 2001-2002, respectively, results that are typical for the general U.S. population (see Barr et al., 2005). Creatinine corrects for urinary dilution in individual specimens, although this dilution variability has little effect on point estimates (e.g., means, percentiles). Interpretation of creatinine corrected results should also recognize that creatinine correction can also partially adjust for differences in lean body mass or renal function among persons.

For dioxins, furans, PCBs, and organochlorine pesticides, serum levels are presented per gram of total lipid and per whole weight of serum. These compounds are lipophilic and concentrate in the body's lipid stores, including the lipid in serum. Serum levels reported per gram of total lipid reflect the amount of these compounds that are stored in body fat. Serum levels per whole weight of serum are also included to facilitate comparison with studies investigating exposure to these chemicals that have published results in these units.

Units of measurement are important. Results are reported here using standard units, generally conforming to those most commonly used in biomonitoring measurements. Useful unit conversions are presented in Table 2.

Selection of Chemicals Included in the Report

Chemicals in the *Report* were selected on the basis of scientific data that suggested exposure in the U.S. population; the seriousness of health effects known or suspected to result from some levels of exposure; the need to assess the efficacy of public health actions to reduce exposure to a chemical; the availability of a biomonitoring analytical method with adequate accuracy, precision, sensitivity, specificity, and throughput; the availability of adequate blood or urine samples; and the incremental analytical cost to perform the biomonitoring analysis for the chemical. The availability of biomonitoring methods with adequate performance and acceptable cost was a major consideration.

In October 2002, CDC solicited nominations for candidate chemicals or categories of chemical to include in future *Reports* (*Federal Register*, Vol. 67, No. 194, October 7, 2002) and received nominations for hundreds of chemicals. Details on the prioritization process for scoring the nominated chemicals and the resulting scores are available at www.cdc.gov/exposurereport/chemical_nominations.htm.

Table 2. Units of Measurements and Conversions

Unit	Abbreviation	Value
liter	L	
deciliter	dL	10 ⁻¹ liters
milliliter	mL	10 ⁻³ liters
gram	g	
milligram	mg	10 ⁻³ grams
microgram	µg	10 ⁻⁶ grams
nanogram	ng	10 ⁻⁹ grams
picogram	pg	10 ⁻¹² grams
femtogram	fg	10 ⁻¹⁵ grams
parts-per-million	ppm	1 µg/g, or approximately 1 µg/mL or 1 mg/L
parts-per-billion	ppb	1 ng/g, or approximately 1 ng/mL or 1 µg/L
parts-per-trillion	ppt	1 pg/g, or approximately 1 pg/mL or 1 ng/L
parts-per-quadrillion	ppq	1 fg/g, or approximately 1 fg/mL or 1 pg/L

Data Sources and Data Analysis

The National Health and Nutrition Examination Survey (NHANES)

Biomonitoring measurements for the *Report* were made in samples from participants in NHANES. NHANES is a series of surveys conducted by CDC's National Center for Health Statistics (NCHS) that is designed to collect data on the health and nutritional status of the U.S. population. NHANES collects information about a wide range of health-related behaviors, performs a physical examination and collects samples for laboratory tests. NHANES is unique in its ability to examine public health issues in the U.S. population, such as risk factors for cardiovascular disease. Beginning in 1999, NHANES became a continuous survey, sampling the U.S. population annually and releasing the data in 2-year cycles. The sampling plan follows a complex, stratified, multistage, probability-cluster design to select a representative sample of the civilian, noninstitutionalized population in the United States.

The NHANES protocol includes a home interview followed by a standardized physical examination in a mobile examination center. As part of the examination component, blood is obtained by venipuncture for participants aged 1 year and older, and urine specimens are collected from people aged 6 years and older. Additional detailed information on the design and conduct of the NHANES survey is available at <http://www.cdc.gov/nchs/nhanes.htm>.

Environmental chemicals were measured in either blood or urine specimens collected as part of the examination component of NHANES. The age range for which a chemical was measured varied by chemical group. Most of the environmental chemicals were measured in randomly selected subsamples within specific age groups. Randomization of subsample selection is built into the NHANES design before sample collection begins. This subsampling was needed to ensure an adequate quantity of sample for analysis and to accommodate the throughput of the mass spectrometry analytical methods.

Age groups and sample sizes for each exposure measurement are provided in each of the tables of results. Blood lead and cadmium levels were measured in all people aged 1 year and older. Serum cotinine was measured in the entire NHANES sample for ages 3 years and older. Total blood mercury was measured in children aged 1-5 years and in women aged 16-49 years. Urine mercury was measured in women aged 16-49 years.

Metals, phthalates, polycyclic aromatic hydrocarbons (PAHs), and phytoestrogens were measured in urine from a random one-third subsample of people aged 6 years and older.

Urinary levels of herbicides, selected pesticides, and metabolites of organophosphate pesticides were measured in a random one-half subsample of children aged 6-11 years in 1999 and 2000, a random one-quarter subsample of people aged 12-59 years in 1999, and a random one-third subsample of people aged 12 years and older in 2000. These chemicals also were measured in a random one-third subsample of people aged 6 years and older in 2001 and 2002. Dioxins, furans, polychlorinated biphenyls (PCBs), and organochlorine pesticides were measured in serum from a random one-third subsample of people aged 12 years and older in 1999 and 2000. In 2001 and 2002, dioxins, furans, and coplanar PCBs were measured in a random one-third subsample of people aged 20 years and older and organochlorine pesticides and other PCBs were measured in a random one-third subsample of people aged 12 years and older.

Data Analysis

Because the NHANES sample design is complex, sample weights must be used to adjust for the unequal probability of selection into the survey. Sample weights also are used to adjust for possible bias resulting from nonresponse and are post-stratified to U.S. Census Bureau estimates of the U.S. population. Data were analyzed using the statistical software package Statistical Analysis System (SAS) (SAS Institute Inc., 2002) and the statistical software package SUDAAN (SUDAAN Release 8.0, 2001). SUDAAN uses sample weights and calculates variance estimates that account for the complex survey design.

Guidelines for the analysis of NHANES data are provided by NCHS at http://www.cdc.gov/nchs/data/nhanes/nhanes_general_guidelines_june_04.pdf. These guidelines note that the previous analysis of 1999-2000 data used a jackknife method (available within SUDAAN) for variance estimation that was based on replicate weights. To better address multiple 2-year data sets and combining 2-year data sets into 4-year data sets, NCHS developed a new approach based on masked variance units that uses a Taylor series (linearization) method that is also available in SUDAAN. More details on this approach are provided in the analytical guidelines.

In the *Third Report*, all variance estimates (both 1999-2000 and 2001-2002 data) were calculated using the Taylor series (linearization) method within SUDAAN. In the *Second Report*, 1999-2000 variance estimates were calculated using the jackknife method (See Appendix C for details). The two methods produce very similar, but not identical, variance estimates. Consequently, some confidence intervals for 1999-2000 presented in the *Second Report* will differ slightly from confidence intervals for the same time period presented in the *Third Report*.

Selected percentiles and unadjusted geometric means of analyte concentrations are presented in tables and charts. Percentile estimates were calculated using SAS Proc Univariate using weighted data. Results are shown for the total population and also by age group, gender, and race/ethnicity as defined in NHANES. For these analyses, race/ethnicity is categorized as Mexican American, non-Hispanic black, and non-Hispanic white. Other racial/ethnic groups are sampled, but the proportion of the total population represented by other racial/ethnic groups is not large enough to produce valid estimates. Other racial/ethnic groups are included in estimates that are based on the entire population sample. Age groups are shown for each chemical in the results table. Gender is coded as male or female.

In the text (not in the tables), results are presented of comparisons of geometric mean levels for different demographic groups using analysis of covariance (ANCOVA), which included as covariates age, gender, race/ethnicity, urine creatinine and serum cotinine, as appropriate. ANCOVA allows for comparison of geometric means of two demographic groups after adjusting for these covariates. For example, when comparing geometric mean blood lead levels for adolescents to those for adults, the ANCOVA would first adjust the geometric mean blood lead level for adolescents for gender, race/ethnicity, and serum cotinine and also the geometric mean blood lead level for adults for gender, race/ethnicity, and serum cotinine. The ANCOVA was performed using SUDAAN with a significance level for statistical testing of $\alpha = 0.025$. These analyses were conducted separately for each two year survey period and differences for each survey period were not statistically compared.

Urine creatinine is included as a continuous variable in the ANCOVA for chemicals measured in urine to adjust for urinary dilution. Cotinine is a major metabolite of nicotine and a good indicator of smoking status. Therefore, log cotinine is also included as a continuous variable in ANCOVA analyses of dioxins; furans; PCBs;

organochlorine pesticides; PAHs; and the metals (lead, cadmium, mercury, antimony, barium, molybdenum, thallium) to adjust for known or probable effects of smoking on the levels of these chemicals in blood or urine, including the contribution of chemicals contained in smoke and the effect of chemicals in smoke on the metabolism of other measured chemicals. The decision to adjust for log cotinine was determined by whether log cotinine was a significant predictor of the chemical's concentration and results of research that examined cotinine as a predictive variable.

Concentrations less than the limit of detection (LOD) were assigned a value equal to the LOD divided by the square root of 2 for calculation of geometric means. The LOD is the level at which the measurement has a 95% probability of being greater than zero (Taylor, 1987). Assigning a value of the LOD divided by 2 made little difference in geometric mean estimates. Percentile estimates that are less than the LOD for the chemical analysis are reported as "< LOD." If the proportion of results below the LOD was greater than 40%, geometric means were not calculated. Appendix A contains a table of LOD values for each chemical. For the same chemical, LOD values may change over time as a result of improvements to analytical methods. One possible consequence is that results may be reported as "< LOD" in the 1999-2000 data but be reported as a concentration value above the LOD in 2001-2002 because the analytical method had improved. Thus, for proper interpretation, the LOD values in the tables of descriptive statistics tables should be referenced to the LOD table in Appendix A.

For most chemicals, the LOD is constant for each sample analyzed. For dioxins, furans, PCBs, organochlorine pesticides, and a few other pesticides, each individual sample has its own LOD. These analyses have an individual LOD for each sample, mostly because the sample volume used for analysis differed for each sample. A higher sample volume results in a lower LOD (i.e., a better ability to detect low levels). For these chemicals, the maximum LOD value is provided in the LOD table in Appendix A. The maximum LOD was the highest LOD among all the individual samples analyzed. In general, the mean LOD was about 40-50% of the maximum LOD.

The same procedure for imputing values below the LOD in calculations of geometric means was used for chemicals with individual LODs for each sample. That is, concentrations less than the individual LOD were assigned a value equal to the individual LOD divided by the square root of 2. For chemicals that had individual

sample LODs, a conservative rule was used for reporting percentiles: if any individual sample LOD in the demographic group was above the percentile estimate, the percentile estimate was not reported.

For chemicals measured in urine, separate tables are presented for the chemical concentration expressed per volume of urine (uncorrected table) and the chemical concentration expressed per gram of creatinine (creatinine corrected table). Geometric mean and percentile calculations were performed separately for each of these concentrations. LOD calculations were performed using the chemical concentration expressed per volume of urine, because this concentration determines the analytical sensitivity. For this reason, LOD results for urine measurements in Appendix A are in weight per volume of urine. In the creatinine corrected tables, a result for a geometric mean or percentile was reported as < LOD if the corresponding geometric mean or percentile was < LOD in the uncorrected table. So for example, if the 50th percentile for males was < LOD in the uncorrected table, it would also be < LOD in the creatinine corrected table.

For chemicals measured in serum lipid, separate tables are presented for the chemical concentration expressed per volume of serum (lipid unadjusted table) and the chemical concentration expressed per amount of lipid (lipid adjusted table). Geometric mean and percentile calculations were performed separately for each of these concentrations. LOD calculations were performed using the chemical concentration expressed per amount of lipid, because this concentration determines the analytical sensitivity. For this reason, LOD results for chemicals measured in serum lipid in Appendix A are in weight per amount of lipid. In the lipid unadjusted tables, a result for a geometric mean or percentile was reported as < LOD if the corresponding geometric mean or percentile was < LOD in the lipid adjusted table.

Toxicology and Health-Risk Information

The *Third Report* presents new data on the exposure of the U.S. population to environmental chemicals. The measurement of an environmental chemical in a person's blood or urine does not by itself mean that the chemical causes disease. Advances in analytical methods allow us to measure lower and lower levels of environmental chemicals in people. Separate studies of varying exposure levels and health effects are required to determine which blood and urine levels are safe and which result in disease.

If available, generally recognized guidelines for blood or urine levels for each chemical are presented in the *Third Report* in the text discussion provided for each chemical or chemical group. These guidelines are usually from federal agencies. One exception is the American Conference of Governmental Industrial Hygienists (ACGIH), a private organization that publishes biological exposure indices (BEIs) which "generally indicate a concentration below which nearly all workers should not experience adverse health effects" (ACGIH, 2001). BEIs are blood or urine levels of a chemical that correspond to air-exposure limits for workers set by ACGIH. This organization notes that these values are for workers and that it is not appropriate to apply them to the general population. Information about the BEI level is provided here for comparison, not to imply that the BEI is a safety level for general population exposure. For most chemicals reported here, such guidelines are not available.

The *Report* also provides written information about each chemical or chemical group regarding uses, sources of human exposure, disposition in the body, and major effects, if known. In addition, selected studies have been provided, where possible, to permit comparison of blood and urine levels of a chemical in other population groups. Although the information in the text is provided as a brief overview for each chemical, it is not intended as a comprehensive review of each chemical. Generally, information was retrieved from major texts, consensus documents, and federal agency reviews, and then supplemented with published scientific investigations obtained by electronic searches in national and international databases.

Information Available on the Internet

Links to nonfederal organizations are provided solely as a service to our readers. These links do not constitute an endorsement of these organizations or their programs by CDC or the federal government. CDC is not responsible for the content of an individual organization's Web pages found at these links. For information about toxicology and health risks, see the following sites:

U.S. Government-Related Internet Links

Centers for Disease Control and Prevention (CDC) and Agency for Toxic Substances and Disease Registry (ATSDR)

- NIOSH Pocket Guide to Chemical Hazards: <http://www.cdc.gov/niosh/npg/npgd0000.html>
- Registry of Toxic Effects of Chemical Substances (RTECS): <http://www.cdc.gov/niosh/rtecs>
- Tobacco Information and Prevention Source: <http://www.cdc.gov/tobacco>
- National Center for Health Statistics: <http://www.cdc.gov/nchs>
- National Health and Nutrition Examination Survey: <http://www.cdc.gov/nchs/nhanes.htm>
- Childhood Lead Poisoning Prevention Program: <http://www.cdc.gov/nceh/lead/lead.htm>
- Pesticides and Public Health: Integrated Methods of Mosquito Management: <http://www.cdc.gov/ncidod/eid/vol7no1/rose.htm>
- National Institute for Occupational Safety and Health (NIOSH), Occupational Health and Safety Guidelines for Chemical Hazards: <http://www.cdc.gov/niosh/81-123.html>
- Agency for Toxic Substances and Disease Registry (ATSDR) Toxicological Profiles and ToxFAQs: <http://www.atsdr.cdc.gov/toxprofiles> or <http://www.atsdr.cdc.gov/toxfaq.html>

U.S. Department of Health and Human Services (U.S. DHHS)

- Environmental Health Policy Committee: <http://web.health.gov/environment>

U.S. Food and Drug Administration (U.S. FDA)

- Center for Devices and Radiological Health: <http://www.fda.gov/cdrh>
- Center for Food Safety and Applied Nutrition: <http://www.cfsan.fda.gov>
- National Center for Toxicological Research: <http://www.fda.gov/nctr>

National Institutes of Health (NIH)

- National Cancer Institute (NCI): <http://www.nci.nih.gov>
- National Institute of Child Health and Human Development (NICHD): <http://www.nichd.nih.gov>
- National Institute for Environmental Health Sciences (NIEHS): <http://www.niehs.nih.gov>
- National Toxicology Program (NTP) Chemical Health and Safety Data: <http://ntp.niehs.nih.gov/index.cfm?objectid=03610FA5-C828-304B-FE31F1182E8F764C>
- National Toxicology Program (NTP) Report on Carcinogens: <http://ntp.niehs.nih.gov/ntpweb/index.cfm?objectid=72016262-BDB7-CEBA-FA60E922B18C2540>
- Chemical Carcinogenesis Research Information System: <http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?CCRIS>
- Hazardous Substances Data Bank (HSDB®): <http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB>

U.S. Environmental Protection Agency (U.S. EPA)

- Office of Air and Radiation (OAR): <http://www.epa.gov/oar>
- Office of Environmental Information (OEI): <http://www.epa.gov/oei>
- Office of Prevention, Pesticides, and Toxic Substances (OPPTS): <http://www.epa.gov/opptsmt/index.htm>
- Office of Research and Development (ORD): <http://www.epa.gov/ORD>
- Office of Water (OW): <http://www.epa.gov/OW>
- Office of Pesticide Programs: <http://www.epa.gov/pesticides>
- EPA Integrated Risk-Information System (IRIS): <http://www.epa.gov/iris>
- EPA Envirofacts: http://www.epa.gov/enviro/index_java.html
- Lead: <http://www.epa.gov/OGWDW/dwh/cioc/lead.html>

U.S. Department of Agriculture (USDA)

- Food Safety and Inspection Service: <http://www.fsis.usda.gov>
- USDA, Forest Service Pesticide Fact Sheets: <http://www.fs.fed.us/foresthealth/pesticide>

U.S. Department of Energy (DOE)

- Office of Environment, Safety and Health: <http://tis.eh.doe.gov/portal/home.htm>

U.S. Department of Housing and Urban Development (HUD)

- Office of Healthy Homes and Lead-Hazard Control: <http://www.hud.gov/offices/lead>

U.S. Consumer Product Safety Commission (CPSC)

- <http://www.cpsc.gov>

U.S. Department of Transportation (DOT)

- Hazardous Materials Emergency-Response Guidebook: <http://hazmat.dot.gov/pubs/erg/gydebook.htm>

U.S. Department of Labor, Occupational Safety and Health Administration (OSHA)

- <http://www.osha.gov/index.html>

Other Related Internet Sites

- American College of Occupational and Environmental Medicine: <http://www.acoem.org>
- Association of Occupational and Environmental Clinics: <http://www.aoec.org>
- Association of Public Health Laboratories: <http://www.aphl.org>
- Chemfinder: <http://www.chemfinder.com>
- International Chemical Safety Cards: <http://www.ilo.org/public/english/protection/safework/cis/products/icsc/dtasht/index.htm>
- International Programme on Chemical Safety (IPCS): <http://www.who.int/pcs>
- Material Safety Data Sheets (MSDS): www.hazard.com/msds
- National Research Council (NRC) Toxicological Effects of Methylmercury: <http://books.nap.edu/books/0309071402/html/index.html>

Results by Chemical Group

Metals

Antimony

CAS No. 7440-36-0

General Information

In nature, antimony can be found in ores or other minerals, often combined with oxygen to form antimony trioxide. Elemental antimony can exist in one of four valences in its various chemical and physical forms: -3, 0, +3 and +5. Antimony is used in metal alloys, storage batteries, solder, sheet and pipe metal, ammunition, metal bearings, castings, and pewter. Antimony is used as a fire-retardant in textiles and plastics. It is also used in paints, ceramics, fireworks, enamels, and glass. Stibine is a metal hydride form of antimony used in the semiconductor industry. Two antimony compounds (sodium stibogluconate and antimony potassium tartrate) are used as antiparasitic medications.

Antimony enters the environment from natural sources and from its use in industry. People are exposed to

antimony primarily from food and to a lesser extent from air and drinking water. Workplace exposures occur as a result of breathing the air near industries such as smelters, coal-fired plants, and refuse incinerators that process or release antimony. Dermal contact with soil, water, or other substances containing antimony is another means of exposure.

The absorption, distribution, and excretion of antimony vary depending on its oxidation state. Urinary excretion appears to be greater for pentavalent antimony than for trivalent compounds (Elinder and Friberg, 1986). An elimination half-life of about 95 hours has been estimated after occupational exposures (Kentner et al., 1995).

Inorganic antimony salts irritate the mucous membranes, skin, and eyes. Acute inhalational exposure to antimony has been associated with irritation of the respiratory tract

Table 3. Antimony

Geometric mean and selected percentiles of urine concentrations (in µg/L) for the U.S. population aged 6 years and older, National Health and Nutrition Examination Survey, 1999-2002.

	Survey years	Geometric mean (95% conf. interval)	Selected percentiles (95% confidence interval)				Sample size
			50th	75th	90th	95th	
Total, age 6 and older	99-00	.132 (.120-.145)	.130 (.120-.140)	.210 (.200-.230)	.330 (.300-.350)	.420 (.390-.460)	2276
	01-02	.134 (.126-.142)	.130 (.120-.130)	.180 (.180-.190)	.260 (.240-.300)	.340 (.320-.390)	2690
Age group							
6-11 years	99-00	.176 (.154-.200)	.190 (.160-.200)	.260 (.230-.280)	.350 (.300-.390)	.400 (.320-.600)	316
	01-02	.146 (.134-.160)	.150 (.130-.160)	.200 (.180-.210)	.260 (.230-.310)	.330 (.280-.380)	368
12-19 years	99-00	.158 (.141-.178)	.170 (.150-.180)	.230 (.210-.270)	.340 (.290-.420)	.460 (.350-.510)	663
	01-02	.169 (.156-.184)	.150 (.140-.180)	.230 (.220-.250)	.350 (.320-.400)	.460 (.360-.480)	762
20 years and older	99-00	.123 (.112-.137)	.110 (.100-.120)	.190 (.180-.220)	.310 (.280-.330)	.420 (.390-.470)	1297
	01-02	.128 (.119-.136)	.120 (.120-.130)	.170 (.160-.190)	.240 (.220-.280)	.330 (.280-.380)	1560
Gender							
Males	99-00	.143 (.131-.157)	.140 (.130-.150)	.240 (.220-.250)	.350 (.320-.370)	.470 (.390-.570)	1132
	01-02	.145 (.136-.154)	.130 (.130-.150)	.200 (.180-.200)	.310 (.280-.330)	.390 (.350-.430)	1335
Females	99-00	.122 (.109-.137)	.120 (.100-.130)	.190 (.180-.220)	.300 (.270-.330)	.390 (.350-.460)	1144
	01-02	.125 (.117-.133)	.110 (.110-.120)	.180 (.160-.190)	.240 (.210-.260)	.310 (.260-.350)	1355
Race/ethnicity							
Mexican Americans	99-00	.132 (.108-.161)	.130 (.110-.160)	.200 (.180-.240)	.300 (.250-.370)	.410 (.330-.560)	787
	01-02	.142 (.130-.154)	.120 (.110-.130)	.200 (.160-.220)	.250 (.230-.300)	.360 (.300-.390)	683
Non-Hispanic blacks	99-00	.175 (.148-.207)	.180 (.150-.200)	.260 (.220-.290)	.390 (.310-.470)	.490 (.400-.620)	554
	01-02	.180 (.164-.197)	.160 (.150-.180)	.250 (.210-.280)	.350 (.320-.410)	.450 (.370-.530)	667
Non-Hispanic whites	99-00	.128 (.115-.144)	.120 (.110-.140)	.210 (.180-.220)	.320 (.280-.350)	.400 (.350-.500)	768
	01-02	.126 (.117-.135)	.120 (.120-.130)	.180 (.170-.180)	.240 (.220-.270)	.340 (.310-.390)	1132

and impaired pulmonary function (Renes, 1953). Pulmonary edema may occur in severe cases (Cordasco et al., 1973). Dysrhythmias and T-wave changes on electrocardiogram have also been noted in people after both therapeutic (Berman, 1988; Ming-Hsin et al., 1958) and occupational exposures (Briegner et al., 1954). Ingestion of antimony may cause people to experience a metallic taste, and gastrointestinal symptoms such as vomiting, diarrhea, abdominal pain, and ulcers (Werrin, 1962). The toxicity of stibine after acute inhalational exposure has been reported to be similar to that of arsine, resulting in hemolysis with abdominal and back pain (Dernehl et al., 1944).

Workplace standards for air exposure to antimony have been established by OSHA and ACGIH. Antimony trioxide is rated as being possibly carcinogenic to humans by IARC. Information about external exposure (i.e., environmental levels) and health effects is available from the U.S. EPA's IRIS Web site at <http://www.epa.gov/iris> and from ATSDR's Toxicological Profiles at

<http://www.atsdr.cdc.gov/toxprofiles>.

Interpreting Levels of Urinary Antimony Reported in the Tables

Urinary antimony levels were measured in a subsample of NHANES participants aged 6 years and older. Participants were selected within the specified age range to be a representative sample of the U.S. population. Previous studies reporting measurements in general populations (Minoia et al., 1990; Paschal et al., 1998) or compiled reference ranges (Hamilton et al., 1994) have found values slightly higher than those reported here, which may be due to methodologic, population, or exposure differences. Several investigations of airborne exposures to antimony in workers show urinary levels that are many times higher than those seen in this *Report*, even when exposure levels were below workplace air standards (Iavicoli et al., 2002; Kentner et al., 1995; Ludersdorf et al., 1987; Bailly et al., 1991).

Table 4. Antimony (creatinine corrected)

Geometric mean and selected percentiles of urine concentrations (in µg/g of creatinine) for the U.S. population aged 6 years and older, National Health and Nutrition Examination Survey, 1999-2002.

	Survey years	Geometric mean (95% conf. interval)	Selected percentiles (95% confidence interval)				Sample size
			50th	75th	90th	95th	
Total, age 6 and older	99-00	.124 (.108-.143)	.119 (.102-.143)	.185 (.163-.213)	.274 (.233-.333)	.382 (.333-.430)	2276
	01-02	.126 (.119-.134)	.120 (.115-.126)	.173 (.162-.188)	.265 (.242-.296)	.364 (.320-.414)	2689
Age group							
6-11 years	99-00	.191 (.147-.248)	.183 (.156-.220)	.250 (.196-.414)	.439 (.271-.741)	.537 (.333-1.30)	316
	01-02	.178 (.159-.200)	.173 (.150-.193)	.228 (.200-.272)	.338 (.265-.480)	.469 (.313-.727)	368
12-19 years	99-00	.121 (.104-.140)	.119 (.095-.146)	.176 (.146-.206)	.259 (.206-.310)	.310 (.228-.421)	663
	01-02	.121 (.112-.131)	.115 (.106-.127)	.159 (.138-.186)	.224 (.199-.245)	.266 (.244-.310)	762
20 years and older	99-00	.118 (.104-.135)	.111 (.096-.136)	.174 (.149-.209)	.263 (.227-.320)	.352 (.320-.391)	1297
	01-02	.122 (.115-.129)	.115 (.108-.121)	.167 (.153-.179)	.265 (.241-.296)	.364 (.318-.405)	1559
Gender							
Males	99-00	.112 (.099-.127)	.108 (.095-.127)	.164 (.146-.181)	.226 (.204-.268)	.319 (.235-.391)	1132
	01-02	.114 (.107-.123)	.108 (.103-.115)	.153 (.138-.171)	.228 (.205-.250)	.333 (.272-.421)	1334
Females	99-00	.137 (.117-.161)	.131 (.108-.164)	.212 (.176-.247)	.318 (.257-.400)	.425 (.357-.485)	1144
	01-02	.139 (.131-.148)	.132 (.124-.140)	.196 (.178-.211)	.295 (.267-.317)	.371 (.333-.444)	1355
Race/ethnicity							
Mexican Americans	99-00	.120 (.107-.135)	.114 (.105-.129)	.167 (.148-.203)	.249 (.207-.313)	.333 (.280-.357)	787
	01-02	.138 (.128-.149)	.129 (.117-.143)	.182 (.159-.203)	.269 (.229-.308)	.338 (.308-.429)	682
Non-Hispanic blacks	99-00	.114 (.099-.133)	.112 (.098-.130)	.163 (.144-.183)	.236 (.195-.338)	.339 (.255-.425)	554
	01-02	.123 (.113-.134)	.115 (.106-.126)	.163 (.150-.181)	.232 (.208-.267)	.300 (.248-.373)	667
Non-Hispanic whites	99-00	.129 (.109-.152)	.124 (.102-.152)	.195 (.167-.225)	.298 (.239-.352)	.400 (.333-.444)	768
	01-02	.127 (.117-.138)	.120 (.113-.130)	.176 (.159-.198)	.280 (.241-.317)	.380 (.318-.471)	1132

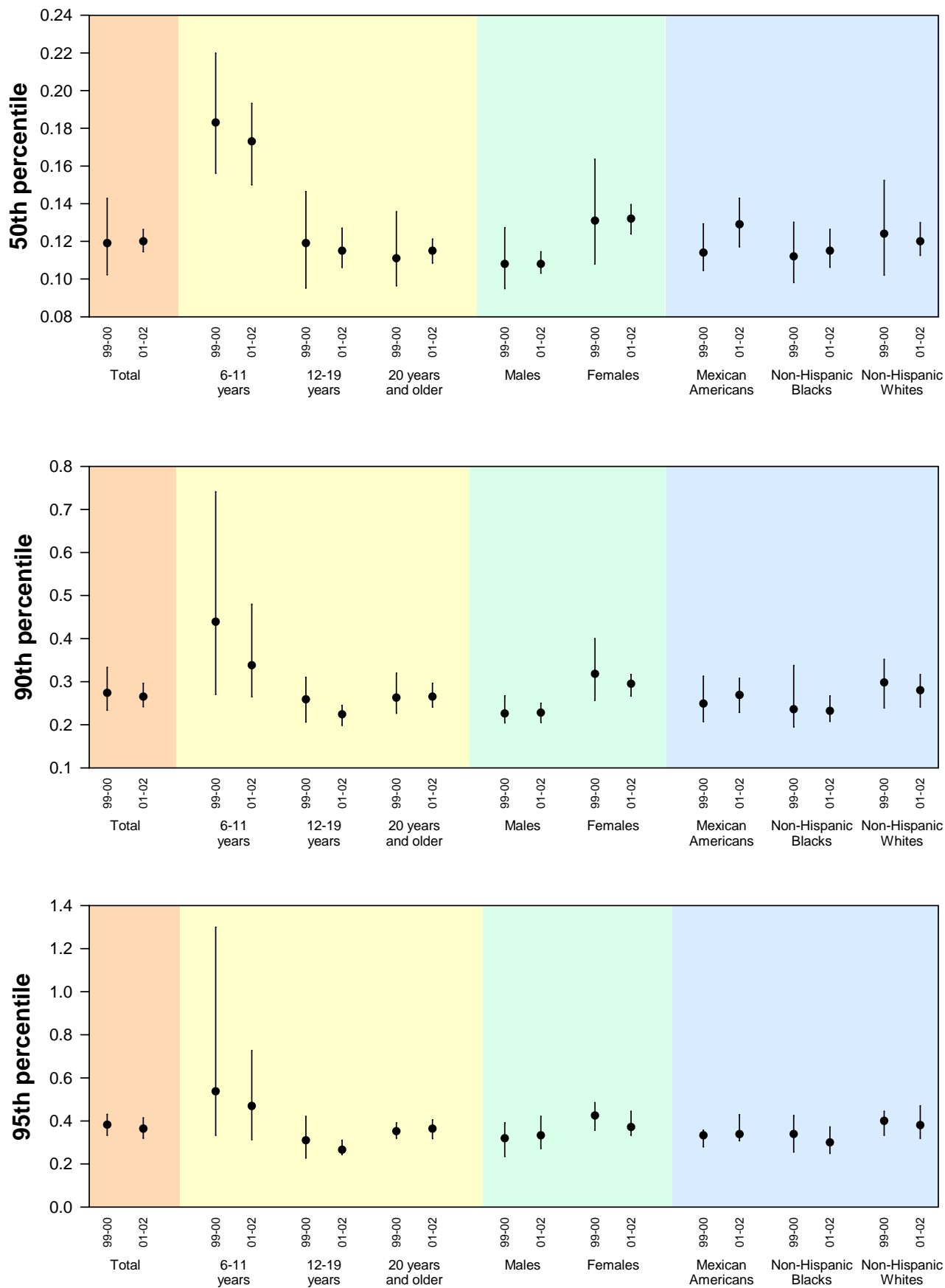
Comparing Adjusted Geometric Means

Geometric mean levels of urinary antimony for the demographic groups were compared after adjusting for the covariates of race/ethnicity, age, gender, log serum cotinine, and urinary creatinine (data not shown). In NHANES 2001-2002, adjusted geometric mean levels of urinary antimony were slightly higher for the group aged 6-11 years than for either groups aged 12-19 years or 20 years and older. The group aged 12-19 years had higher levels than the group aged 20 years and older. Mexican Americans had slightly higher levels than non-Hispanic whites. It is unknown whether these differences associated with age or race/ethnicity represent differences in exposure, pharmacokinetics, or the relationship of dose per body weight.

Finding a measurable amount of antimony in urine does not mean that the level of antimony causes an adverse health effect. Whether antimony at the levels reported here is a cause for health concern is not yet known; more research is needed. These urine antimony data provide physicians with a reference range so that they can determine whether or not people have been exposed to higher levels of antimony than are found in the general population. These data will also help scientists plan and conduct research about exposure to antimony and health effects.

Figure 1. Antimony (creatinine corrected)

Selected percentiles with 95% confidence intervals of urine concentrations (in $\mu\text{g/g}$ of creatinine) for the U.S. population aged 6 years and older, National Health and Nutrition Examination Survey, 1999-2002.



Barium

CAS No. 7440-39-3

General Information

Elemental barium is a silver-white metal. Barium's abundance in the earth's crust is approximately 0.05%. In nature, it combines with other chemicals such as sulfur or carbon and oxygen to form numerous barium salts. Of the various salts, approximately half are freely soluble in water, whereas the others are practically insoluble (notably barium sulfate and barium carbonate). Barium compounds are used by the oil and gas industries to make drilling muds. These compounds are also produced commercially for use in paint, bricks, tiles, glass, rubber, depilatories, fireworks, and ceramics. Medically, barium sulfate is used as a contrast medium for taking radiographs of the gastrointestinal tract. Barium salts are available for sale as rodenticides.

People can be exposed to barium in air, water, and food.

Small amounts of barium can be released into the air during mining and other industrial processes. Workers employed by industries that make or use barium compounds are exposed to barium dust. Certain foods, such as brazil nuts, are exceptionally high in barium (Genter, 2001).

The health effects of exposure to barium compounds depend on the dose, chemical form, water solubility, and route of exposure. Toxicity from soluble barium salts is rare but occurs after intentional or accidental ingestion of barium carbonate in rodenticides (Genter, 2001). Barium blocks cellular efflux of potassium resulting in extracellular profound hypokalemia. Symptoms include perioral paresthesias, vomiting, diarrhea, weakness, paralysis, hypertension, and cardiac dysrhythmias. The lethal dose of barium by ingestion is reported to be between 0.8-0.9 grams (Jourdan et al., 2001).

Table 5. Barium

Geometric mean and selected percentiles of urine concentrations (in µg/L) for the U.S. population aged 6 years and older, National Health and Nutrition Examination Survey, 1999-2002.

	Survey years	Geometric mean	Selected percentiles				Sample size
		(95% conf. interval)	(95% confidence interval)				
			50th	75th	90th	95th	
Total, age 6 and older	99-00	1.50 (1.35-1.66)	1.50 (1.30-1.70)	3.00 (2.70-3.40)	5.40 (4.60-6.10)	6.80 (6.20-8.40)	2180
	01-02	1.52 (1.41-1.65)	1.63 (1.50-1.75)	3.12 (2.76-3.51)	5.22 (4.73-5.74)	7.48 (6.54-8.12)	2690
Age group							
6-11 years	99-00	2.15 (1.70-2.72)	2.20 (1.80-2.30)	3.90 (2.60-6.10)	6.40 (5.20-8.30)	8.30 (5.00-76.2)	297
	01-02	1.80 (1.44-2.26)	2.08 (1.74-2.49)	3.62 (2.86-4.39)	5.37 (4.26-7.38)	6.88 (5.37-8.49)	368
12-19 years	99-00	1.97 (1.78-2.19)	2.00 (1.60-2.30)	3.50 (3.10-4.00)	5.90 (4.80-7.00)	9.70 (5.90-13.1)	621
	01-02	2.03 (1.76-2.34)	2.27 (1.96-2.53)	4.09 (3.48-4.72)	6.69 (5.55-7.87)	9.00 (7.25-11.4)	762
20 years and older	99-00	1.36 (1.24-1.51)	1.40 (1.30-1.70)	2.70 (2.50-3.00)	5.00 (4.20-5.50)	6.40 (5.70-8.30)	1262
	01-02	1.43 (1.32-1.54)	1.50 (1.39-1.65)	2.85 (2.55-3.26)	4.86 (4.53-5.47)	7.14 (6.08-8.12)	1560
Gender							
Males	99-00	1.70 (1.54-1.88)	1.80 (1.70-2.00)	3.10 (2.80-3.40)	5.50 (4.20-6.30)	7.50 (5.90-9.40)	1083
	01-02	1.64 (1.47-1.82)	1.80 (1.63-1.98)	3.15 (2.76-3.73)	5.52 (4.82-6.35)	7.87 (6.49-9.32)	1335
Females	99-00	1.33 (1.15-1.53)	1.50 (1.20-1.60)	2.80 (2.30-3.10)	5.10 (4.20-5.90)	6.80 (5.60-10.4)	1097
	01-02	1.43 (1.30-1.56)	1.43 (1.28-1.63)	3.10 (2.73-3.43)	4.86 (4.44-5.88)	7.15 (6.32-7.86)	1355
Race/ethnicity							
Mexican Americans	99-00	1.35 (1.25-1.46)	1.30 (1.20-1.50)	2.60 (2.30-2.90)	4.50 (4.00-5.10)	6.30 (5.50-6.80)	692
	01-02	1.21 (1.06-1.37)	1.24 (1.08-1.45)	2.55 (2.04-2.90)	4.31 (3.65-5.49)	6.08 (5.21-8.22)	683
Non-Hispanic blacks	99-00	1.34 (1.12-1.62)	1.30 (1.20-1.50)	2.50 (2.20-2.80)	5.10 (3.70-6.40)	7.40 (5.40-13.9)	540
	01-02	1.30 (1.14-1.48)	1.41 (1.22-1.62)	2.61 (2.31-2.82)	4.28 (3.70-5.18)	5.99 (4.87-7.26)	667
Non-Hispanic whites	99-00	1.56 (1.36-1.80)	1.70 (1.60-2.00)	3.30 (2.80-3.70)	5.40 (4.50-6.20)	7.20 (6.20-8.80)	765
	01-02	1.61 (1.46-1.77)	1.67 (1.53-1.82)	3.30 (2.86-3.73)	5.66 (4.94-6.30)	7.70 (6.61-8.49)	1132

Chronic accumulation of inhaled barium dust in the lung tissue may cause baritosis, a benign condition that may occur among barite ore miners. Chronic exposures to natural levels of barium in drinking water have not produced general health effects or evidence of cardiovascular risk (Brenniman and Levy, 1984; Wones et al., 1990). Workplace standards for external air exposure to various barium salts have been established by OSHA and a drinking water standard has been established by U.S. EPA. Barium is not rated for carcinogenicity. Information about external exposure and health effects is available from the U.S. EPA's IRIS Web site at <http://www.epa.gov/iris> and from ATSDR's Toxicological Profiles at <http://www.atsdr.cdc.gov/toxprofiles>.

Interpreting Levels of Urinary Barium Reported in the Tables

Urinary barium levels were measured in a subsample of NHANES participants aged 6 years and older. Participants were selected within the specified age range

to be a representative sample of the U.S. population. Previous studies reporting urinary levels of barium in general populations have found values generally similar to those documented in this *Report* (Minoia et al., 1990; Paschal et al., 1998). In addition, levels determined in clinically submitted specimens are broadly comparable (Komaromy-Hiller et al., 2000). Median urinary levels of barium found in welders of barium-containing electrodes were 60 times higher than the median levels in this *Report* (Zschesche et al., 1992) without obvious adverse effects. Urinary concentrations in acute poisonings are often hundreds to thousands times higher.

Comparing Adjusted Geometric Means

Geometric mean levels of urinary barium for the demographic groups were compared after adjusting for the covariates of race/ethnicity, age, gender, log serum cotinine and urinary creatinine (data not shown). In NHANES 2001-2002, adjusted geometric mean levels of urinary barium were higher for the group aged 6-11 years than either groups aged 12-19 years or aged 20 years and

Table 6. Barium (creatinine corrected)

Geometric mean and selected percentiles of urine concentrations (in µg/g of creatinine) for the U.S. population aged 6 years and older, National Health and Nutrition Examination Survey, 1999-2002.

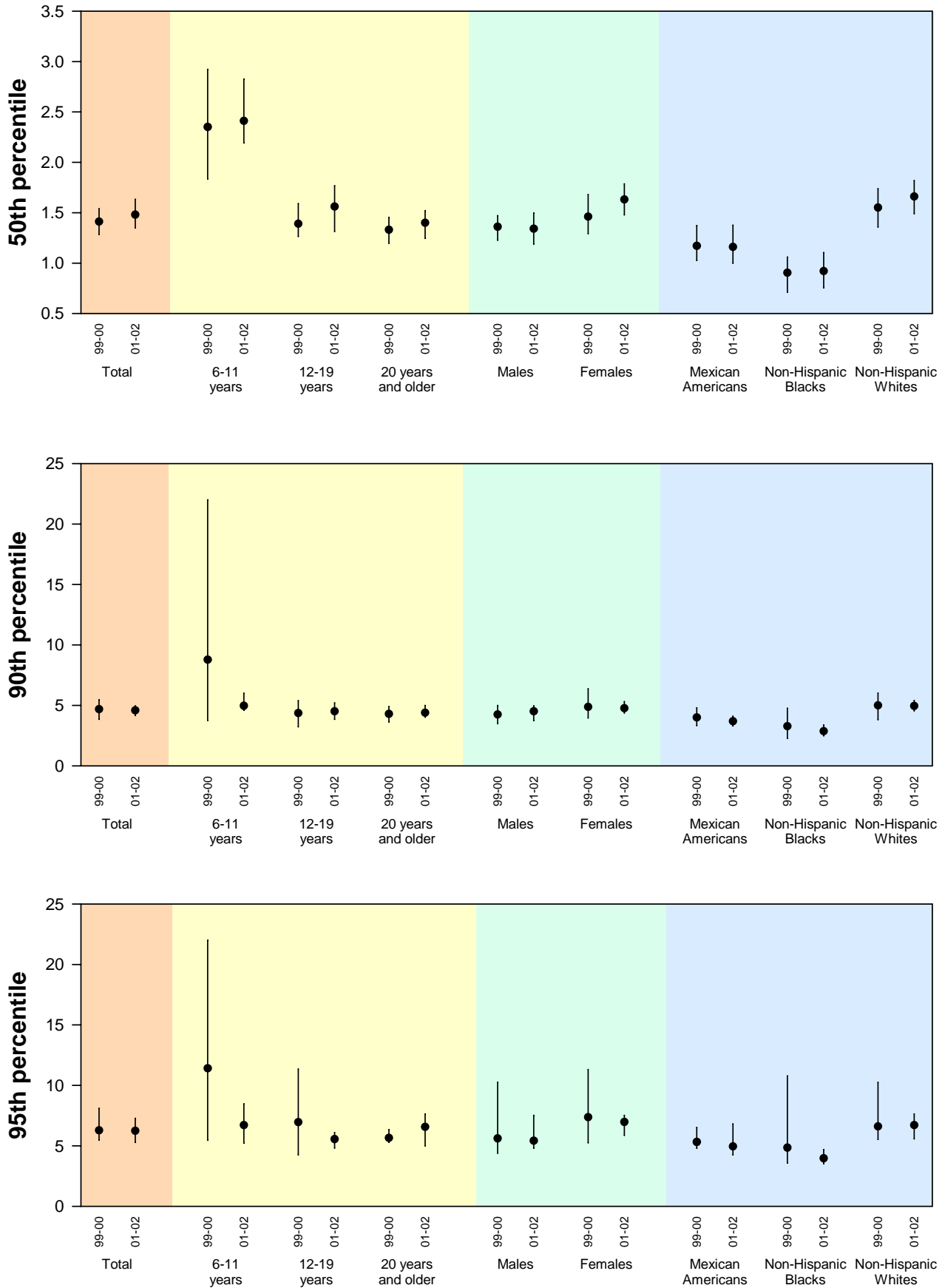
	Survey years	Geometric mean (95% conf. interval)	Selected percentiles (95% confidence interval)				Sample size
			50th	75th	90th	95th	
Total, age 6 and older	99-00	1.40 (1.26-1.56)	1.41 (1.28-1.54)	2.54 (2.18-2.89)	4.68 (3.85-5.47)	6.27 (5.47-8.09)	2180
	01-02	1.44 (1.31-1.58)	1.48 (1.35-1.63)	2.76 (2.51-3.03)	4.58 (4.14-4.95)	6.24 (5.28-7.27)	2689
Age group							
6-11 years	99-00	2.37 (1.68-3.32)	2.35 (1.83-2.92)	4.46 (2.55-6.46)	8.77 (3.75-22.0)	11.4 (5.45-22.0)	297
	01-02	2.20 (1.91-2.52)	2.41 (2.19-2.83)	3.91 (3.29-4.51)	4.96 (4.58-6.00)	6.71 (5.20-8.47)	368
12-19 years	99-00	1.51 (1.34-1.70)	1.39 (1.26-1.59)	2.48 (1.97-3.06)	4.36 (3.23-5.39)	6.95 (4.24-11.4)	621
	01-02	1.45 (1.33-1.59)	1.56 (1.31-1.77)	2.88 (2.68-3.12)	4.50 (3.84-5.20)	5.55 (4.81-6.10)	762
20 years and older	99-00	1.30 (1.19-1.42)	1.33 (1.20-1.45)	2.32 (2.08-2.62)	4.29 (3.62-4.91)	5.65 (5.28-6.33)	1262
	01-02	1.37 (1.24-1.50)	1.40 (1.24-1.52)	2.53 (2.23-2.84)	4.38 (4.02-5.00)	6.55 (5.00-7.64)	1559
Gender							
Males	99-00	1.32 (1.22-1.42)	1.36 (1.23-1.47)	2.39 (2.11-2.57)	4.24 (3.48-5.00)	5.61 (4.39-10.2)	1083
	01-02	1.30 (1.16-1.45)	1.34 (1.19-1.50)	2.46 (2.14-2.83)	4.50 (3.73-4.95)	5.42 (4.81-7.51)	1334
Females	99-00	1.49 (1.27-1.74)	1.46 (1.29-1.68)	2.65 (2.13-3.46)	4.86 (3.96-6.38)	7.36 (5.25-11.3)	1097
	01-02	1.59 (1.45-1.75)	1.63 (1.48-1.79)	2.98 (2.75-3.30)	4.76 (4.38-5.31)	6.97 (5.86-7.52)	1355
Race/ethnicity							
Mexican Americans	99-00	1.21 (1.10-1.33)	1.17 (1.03-1.37)	2.39 (2.10-2.59)	4.00 (3.33-4.80)	5.31 (4.80-6.51)	692
	01-02	1.18 (1.03-1.34)	1.16 (1.00-1.38)	2.33 (1.90-2.61)	3.68 (3.29-4.10)	4.95 (4.24-6.80)	682
Non-Hispanic blacks	99-00	.881 (.703-1.11)	.904 (.710-1.06)	1.64 (1.36-2.00)	3.27 (2.26-4.76)	4.84 (3.57-10.8)	540
	01-02	.891 (.777-1.02)	.920 (.754-1.11)	1.64 (1.44-2.03)	2.86 (2.48-3.37)	3.96 (3.52-4.68)	667
Non-Hispanic whites	99-00	1.56 (1.38-1.77)	1.55 (1.36-1.74)	2.72 (2.27-3.24)	5.00 (3.81-6.02)	6.60 (5.52-10.2)	765
	01-02	1.62 (1.49-1.76)	1.66 (1.49-1.82)	3.04 (2.76-3.32)	4.95 (4.55-5.41)	6.71 (5.57-7.64)	1132

older. Levels in the group aged 12-19 years were higher than the group aged 20 years and older. Levels in non-Hispanic whites were higher than in non-Hispanic blacks and Mexican Americans. It is unknown whether these differences associated with age or race/ethnicity represent differences in exposure, pharmacokinetics, or the relationship of dose per body weight.

Finding a measurable amount of barium in urine does not mean that the level of barium causes an adverse health effect. Whether barium at the levels reported here is a cause for health concern is not yet known; more research is needed. These urine barium data provide physicians with a reference range so that they can determine whether people have been exposed to higher levels of barium than are found in the general population. These data will also help scientists plan and conduct research about exposure to barium and health effects.

Figure 2. Barium (creatinine corrected)

Selected percentiles with 95% confidence intervals of urine concentrations (in $\mu\text{g/g}$ of creatinine) for the U.S. population aged 6 years and older, National Health and Nutrition Examination Survey, 1999-2002.



Beryllium

CAS No. 7440-41-7

General Information

Pure beryllium is a hard gray metal. The lightest of all metals, beryllium can be found in mineral rocks, coal, soil, and volcanic dust. Beryllium compounds are commercially mined, and the beryllium is refined for use in mirrors and in special metal alloys used in the automobile, computer, nuclear, electrical, aircraft, and machine-parts industries. Beryllium is also used in the production of sports equipment such as golf clubs and bike frames. In medicine, beryllium is used in instruments, x-ray machines, and dental bridges.

Exposure to beryllium occurs mostly in the workplace, near some hazardous waste sites, and from breathing tobacco smoke. Two types of minerals, bertrandite and beryl, are mined for commercial recovery of beryllium. In the workplace, beryllium dust enters the body

primarily through the lungs, where it remains for years, but there are little data available on how the metal accumulates in the lungs. Low-level beryllium exposure occurs through breathing air, eating food, or drinking water containing the metal. Small amounts of beryllium dust can enter air from burning coal and oil.

Beryllium may be harmful if inhaled. The effects depend on the concentration of beryllium in the inhaled air and the duration of air exposure. Air levels greater than 100 $\mu\text{g}/\text{m}^3$ can result in erythema and edema of the lung mucosa, producing pneumonitis. Chronic beryllium disease, or berylliosis, is a granulomatous interstitial lung disease that results from chronic beryllium inhalation and immunologic response. Skin contact with beryllium may also produce dermatitis, and some people demonstrate a hypersensitivity reaction to beryllium. Contact dermatitis and subcutaneous nodules have been reported with skin

Table 7. Beryllium

Geometric mean and selected percentiles of urine concentrations (in $\mu\text{g}/\text{L}$) for the U.S. population aged 6 years and older, National Health and Nutrition Examination Survey, 1999-2002.

	Survey years	Geometric mean (95% conf. interval)	Selected percentiles (95% confidence interval)				Sample size
			50th	75th	90th	95th	
Total, age 6 and older	99-00	*	< LOD	< LOD	< LOD	< LOD	2465
	01-02	*	< LOD	< LOD	< LOD	< LOD	2690
Age group							
6-11 years	99-00	*	< LOD	< LOD	< LOD	< LOD	340
	01-02	*	< LOD	< LOD	< LOD	< LOD	368
12-19 years	99-00	*	< LOD	< LOD	< LOD	< LOD	719
	01-02	*	< LOD	< LOD	< LOD	.140 (<LOD-.160)	762
20 years and older	99-00	*	< LOD	< LOD	< LOD	< LOD	1406
	01-02	*	< LOD	< LOD	< LOD	< LOD	1560
Gender							
Males	99-00	*	< LOD	< LOD	< LOD	< LOD	1227
	01-02	*	< LOD	< LOD	< LOD	.130 (<LOD-.150)	1335
Females	99-00	*	< LOD	< LOD	< LOD	< LOD	1238
	01-02	*	< LOD	< LOD	< LOD	< LOD	1355
Race/ethnicity							
Mexican Americans	99-00	*	< LOD	< LOD	< LOD	< LOD	884
	01-02	*	< LOD	< LOD	< LOD	< LOD	683
Non-Hispanic blacks	99-00	*	< LOD	< LOD	< LOD	< LOD	568
	01-02	*	< LOD	< LOD	< LOD	< LOD	667
Non-Hispanic whites	99-00	*	< LOD	< LOD	< LOD	< LOD	822
	01-02	*	< LOD	< LOD	< LOD	< LOD	1132

< LOD means less than the limit of detection, which may vary for some chemicals by year and by individual sample. See Appendix A for LODs.

* Not calculated. Proportion of results below limit of detection was too high to provide a valid result.

exposure to beryllium.

Workplace air standards for external exposure have been established by OSHA and ACGIH. NTP considers beryllium to be a known carcinogen. IARC states that beryllium is an animal carcinogen, and sufficient evidence exists to consider beryllium and beryllium compounds as carcinogenic in people, causing lung and central nervous system cancers. More information about external exposure (i.e., environmental levels) and health effects is available from the U.S. EPA's IRIS Web site at <http://www.epa.gov/iris> and from ATSDR's Toxicological Profiles at <http://www.atsdr.cdc.gov/toxprofiles>.

Interpreting Levels of Urinary Beryllium Reported in the Tables

Urinary beryllium levels were measured in a subsample of NHANES participants aged 6 years old and older. Participants were selected within the specified age range

to be a representative sample of the U.S. population. Comparable to the 1999-2000 subsample analysis, levels of beryllium were mostly undetectable. Previous studies have reported urinary levels for general populations as either undetectable concentrations or have not had comparable detection limits (Komaromy-Hiller et al., 2000; Minoia et al., 1990; Paschal et al., 1998). A summary of reference ranges taken from previous studies suggested that a true reference range for urinary beryllium is below the detection limits in past applications (less than 1 µg/L) (Hamilton et al., 1994). Apostoli and Schaller (2001) suggest that previous detection limits are inadequate to quantitate normal human exposure. In that study, urinary beryllium in workers correlated with air exposure measures, and when air levels were below the recommended threshold limit value, urinary beryllium concentrations ranged from 0.12 to 0.15 µg/L. The 95th percentiles in this *Report* for people aged 12-19 years and for males (0.14 µg/L and 0.13 µg/L, respectively) are similar to those values reported by Apostoli and Schaller (2001). Because the

Table 8. Beryllium (creatinine corrected)

Geometric mean and selected percentiles of urine concentrations (in µg/g of creatinine) for the U.S. population aged 6 years and older, National Health and Nutrition Examination Survey, 1999-2002.

	Survey years	Geometric mean (95% conf. interval)	Selected percentiles (95% confidence interval)				Sample size
			50th	75th	90th	95th	
Total, age 6 and older	99-00	*	< LOD	< LOD	< LOD	< LOD	2465
	01-02	*	< LOD	< LOD	< LOD	< LOD	2689
Age group							
6-11 years	99-00	*	< LOD	< LOD	< LOD	< LOD	340
	01-02	*	< LOD	< LOD	< LOD	< LOD	368
12-19 years	99-00	*	< LOD	< LOD	< LOD	< LOD	719
	01-02	*	< LOD	< LOD	< LOD	.231 (.173-.273)	762
20 years and older	99-00	*	< LOD	< LOD	< LOD	< LOD	1406
	01-02	*	< LOD	< LOD	< LOD	< LOD	1559
Gender							
Males	99-00	*	< LOD	< LOD	< LOD	< LOD	1227
	01-02	*	< LOD	< LOD	< LOD	.281 (.237-.333)	1334
Females	99-00	*	< LOD	< LOD	< LOD	< LOD	1238
	01-02	*	< LOD	< LOD	< LOD	< LOD	1355
Race/ethnicity							
Mexican Americans	99-00	*	< LOD	< LOD	< LOD	< LOD	884
	01-02	*	< LOD	< LOD	< LOD	< LOD	682
Non-Hispanic blacks	99-00	*	< LOD	< LOD	< LOD	< LOD	568
	01-02	*	< LOD	< LOD	< LOD	< LOD	667
Non-Hispanic whites	99-00	*	< LOD	< LOD	< LOD	< LOD	822
	01-02	*	< LOD	< LOD	< LOD	< LOD	1132

< LOD means less than the limit of detection, which may vary for some chemicals by year and by individual sample. See Appendix A for LODs.

* Not calculated. Proportion of results below limit of detection was too high to provide a valid result.

detection limit documented in this *Report* was 0.13 µg/L and because most of the samples were undetectable, these NHANES 1999-2002 levels are likely to be lower than levels considered safe for workers.

Finding a measurable amount of beryllium in urine does not mean that the level of beryllium causes an adverse health effect. Whether beryllium at the levels reported here is a cause for health concern is not yet known; more research is needed. These urinary beryllium data provide physicians with a reference range so that they can determine whether or not people have been exposed to higher levels of beryllium than are found in the general population. These data will also help scientists plan and conduct research about exposure to beryllium and health effects.

Cadmium

CAS No. 7440-43-9

General Information

Cadmium is a soft, malleable, bluish-white metal that is obtained chiefly as a by-product during the processing of zinc-containing ores (principally sphalerite, as zinc sulfide) and to a lesser extent during the refining of lead and copper from sulfide ore. The predominant commercial use of cadmium is in the manufacture of batteries (78% of uses). The use of cadmium in pigments accounts for 12% of consumption; in coatings and plating another 8% is used, and the remainder is used in stabilizers for plastic (1.5%), and nonferrous alloys and other uses (0.5%). From 2001 through 2004, the commercial use of cadmium declined approximately 70% in response to environmental concerns (U.S. Geological

Survey, 2004). Emissions of cadmium into the environment occur mainly via anthropogenic activities, such as secondary lead smelting, primary copper smelting, primary lead production, hazardous and municipal waste incineration, and petroleum refining (U.S. EPA, 1992).

Cadmium is absorbed via inhalation and ingestion. Inhalation of cigarette smoke is a predominant source in smokers. For nonsmokers who are not exposed to cadmium in the workplace, food is the largest source of cadmium intake and absorption. An analysis of food-intake rates and food-cadmium concentrations for the U.S. population recently estimated a geometric mean daily cadmium intake of 18.9 µg/day, or 0.4 µg/kg/day

Table 9. Cadmium in blood

Geometric mean and selected percentiles of blood concentrations (in µg/L) for the U.S. population aged 1 year and older, National Health and Nutrition Examination Survey, 1999-2002.

	Survey years	Geometric mean (95% conf. interval)	Selected percentiles (95% confidence interval)				Sample size
			50th	75th	90th	95th	
Total, age 1 and older	99-00	.412 (.378-.449)	.300 (.300-.400)	.600 (.500-.600)	1.00 (.900-1.00)	1.30 (1.20-1.40)	7970
	01-02	*	.300 (<LOD-.300)	.400 (.400-.500)	.900 (.900-1.10)	1.30 (1.20-1.60)	8945
Age group							
1-5 years	99-00	*	< LOD	.300 (<LOD-.300)	.400 (.300-.400)	.400 (.300-.400)	723
	01-02	*	< LOD	< LOD	< LOD	.300 (<LOD-.300)	898
6-11 years	99-00	*	< LOD	.300 (<LOD-.300)	.400 (.300-.400)	.400 (.400-.500)	905
	01-02	*	< LOD	< LOD	< LOD	.400 (.300-.400)	1044
12-19 years	99-00	.333 (.304-.366)	.300 (<LOD-.300)	.300 (.300-.400)	.800 (.600-.900)	1.10 (.900-1.10)	2135
	01-02	*	< LOD	.300 (<LOD-.300)	.400 (.400-.500)	.800 (.600-1.10)	2231
20 years and older	99-00	.468 (.426-.513)	.400 (.300-.400)	.600 (.600-.700)	1.00 (1.00-1.10)	1.50 (1.40-1.60)	4207
	01-02	*	.300 (.300-.400)	.600 (.500-.600)	1.10 (.900-1.20)	1.60 (1.30-1.80)	4772
Gender							
Males	99-00	.403 (.368-.441)	.400 (.300-.400)	.600 (.500-.600)	1.00 (.900-1.10)	1.30 (1.20-1.50)	3913
	01-02	*	.300 (<LOD-.300)	.400 (.400-.500)	.900 (.900-1.10)	1.40 (1.20-1.80)	4339
Females	99-00	.421 (.386-.460)	.300 (.300-.400)	.600 (.500-.600)	1.00 (.800-1.00)	1.30 (1.10-1.40)	4057
	01-02	*	.300 (.300-.400)	.500 (.500-.600)	1.00 (.900-1.10)	1.40 (1.20-1.60)	4606
Race/ethnicity							
Mexican Americans	99-00	.395 (.367-.424)	.400 (.300-.400)	.400 (.400-.500)	.700 (.700-.900)	1.10 (.900-1.30)	2742
	01-02	*	< LOD	.300 (.300-.400)	.600 (.500-.700)	1.00 (.700-1.30)	2268
Non-Hispanic blacks	99-00	.393 (.361-.427)	.300 (.300-.400)	.600 (.500-.600)	1.00 (.800-1.10)	1.40 (1.10-1.50)	1842
	01-02	*	< LOD	.400 (.400-.500)	1.00 (.900-1.00)	1.40 (1.20-1.50)	2219
Non-Hispanic whites	99-00	.420 (.376-.470)	.400 (.300-.400)	.500 (.500-.600)	1.00 (.900-1.10)	1.30 (1.20-1.40)	2716
	01-02	*	< LOD	.500 (.500-.600)	.900 (.900-1.10)	1.40 (1.20-1.80)	3806

< LOD means less than the limit of detection, which may vary for some chemicals by year and by individual sample. See Appendix A for LODs.

* Not calculated. Proportion of results below limit of detection was too high to provide a valid result.

(Choudhury et al., 2001). Although several studies have found that the average gastrointestinal absorption of dietary cadmium is on the order of 5% (Diamond et al., 2003), two balance studies have suggested that this value may be five- to ten-fold greater in young women than in the general population (Kikuchi et al., 2003; Horiguchi et al., 2004a). With chronic exposure, cadmium accumulates in the liver and the kidney, with one-third to one half of the total amount accumulating in the kidney (Nordberg and Nordberg, 2001). In both organs, cadmium tightly binds to metallothionein, an inducible metal-binding protein that provides protection against many of cadmium's toxic effects (Klaasen et al., 1999). The estimated half-life of cadmium in the kidney is one to four decades (ATSDR, 1999; Diamond et al., 2003).

The kidney is a critical target for cadmium. Renal tubular damage and glomerular damage can be caused by high-dose chronic exposure, which may occur in people who are occupationally exposed, and is manifested by irreversible proteinuria and progressive reductions in

glomerular filtration rate (Roels et al., 1999). Increased urinary excretion of calcium and phosphorus and decreased hydroxylation of vitamin D metabolites that accompany advanced tubular damage may result in overt, and often painful, osteomalacia or osteoporosis, typified by a condition known as "Itai-Itai disease" that afflicted women living in a cadmium-polluted region of Japan. Several recent epidemiological investigations in Belgium (Staessen et al., 1996; Hotz et al., 1999; Staessen et al., 1999), Sweden (Jarup et al., 2000; Alfvén et al., 2002; Olsson et al., 2002), Japan (Suwazono et al., 2000; Ezaki et al., 2003; Horiguchi et al., 2004b) China (Jin et al., 2004), and the United States (Noonan et al., 2002) have detected an association between relatively low-level environmental cadmium exposure and biomarkers of renal dysfunction or diminished bone mineral density. Environmental exposure from cadmium pollution has been linked to an increased rate of end-stage renal disease in a Swedish population residing in the vicinity of two battery manufacturing plants (Hellstrom et al., 2001). Although all the mechanisms of cadmium toxicity

Table 10. Cadmium in urine

Geometric mean and selected percentiles of urine concentrations (in µg/L) for the U.S. population aged 6 years and older, National Health and Nutrition Examination Survey, 1999-2002.

	Survey years	Geometric mean	Selected percentiles				Sample size
		(95% conf. interval)	(95% confidence interval)				
			50th	75th	90th	95th	
Total, age 6 and older	99-00	.193 (.169-.220)	.232 (.214-.249)	.475 (.436-.519)	.858 (.763-.980)	1.20 (1.06-1.33)	2257
	01-02	.210 (.189-.235)	.229 (.207-.255)	.458 (.423-.482)	.839 (.753-.919)	1.20 (1.07-1.28)	2690
Age group							
6-11 years	99-00	*	.078 (.061-.101)	.141 (.115-.173)	.219 (.178-.233)	.279 (.211-.507)	310
	01-02	.061 (<LOD-.081)	.077 (.067-.092)	.140 (.112-.160)	.219 (.184-.262)	.282 (.260-.326)	368
12-19 years	99-00	.092 (.067-.126)	.128 (.107-.148)	.202 (.183-.232)	.329 (.272-.372)	.424 (.366-.596)	648
	01-02	.109 (.087-.136)	.135 (.114-.157)	.210 (.189-.247)	.327 (.289-.366)	.442 (.366-.480)	762
20 years and older	99-00	.281 (.253-.313)	.306 (.261-.339)	.551 (.510-.621)	.979 (.836-1.13)	1.31 (1.13-1.57)	1299
	01-02	.273 (.249-.299)	.280 (.261-.308)	.545 (.493-.607)	.955 (.855-1.06)	1.28 (1.20-1.43)	1560
Gender							
Males	99-00	.199 (.165-.241)	.227 (.193-.263)	.462 (.381-.539)	.892 (.748-1.15)	1.41 (.980-1.83)	1121
	01-02	.201 (.177-.229)	.223 (.191-.257)	.445 (.393-.481)	.870 (.741-1.03)	1.22 (1.12-1.38)	1335
Females	99-00	.187 (.153-.229)	.239 (.220-.255)	.492 (.456-.540)	.806 (.705-.980)	1.10 (1.01-1.19)	1136
	01-02	.219 (.192-.251)	.234 (.202-.265)	.466 (.433-.519)	.817 (.733-.886)	1.17 (.918-1.36)	1355
Race/ethnicity							
Mexican Americans	99-00	.191 (.157-.233)	.202 (.167-.221)	.438 (.351-.551)	.813 (.686-.977)	1.12 (.886-1.38)	780
	01-02	.160 (.135-.189)	.181 (.171-.198)	.321 (.285-.362)	.559 (.430-.733)	.766 (.633-1.15)	683
Non-Hispanic blacks	99-00	.283 (.208-.387)	.312 (.243-.412)	.633 (.498-.806)	1.22 (.892-1.38)	1.48 (1.30-1.72)	546
	01-02	.277 (.229-.336)	.302 (.257-.354)	.580 (.476-.713)	1.04 (.843-1.38)	1.51 (1.28-1.74)	667
Non-Hispanic whites	99-00	.175 (.148-.206)	.220 (.194-.246)	.455 (.388-.510)	.797 (.714-1.01)	1.17 (.963-1.47)	760
	01-02	.204 (.179-.231)	.221 (.191-.255)	.445 (.394-.479)	.813 (.717-.875)	1.17 (.989-1.24)	1132

* Not calculated. Proportion of results below limit of detection was too high to provide a valid result.

are uncertain, key factors may involve perturbation of zinc-dependent biochemical processes, induction of oxidative stress, aberrant gene expression, estrogenic effects (Johnson et al., 2003), or altered cell signaling and steroidogenesis (Misra et al., 2003; Waalkes, 2003; Henson and Chedrese, 2004).

Acute and heavy airborne exposure to dusts and fumes, as may occur from welding on cadmium-alloyed metals, may result in severe, potentially fatal pneumonitis (Fernandez et al., 1996). Chronic inhalation exposure to cadmium particulate has been associated with changes in pulmonary function and chest radiography consistent with emphysema (Davison et al., 1988). Among U.S. adult smokers and former smokers studied in NHANES III, increases in urinary cadmium were associated with obstructive changes in pulmonary function (Mannino et al., 2004). Workplace exposure to airborne cadmium particulate has been associated with decrements in olfactory function (Mascagni et al., 2003). Animal studies have demonstrated reproductive and teratogenic

effects. Two recent small epidemiologic studies have noted a positive association of environmental cadmium levels in maternal urine or blood with gestational age (Nishijo et al., 2002) and birth height (Zhang et al., 2004). NTP has determined that cadmium is a known human carcinogen. Potential modes of action have recently been reviewed (Waalkes, 2003). Information about external exposure (i.e., environmental levels) and health effects is available from the U.S. EPA's IRIS Web site at <http://www.epa.gov/iris> and from ATSDR's Toxicological Profiles at <http://www.atsdr.cdc.gov/toxprofiles>.

Interpreting Levels of Cadmium in Blood and Urine Reported in the Tables

In the NHANES 2001-2002 sample, blood cadmium levels were measured in all participants aged 1 year and older, and urine cadmium levels were measured in a sample of people aged 6 years and older. Participants were selected to be a representative sample of the U.S.

Table 11. Cadmium in urine (creatinine corrected)

Geometric mean and selected percentiles of urine concentrations (in $\mu\text{g/g}$ of creatinine) for the U.S. population aged 6 years and older, National Health and Nutrition Examination Survey, 1999-2002.

	Survey years	Geometric mean	Selected percentiles				Sample size
		(95% conf. interval)	(95% confidence interval)				
			50th	75th	90th	95th	
Total, age 6 and older	99-00	.181 (.157-.209)	.219 (.199-.238)	.423 (.391-.446)	.712 (.645-.757)	.933 (.826-1.07)	2257
	01-02	.199 (.181-.218)	.212 (.194-.232)	.404 (.377-.440)	.690 (.630-.754)	.917 (.813-.998)	2689
Age group							
6-11 years	99-00	*	.085 (.063-.107)	.147 (.123-.182)	.210 (.171-.316)	.300 (.184-.607)	310
	01-02	.075 (.059-.094)	.100 (.083-.112)	.166 (.136-.192)	.233 (.206-.281)	.291 (.221-.440)	368
12-19 years	99-00	.071 (.051-.098)	.093 (.084-.106)	.147 (.130-.163)	.215 (.204-.240)	.283 (.222-.404)	648
	01-02	.078 (.067-.091)	.091 (.085-.101)	.136 (.123-.143)	.191 (.175-.234)	.280 (.234-.321)	762
20 years and older	99-00	.267 (.247-.289)	.288 (.261-.304)	.484 (.433-.545)	.769 (.727-.818)	1.07 (.927-1.17)	1299
	01-02	.261 (.236-.289)	.273 (.247-.303)	.481 (.426-.518)	.776 (.691-.850)	.979 (.874-1.12)	1559
Gender							
Males	99-00	.154 (.131-.182)	.174 (.158-.191)	.329 (.293-.382)	.617 (.537-.700)	.788 (.696-.929)	1121
	01-02	.159 (.143-.177)	.168 (.157-.182)	.334 (.304-.364)	.532 (.491-.653)	.757 (.690-.856)	1334
Females	99-00	.211 (.170-.261)	.267 (.239-.308)	.473 (.423-.551)	.783 (.690-.917)	1.09 (.813-1.38)	1136
	01-02	.245 (.216-.278)	.263 (.228-.297)	.479 (.414-.541)	.792 (.687-.884)	.985 (.876-1.16)	1355
Race/ethnicity							
Mexican Americans	99-00	.175 (.137-.223)	.181 (.144-.225)	.331 (.266-.418)	.612 (.441-.828)	.843 (.674-1.13)	780
	01-02	.156 (.136-.178)	.170 (.150-.184)	.282 (.263-.340)	.501 (.388-.614)	.693 (.507-.839)	682
Non-Hispanic blacks	99-00	.183 (.140-.240)	.201 (.168-.241)	.414 (.343-.472)	.658 (.516-.827)	.873 (.722-.962)	546
	01-02	.190 (.156-.232)	.195 (.174-.225)	.385 (.336-.449)	.676 (.559-.850)	.917 (.725-1.08)	667
Non-Hispanic whites	99-00	.175 (.146-.209)	.219 (.191-.250)	.432 (.387-.470)	.729 (.666-.783)	1.00 (.826-1.16)	760
	01-02	.205 (.184-.229)	.224 (.208-.242)	.421 (.382-.470)	.719 (.668-.784)	.931 (.806-1.05)	1132

* Not calculated. Proportion of results below limit of detection was too high to provide a valid result.

population. Blood cadmium reflects both recent and cumulative exposures. During typical environmental exposure, urinary cadmium predominantly reflects cumulative exposure and the concentration of cadmium in the kidney (Nordberg and Nordberg, 2001; Lauwerys and Hoet, 2001; Satarug et al., 2002).

A general population survey of approximately 4,700 adults in Germany in 1998 found levels of cadmium in blood and urine that were also similar or slightly higher than the adult values reported in the 1999-2000 and 2001-2002 samples (Becker et al., 2002; Becker et al., 2003). Creatinine-corrected urine cadmium values obtained in a study of 361 subjects from a U.S. community, where smelting activity had occurred in the past, and an unexposed comparison community were also similar to the corresponding values in this *Report* (Noonan et al., 2002). A general population survey of 10,753 adult Japanese women found geometric mean urinary cadmium levels that were approximately four-to five-fold higher than the levels found for U.S. adults in this *Report*. (Ezaki et al., 2003). People who are occupationally exposed may have blood and urine levels that are higher than levels in the general population. The 95th percentiles for blood cadmium levels in this *Report* are less than the OSHA criterion (OSHA, 29 CFR 1910.1027) for blood cadmium (5 µg/L), and the 95th percentile for urine cadmium is less than the OSHA criterion for urine cadmium (3 µg/gram of creatinine). Occupational criteria are provided here for comparison only, not to imply a safety level for general population exposure.

In recent studies, levels of urinary cadmium greater than or equal to 1 µg/gram of creatinine have been associated with increases in urinary protein markers of renal tubular function (Jarup et al., 2000; Moriguchi et al., 2004; Noonan et al., 2002). In addition, a decrease in bone density has been correlated with urinary cadmium excretion among middle-aged women with a mean urinary cadmium concentration of approximately 1 µg/gram creatinine (Staessen et al., 1999), and among women older than 60 years, the odds of low bone-mineral density increased by nearly three-fold when the blood cadmium level exceeded 1.1 µg/L (Alfven et al., 2002). In this *Report*, the urinary and blood cadmium levels at the 95th and 90th percentiles, respectively, approach these cited values associated with subclinical changes in renal function and bone mineral density. Further research is needed to address the public health consequences of such exposure in the United States.

Comparing Adjusted Geometric Means

Geometric mean levels of blood cadmium in the NHANES 2001-2002 sample could not be calculated for

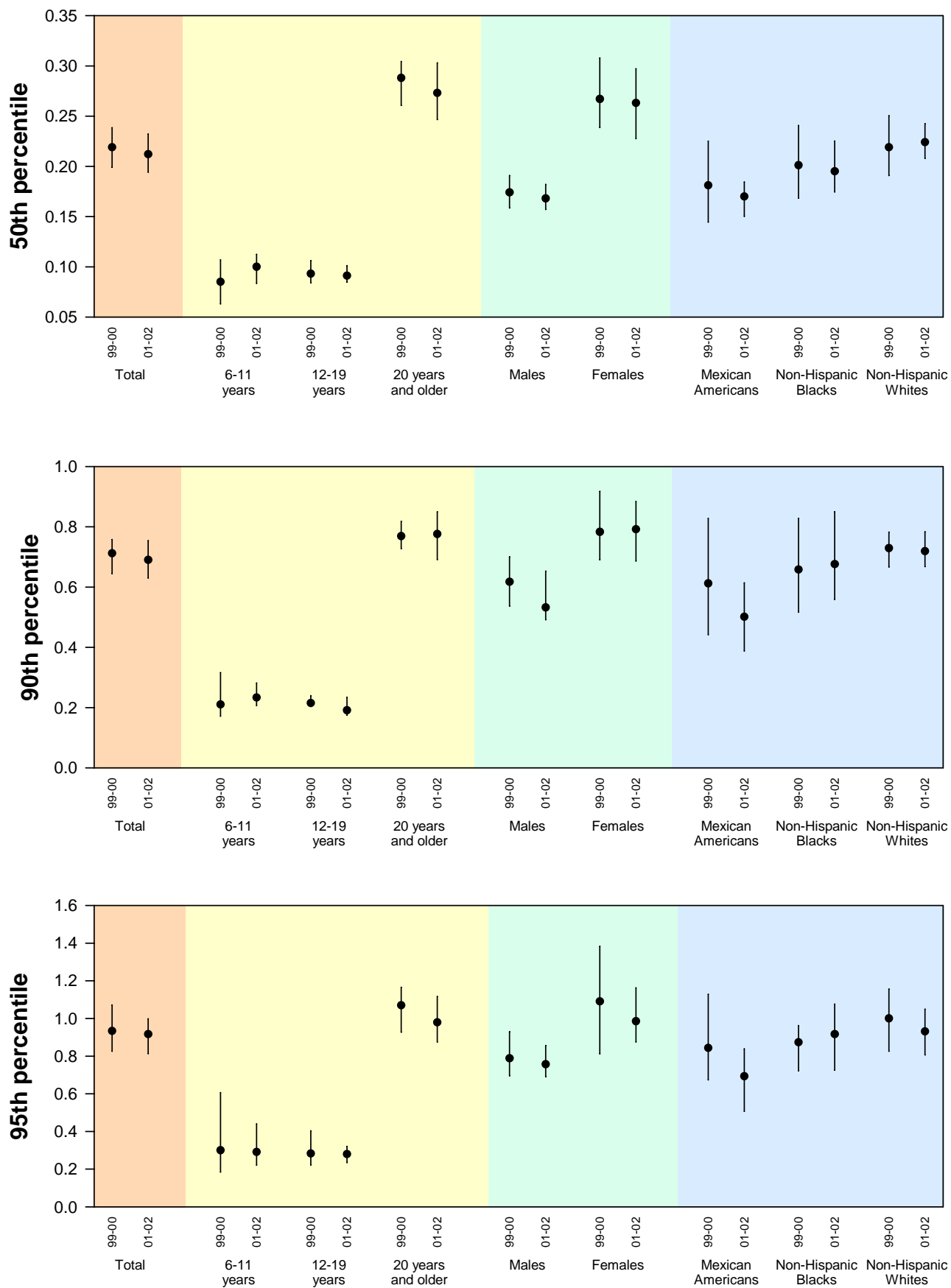
this *Report* due to an insufficient number of samples with detectable levels of cadmium. The adjusted geometric mean levels of blood cadmium for the demographic groups were compared previously in the NHANES 1999-2000 sample after adjusting for the covariates of race/ethnicity, age, gender, log serum cotinine, and urinary creatinine (data not shown). Adjusted geometric mean levels of blood cadmium were slightly higher in the group aged 20 years and older than either of the groups aged 6-11 or the 12-19 years; and the group aged 12-19 years was higher than the group aged 6-11 years. Females had slightly higher blood cadmium levels than males. Mexican Americans had higher adjusted geometric mean levels of blood cadmium than non-Hispanic whites or non-Hispanic blacks; and non-Hispanic blacks had higher blood cadmium levels than non-Hispanic whites. Similar relationships for age and gender were found in a study of NHANES III participants (Paschal et al., 2000).

Due to a recently demonstrated interference from molybdenum oxide when measuring low-level urinary cadmium using the ICP-MS method, both the 1999-2000 and 2001-2002 data were corrected for this interference based on the molybdenum measurement and expected proportion of molybdenum oxide. Geometric mean levels of urinary cadmium for the demographic groups were compared after adjusting for the covariates of race/ethnicity, age, gender, log serum cotinine, and urinary creatinine. In the NHANES 2001-2002 sample, the group aged 20 years and older had higher adjusted geometric mean levels of urinary cadmium than in either of the groups aged 6-11 or 12-19 years. Females had higher urinary cadmium levels than males. Higher urinary cadmium values in females than in males have been observed in other general population studies (Olsson et al., 2002) and, as noted previously, may be a possible consequence of increased gastrointestinal absorption of cadmium in females. It is unknown whether these differences associated with age, gender or race/ethnicity represent differences in exposure, pharmacokinetics, or the relationship of dose per body weight.

Finding a measurable amount of cadmium in blood or urine does not mean that the level of cadmium will result in an adverse health effect. These data provide physicians with a reference range so they can determine whether or not people have been exposed to higher levels of cadmium than are found in the general population. These data also will help scientists plan and conduct research about the relation between exposure to cadmium and health effects.

Figure 3. Cadmium in urine (creatinine corrected)

Selected percentiles with 95% confidence intervals of urine concentrations (in $\mu\text{g/g}$ of creatinine) for the U.S. population aged 6 years and older, National Health and Nutrition Examination Survey, 1999-2002.



Cesium

CAS No. 7440-46-2

General Information

Cesium is a silver-white metal that is found naturally in rock, soil, and clay. Inorganic cesium compounds are commonly used in photomultiplier tubes, vacuum tubes, scintillation counters, infrared lamps, semiconductors, high-power gas-ion devices, and as polymerization catalysts and photographic emulsions. Radioactive ^{137}Cs has been used medically to treat cancer.

Most human exposure to cesium occurs through the diet. For absorbed cesium salts, the body half-life is estimated to be 70-109 days based on ^{137}Cs exposures. Little is known about the health effects of this metal although cesium is generally of low toxicity when given to animals. However, cesium hydroxide is corrosive and irritating when concentrations are high. Workplace air standards for external exposure for certain cesium salts

are recommended by NIOSH on the basis of these irritant effects. It is not known whether cesium compounds are carcinogenic.

Interpreting Levels of Urinary Cesium Reported in the Tables

Urinary cesium levels were measured in a subsample of NHANES participants aged 6 years and older. Participants were selected within the specified age range to be a representative sample of the U.S. population. In one study of clinically submitted specimens (Komaromy-Hiller et al., 2000), mean urinary cesium concentrations were slightly lower than those reported here. In a small population study of Europeans, Minoia et al. (1990) found average urinary cesium levels to be comparable to levels in this *Report*. Median values in the NHANES 1999-2000 and 2001-2002 subsamples are more than

Table 12. Cesium

Geometric mean and selected percentiles of urine concentrations (in $\mu\text{g/L}$) for the U.S. population aged 6 years and older, National Health and Nutrition Examination Survey, 1999-2002.

	Survey years	Geometric mean (95% conf. interval)	Selected percentiles (95% confidence interval)				Sample size
			50th	75th	90th	95th	
Total, age 6 and older	99-00	4.35 (4.00-4.74)	4.80 (4.40-5.30)	7.10 (6.50-7.70)	9.60 (8.80-10.3)	11.4 (10.2-12.9)	2464
	01-02	4.81 (4.40-5.26)	5.49 (5.09-5.89)	7.91 (7.47-8.39)	10.4 (9.56-11.4)	12.6 (11.1-13.8)	2690
Age group							
6-11 years	99-00	4.87 (4.08-5.81)	5.60 (4.60-6.70)	7.30 (6.70-8.00)	9.00 (7.90-10.1)	9.70 (8.90-10.4)	340
	01-02	4.87 (4.08-5.82)	5.61 (4.69-6.52)	7.96 (6.77-8.84)	9.79 (8.64-10.6)	11.1 (10.2-12.4)	368
12-19 years	99-00	4.55 (4.09-5.05)	5.10 (4.30-5.60)	6.80 (6.10-7.70)	8.80 (8.00-9.40)	10.4 (8.90-12.3)	718
	01-02	5.22 (4.57-5.95)	5.62 (5.13-6.12)	7.55 (7.13-8.04)	9.71 (9.12-11.1)	12.0 (10.0-15.0)	762
20 years and older	99-00	4.26 (3.94-4.62)	4.80 (4.40-5.30)	7.10 (6.50-7.60)	9.80 (8.80-10.5)	11.6 (10.2-13.2)	1406
	01-02	4.74 (4.32-5.20)	5.43 (5.05-5.87)	7.96 (7.43-8.52)	10.6 (9.73-11.5)	12.8 (11.2-14.2)	1560
Gender							
Males	99-00	4.84 (4.35-5.38)	5.50 (4.60-5.90)	7.50 (6.90-8.20)	9.70 (8.60-10.7)	11.6 (10.3-13.0)	1226
	01-02	5.34 (4.89-5.84)	6.11 (5.61-6.64)	8.26 (7.84-9.08)	10.8 (10.1-12.1)	12.8 (11.3-15.0)	1335
Females	99-00	3.95 (3.63-4.29)	4.50 (4.10-4.80)	6.60 (6.20-7.30)	9.10 (8.30-9.90)	11.1 (9.90-12.9)	1238
	01-02	4.36 (3.95-4.81)	4.87 (4.45-5.25)	7.29 (6.71-8.01)	9.77 (9.07-11.0)	12.4 (10.4-13.8)	1355
Race/ethnicity							
Mexican Americans	99-00	4.32 (3.82-4.89)	4.70 (4.20-5.10)	6.60 (6.20-7.10)	9.10 (8.00-9.80)	10.9 (9.50-12.6)	884
	01-02	4.63 (4.10-5.24)	5.29 (4.59-5.89)	7.08 (6.42-7.99)	9.13 (7.86-11.3)	11.3 (8.81-14.9)	683
Non-Hispanic blacks	99-00	4.94 (4.33-5.64)	5.40 (4.80-6.30)	7.40 (6.80-8.20)	9.80 (8.80-10.8)	11.5 (9.80-13.1)	568
	01-02	4.93 (4.70-5.17)	5.31 (5.05-5.63)	7.36 (6.97-7.59)	9.44 (8.71-9.68)	10.7 (10.1-12.3)	667
Non-Hispanic whites	99-00	4.25 (3.83-4.72)	4.70 (4.20-5.50)	7.10 (6.50-7.80)	9.60 (8.80-10.4)	11.7 (10.3-13.3)	821
	01-02	4.77 (4.27-5.32)	5.46 (4.97-6.03)	7.97 (7.43-8.55)	10.4 (9.54-11.4)	12.6 (11.0-13.8)	1132

twice the median values reported in a nonrandom subsample from NHANES III (1988-1994) (Paschal et al., 1998), which may be due to methodologic, population, or exposure differences.

Comparing Adjusted Geometric Means

Geometric mean levels of urinary cesium for the demographic groups were compared after adjusting for the covariates of race/ethnicity, age, gender, log serum cotinine, and urinary creatinine (data not shown). In NHANES 2001-2002, adjusted geometric mean levels of urinary cesium were slightly higher for children aged 6-11 years than for either of the groups aged 12-19 years or 20 years and older. The group aged 12-19 years had lower levels than the 20 year and older group. Mexican Americans had higher levels than non-Hispanic blacks. Non-Hispanic whites had higher levels than non-Hispanic blacks. It is unknown whether these differences associated with age or race/ethnicity represent

differences in exposure, pharmacokinetics, or the relationship of dose per body weight.

Finding a measurable amount of cesium in urine does not mean that the level of cesium causes an adverse health effect. Whether cesium at the levels reported here is a cause for health concern is not yet known; more research is needed. These urinary cesium data provide physicians with a reference range so that they can determine whether or not people have been exposed to higher levels of cesium than levels found in the general population. These data will also help scientists plan and conduct research about exposure to cesium and health effects.

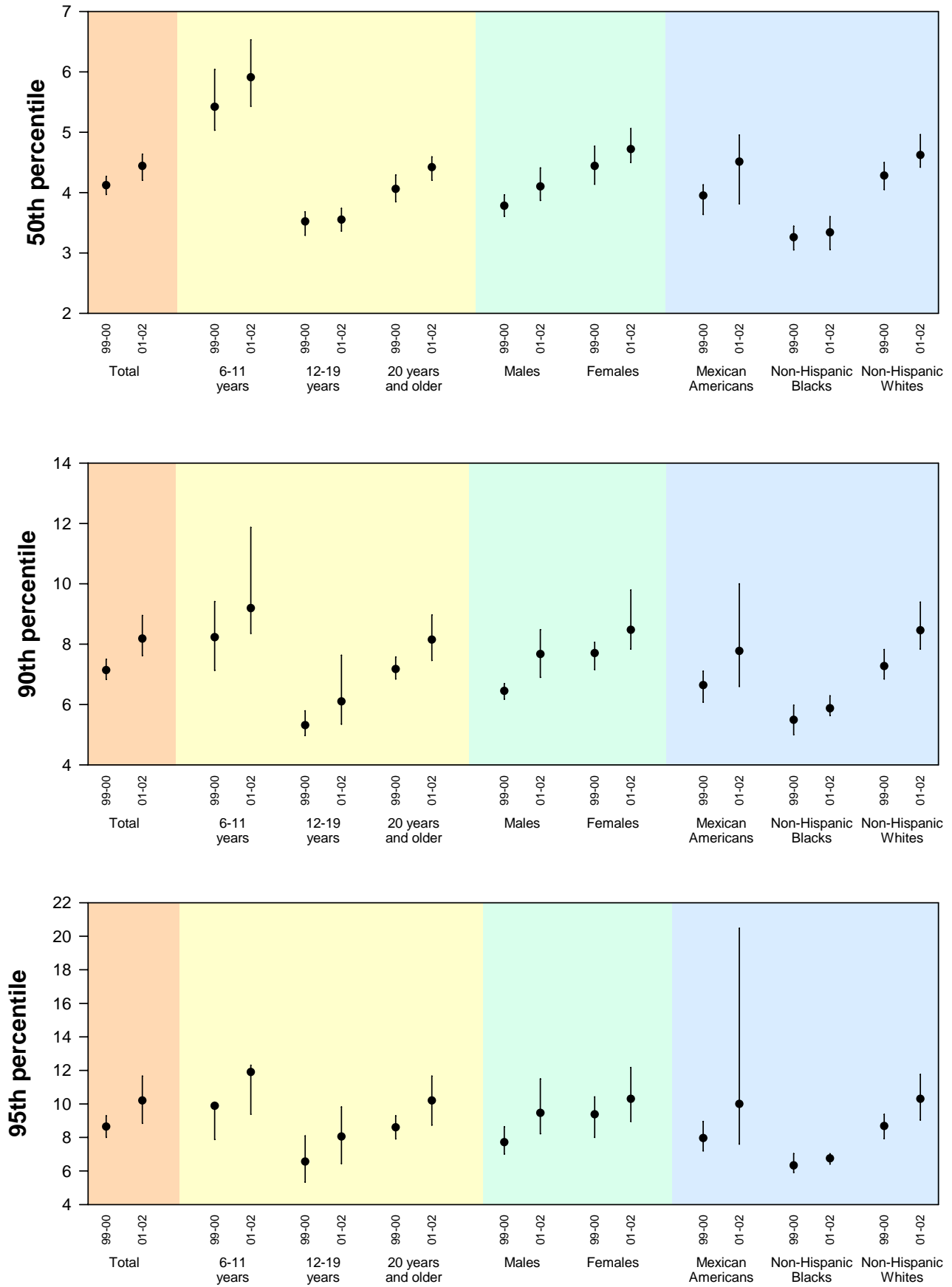
Table 13. Cesium (creatinine corrected)

Geometric mean and selected percentiles of urine concentrations (in $\mu\text{g/g}$ of creatinine) for the U.S. population aged 6 years and older, National Health and Nutrition Examination Survey, 1999-2002.

	Survey years	Geometric mean (95% conf. interval)	Selected percentiles (95% confidence interval)				Sample size
			50th	75th	90th	95th	
Total, age 6 and older	99-00	4.10 (3.96-4.25)	4.12 (3.97-4.27)	5.41 (5.21-5.70)	7.14 (6.83-7.50)	8.64 (8.00-9.30)	2464
	01-02	4.54 (4.30-4.79)	4.44 (4.20-4.64)	6.06 (5.66-6.47)	8.18 (7.62-8.95)	10.2 (8.84-11.7)	2689
Age group							
6-11 years	99-00	5.34 (5.03-5.67)	5.42 (5.03-6.04)	6.63 (6.18-7.13)	8.23 (7.13-9.41)	9.89 (7.88-10.1)	340
	01-02	5.95 (5.48-6.46)	5.91 (5.43-6.53)	7.77 (7.00-8.28)	9.19 (8.35-11.9)	11.9 (9.38-12.3)	368
12-19 years	99-00	3.43 (3.29-3.58)	3.52 (3.29-3.68)	4.35 (4.17-4.56)	5.31 (4.97-5.79)	6.56 (5.33-8.09)	718
	01-02	3.73 (3.41-4.08)	3.55 (3.36-3.74)	4.74 (4.40-5.13)	6.10 (5.35-7.63)	8.05 (6.44-9.82)	762
20 years and older	99-00	4.08 (3.88-4.29)	4.06 (3.85-4.29)	5.38 (5.04-5.85)	7.17 (6.84-7.58)	8.60 (7.91-9.30)	1406
	01-02	4.54 (4.30-4.78)	4.42 (4.20-4.59)	5.94 (5.64-6.40)	8.15 (7.46-8.97)	10.2 (8.74-11.7)	1559
Gender							
Males	99-00	3.78 (3.65-3.91)	3.78 (3.60-3.96)	4.96 (4.72-5.20)	6.45 (6.18-6.70)	7.71 (7.01-8.64)	1226
	01-02	4.22 (3.96-4.51)	4.10 (3.87-4.41)	5.60 (5.27-6.03)	7.67 (6.90-8.48)	9.46 (8.22-11.5)	1334
Females	99-00	4.43 (4.20-4.68)	4.44 (4.14-4.77)	5.92 (5.36-6.47)	7.70 (7.16-8.06)	9.38 (8.00-10.4)	1238
	01-02	4.86 (4.58-5.16)	4.72 (4.50-5.06)	6.54 (5.93-7.00)	8.47 (7.84-9.79)	10.3 (8.95-12.2)	1355
Race/ethnicity							
Mexican Americans	99-00	3.99 (3.73-4.25)	3.95 (3.64-4.13)	5.09 (4.79-5.38)	6.64 (6.08-7.10)	7.96 (7.20-8.95)	884
	01-02	4.51 (4.00-5.08)	4.51 (3.82-4.95)	5.91 (5.31-6.64)	7.77 (6.60-10.0)	10.0 (7.60-20.5)	682
Non-Hispanic blacks	99-00	3.21 (2.90-3.56)	3.26 (3.05-3.44)	4.30 (4.00-4.55)	5.49 (5.00-5.98)	6.33 (5.91-7.04)	568
	01-02	3.38 (3.19-3.57)	3.34 (3.05-3.60)	4.41 (4.15-4.78)	5.87 (5.63-6.29)	6.75 (6.41-7.03)	667
Non-Hispanic whites	99-00	4.26 (4.07-4.47)	4.28 (4.05-4.50)	5.63 (5.26-6.05)	7.27 (6.84-7.83)	8.68 (7.93-9.38)	821
	01-02	4.81 (4.55-5.08)	4.62 (4.42-4.96)	6.33 (5.91-6.68)	8.46 (7.84-9.39)	10.3 (9.04-11.8)	1132

Figure 4. Cesium in urine (creatinine corrected)

Selected percentiles with 95% confidence intervals of urine concentrations (in $\mu\text{g/g}$ of creatinine) for the U.S. population aged 6 years and older, National Health and Nutrition Examination Survey, 1999-2002.



Cobalt

CAS No. 7440-48-4

General Information

Cobalt is a magnetic element that occurs in nature either as a steel-gray, shiny, hard metal or in combination with other elements. The cobalt used in U.S. industry is imported or obtained by recycling scrap metal that contains cobalt. Among its many uses are the manufacture of superalloys used in gas turbines in aircraft engines, hard-metal alloys (in combination with tungsten carbide), blue-colored pigments, and fertilizers. Cobalt is used as a drying agent in paints, varnishes, and inks. It is also a component of porcelain enamel applied to steel bathroom fixtures, large appliances, and kitchenware. Cobalt compounds are used as catalysts in the production of oil and gas and in the synthesis of polyester and other materials. Cobalt compounds are also used in the manufacture of battery electrodes, steel-belted radial tires, automobile airbags, diamond-polishing

wheels, and magnetic recording media.

Cobalt occurs naturally in airborne dust, seawater, and many types of soil. It is also emitted into the environment from burning coal and oil and from car and truck exhaust. Usual human exposure is from food sources. Cobalt may be released into the systemic circulation of patients who receive joint prostheses that are fabricated from cobalt alloys (Lhotka et al., 2003). Exposure in the workplace may come from electroplating, the refining or processing of alloys, the grinding of tungsten carbide-type, hard-metal cutting tools, and the use of diamond-polishing wheels containing cobalt metal. Workplace standards for external air exposure to cobalt and several of its compounds have been established by OSHA and ACGIH.

Table 14. Cobalt

Geometric mean and selected percentiles of urine concentrations (in µg/L) for the U.S. population aged 6 years and older, National Health and Nutrition Examination Survey, 1999-2002.

	Survey years	Geometric mean (95% conf. interval)	Selected percentiles (95% confidence interval)				Sample size
			50th	75th	90th	95th	
Total, age 6 and older	99-00	.375 (.336-.419)	.400 (.360-.440)	.630 (.570-.670)	.940 (.890-1.03)	1.32 (1.16-1.48)	2465
	01-02	.379 (.355-.404)	.410 (.380-.430)	.610 (.570-.660)	.930 (.860-1.00)	1.27 (1.15-1.44)	2690
Age group							
6-11 years	99-00	.499 (.427-.583)	.520 (.430-.600)	.740 (.610-.900)	1.03 (.860-1.12)	1.22 (1.03-1.50)	340
	01-02	.452 (.377-.543)	.510 (.430-.610)	.710 (.660-.810)	1.07 (.940-1.21)	1.28 (1.17-1.53)	368
12-19 years	99-00	.519 (.463-.581)	.520 (.480-.550)	.810 (.670-.890)	1.16 (1.01-1.47)	1.52 (1.26-2.56)	719
	01-02	.515 (.469-.564)	.520 (.470-.570)	.750 (.690-.840)	1.23 (1.07-1.32)	1.59 (1.37-1.99)	762
20 years and older	99-00	.343 (.305-.386)	.360 (.320-.410)	.560 (.510-.640)	.880 (.800-.950)	1.28 (1.07-1.39)	1406
	01-02	.352 (.333-.373)	.370 (.350-.400)	.550 (.520-.590)	.860 (.790-.930)	1.15 (1.04-1.42)	1560
Gender							
Males	99-00	.371 (.331-.416)	.400 (.360-.440)	.580 (.530-.630)	.810 (.730-.890)	1.01 (.900-1.12)	1227
	01-02	.367 (.338-.399)	.390 (.360-.410)	.540 (.510-.590)	.780 (.740-.840)	1.05 (.960-1.14)	1335
Females	99-00	.379 (.333-.431)	.410 (.340-.460)	.670 (.590-.790)	1.17 (.930-1.36)	1.49 (1.28-1.98)	1238
	01-02	.390 (.364-.417)	.430 (.390-.440)	.660 (.620-.700)	1.05 (.980-1.16)	1.44 (1.22-1.81)	1355
Race/ethnicity							
Mexican Americans	99-00	.418 (.348-.502)	.470 (.370-.530)	.660 (.620-.760)	1.05 (.950-1.19)	1.47 (1.25-1.67)	884
	01-02	.398 (.373-.424)	.420 (.400-.450)	.640 (.600-.710)	.950 (.850-1.03)	1.20 (1.06-1.48)	683
Non-Hispanic blacks	99-00	.434 (.405-.465)	.420 (.390-.470)	.680 (.620-.750)	1.15 (1.02-1.25)	1.45 (1.23-2.04)	568
	01-02	.435 (.388-.487)	.410 (.380-.440)	.650 (.540-.810)	1.15 (.840-1.63)	1.75 (1.32-2.22)	667
Non-Hispanic whites	99-00	.369 (.316-.431)	.400 (.340-.450)	.620 (.550-.700)	.930 (.820-1.07)	1.29 (1.02-1.65)	822
	01-02	.359 (.327-.394)	.380 (.350-.430)	.580 (.520-.650)	.870 (.800-.930)	1.16 (1.04-1.32)	1132

Cobalt constitutes 4% by weight of vitamin B-12 (cobalamin), an essential human nutrient. A nutritional requirement for cobalt not contained in dietary cobalamin has not been established. Cobalt is absorbed by oral and pulmonary routes. Human studies with ⁶⁰Co administered as soluble cobalt chloride have measured oral absorption ranging from approximately 1% to 25% (Smith et al., 1972). Once absorbed and distributed in the body, cobalt is excreted predominantly in the urine and to a lesser extent in the feces. Elimination reflects a multi-compartmental model dominated by compartments with half-lives on the order of several hours to a week, but with a minor fraction (10% to 15%) exhibiting a half-life of several years (Smith et al., 1972; Mosconi et al., 1994). A portion of cobalt retained for long periods is concentrated in the liver. Lung retention of cobalt compounds of low solubility, such as cobalt oxide, may be prolonged, with some fractions exhibiting pulmonary clearance half-lives of 1-2 years (Hedge et al., 1979). Recent inhalation exposure to soluble cobalt compounds may be monitored effectively by measuring cobalt in

urine or blood (Lison et al., 1994).

Most toxic effects of cobalt have been encountered in workplace situations. Cobalt compounds are a recognized cause of allergic contact dermatitis (Dickel et al., 2001; Lisi et al., 2003; Thomssen et al., 2001). Occupational exposure to cobalt-containing dusts has caused occupational asthma (Shirakawa et al., 1989; Pisati and Zedda, 1994). "Hard metal disease," an interstitial lung disorder with findings that range from alveolitis to pulmonary fibrosis, has been associated with exposure to dusts that contain cobalt, usually in combination with tungsten carbide (Cugell et al., 1990). The extent to which cobalt exposure alone causes interstitial lung disease is unresolved (Swennen et al., 1993; Linna et al., 2003)

Cobalt was once added as a foaming agent to beer and caused outbreaks of cardiomyopathy among heavy drinkers in the mid-1960s (Alexander et al., 1972). Other case reports have suggested a link between occupational cobalt exposure and cardiomyopathy (Jarvis et al., 1992).

Table 15. Cobalt (creatinine corrected)

Geometric mean and selected percentiles of urine concentrations (in µg/g of creatinine) for the U.S. population aged 6 years and older, National Health and Nutrition Examination Survey, 1999-2002.

	Survey years	Geometric mean (95% conf. interval)	Selected percentiles (95% confidence interval)				Sample size
			50th	75th	90th	95th	
Total, age 6 and older	99-00	.353 (.319-.391)	.328 (.302-.365)	.515 (.457-.581)	.810 (.679-.963)	1.16 (.938-1.50)	2465
	01-02	.358 (.333-.384)	.335 (.313-.360)	.523 (.487-.562)	.844 (.750-.955)	1.15 (1.00-1.28)	2689
Age group							
6-11 years	99-00	.547 (.467-.640)	.554 (.449-.647)	.774 (.626-.938)	1.00 (.830-1.48)	1.23 (.895-1.50)	340
	01-02	.552 (.508-.599)	.548 (.503-.609)	.756 (.660-.829)	1.00 (.900-1.25)	1.30 (1.03-1.73)	368
12-19 years	99-00	.391 (.353-.433)	.378 (.329-.407)	.535 (.469-.595)	.824 (.632-1.16)	1.44 (.821-3.54)	719
	01-02	.368 (.343-.396)	.352 (.327-.372)	.534 (.471-.611)	.851 (.673-.949)	1.06 (.932-1.24)	762
20 years and older	99-00	.328 (.297-.362)	.306 (.280-.328)	.471 (.428-.522)	.727 (.632-.905)	1.12 (.861-1.36)	1406
	01-02	.337 (.313-.363)	.312 (.293-.336)	.474 (.435-.513)	.792 (.704-.955)	1.15 (.963-1.33)	1559
Gender							
Males	99-00	.290 (.259-.324)	.279 (.248-.301)	.400 (.365-.449)	.608 (.534-.728)	.833 (.667-1.10)	1227
	01-02	.290 (.272-.310)	.277 (.256-.297)	.392 (.361-.425)	.642 (.574-.707)	.848 (.786-.929)	1334
Females	99-00	.426 (.378-.479)	.407 (.362-.457)	.605 (.550-.694)	.955 (.781-1.29)	1.50 (1.11-1.83)	1238
	01-02	.435 (.404-.468)	.408 (.382-.438)	.635 (.560-.708)	.993 (.867-1.16)	1.29 (1.12-1.60)	1355
Race/ethnicity							
Mexican Americans	99-00	.386 (.339-.439)	.376 (.333-.419)	.598 (.500-.669)	.895 (.826-1.00)	1.23 (1.11-1.35)	884
	01-02	.388 (.361-.417)	.361 (.333-.394)	.591 (.500-.662)	.872 (.777-.990)	1.10 (.990-1.27)	682
Non-Hispanic blacks	99-00	.282 (.275-.289)	.257 (.243-.278)	.417 (.378-.462)	.707 (.600-.785)	.975 (.757-1.60)	568
	01-02	.298 (.275-.323)	.268 (.251-.294)	.444 (.392-.511)	.728 (.582-.917)	1.03 (.740-1.55)	667
Non-Hispanic whites	99-00	.369 (.324-.421)	.351 (.313-.387)	.533 (.452-.611)	.861 (.667-1.16)	1.25 (.895-1.57)	822
	01-02	.362 (.331-.396)	.343 (.313-.368)	.523 (.479-.562)	.830 (.736-.983)	1.16 (.983-1.33)	1132

Cobalt compounds were formerly used in the treatment of anemia, a probable consequence of their capacity to stimulate erythropoietin production (Goldberg et al., 1988). A recent study observed elevated serum cobalt levels in association with excessive erythrocytosis among residents of a high-altitude mining community (Jefferson et al., 2002). Pharmaceutical preparations of cobalt used in the past as hematinics have been associated with the development of overt hypothyroidism (Kriss et al., 1955), and a subclinical decrement in thyroid production was observed in a study of cobalt-production workers (Swennen et al., 1993).

Cobalt compounds have elicited numerous genotoxic effects in both *in vitro* and *in vivo* assays (De Boeck et al., 2003) and have produced lung cancer in rats and mice following chronic inhalation (Bucher et al., 1999). An industry-wide study of hard-metal workers in France observed an increased mortality from lung cancer (Moulin et al., 1998). IARC considers cobalt and its compounds as possibly carcinogenic to humans. Information about external exposure (i.e., environmental levels) and health effects is available from ATSDR's Toxicological Profiles at <http://www.atsdr.cdc.gov/toxprofiles>.

Interpreting Levels of Urinary Cobalt Reported in the Tables

Urinary cobalt levels were measured in a subsample of NHANES participants aged 6 years and older. Participants were selected within the specified age range to be a representative sample of the U.S. population. The levels of cobalt measured in the adults in the NHANES 2001-2002 also are similar to those found in recent smaller general population surveys of European adults (Kristiansen et al., 1997; White and Sabbioni, 1998). Because concentrations of cobalt in the urine decline rapidly within 24 hours after an exposure ceases (Alexandersson et al., 1988), urinary measurements mainly reflect recent exposure, although substantial occupational exposure can produce elevated urinary levels for many weeks.

Individuals with occupational exposure to cobalt often have urinary cobalt levels that are many times higher than those of the general population (ATSDR, 2004). The ACGIH biological exposure index (BEI) for inorganic forms of cobalt (except insoluble cobalt oxides) is 15 µg/L (ACGIH, 2001). Information about the BEI is provided here for comparison, not to imply that the BEI is a safety level for general population exposure. For workers exposed to cobalt in the air, a distinction should be made between soluble cobalt and

insoluble (oxides and metallic) cobalt (Christensen and Poulsen, 1994; Lison et al., 1994). Exposure to soluble cobalt salts will produce proportionately higher urinary levels because of better absorption. Correlations between air-exposure levels and urinary cobalt levels in hard-metal fabricators are well documented (Ichikawa et al., 1985; Linnainmaa and Kiilunen, 1997; ACGIH 2001; Kraus et al., 2001; Lauwers and Hoet, 2001).

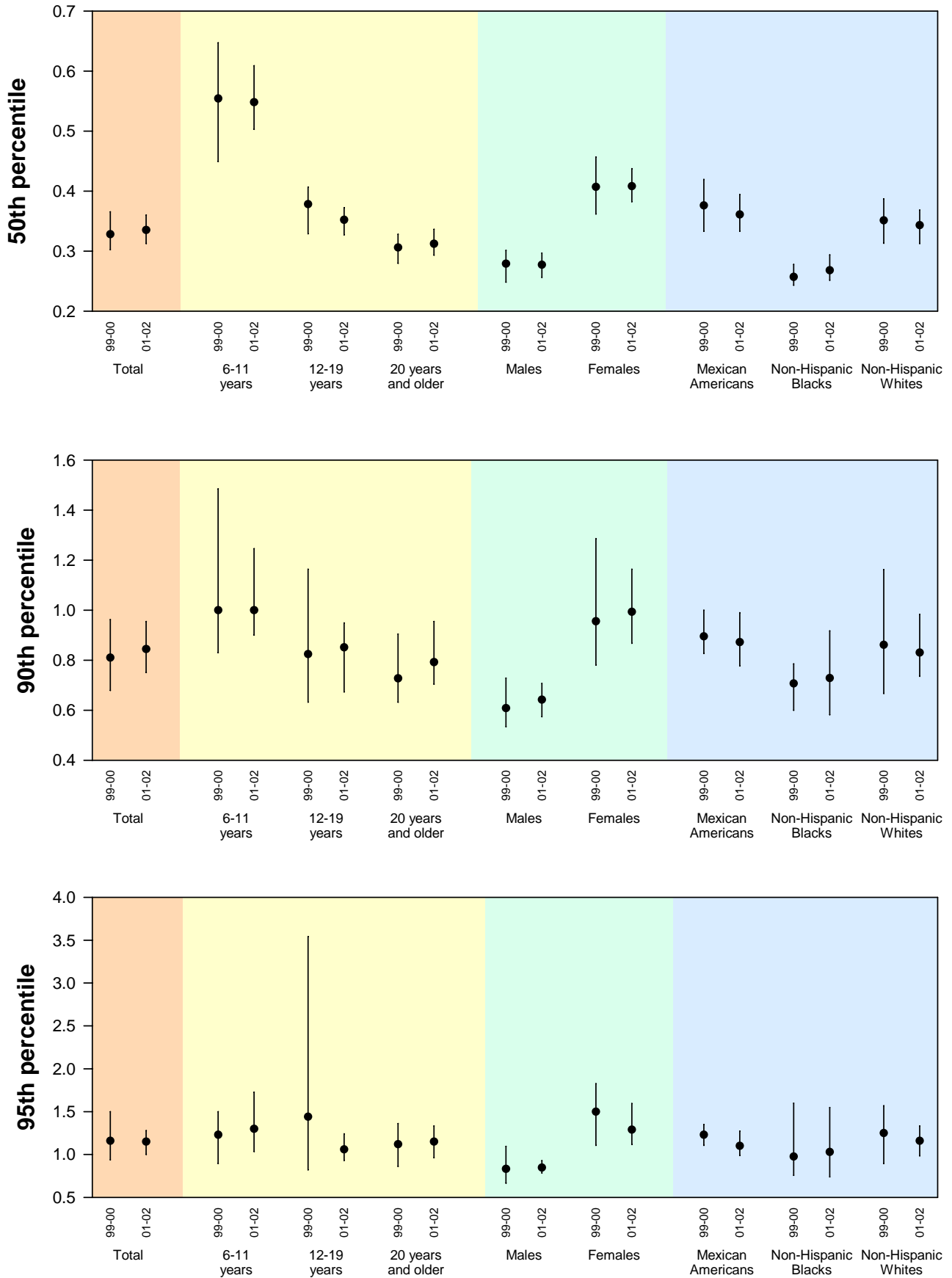
Comparing Adjusted Geometric Means

Geometric mean levels of urinary cobalt for the demographic groups were compared after adjusting for the covariates of race/ethnicity, age, gender, log serum cotinine, and urinary creatinine (data not shown). In NHANES 2001-2002, adjusted geometric mean levels of urinary cobalt were slightly higher for children aged 6-11 years than for people aged 12-19 years, with both age groups having higher levels than people aged 20 years and older. Urinary cobalt levels in females were higher than in males, and levels in non-Hispanic blacks were slightly lower than in either Mexican Americans or non-Hispanic whites. Relative higher urinary cobalt levels in females than in males have been noted in other investigations and may reflect increased cobalt uptake by premenopausal women (Kristiansen et al., 1997). It is unknown whether these differences associated with age, gender, or race/ethnicity represent differences in exposure, pharmacokinetics, or the relationship of dose per body weight.

Finding a measurable amount of cobalt in urine does not mean that the level of cobalt causes an adverse health effect. Whether cobalt at the levels reported here is a cause for health concern is not yet known; more research is needed. These urinary cobalt data provide physicians with a reference range so that they can determine whether or not people have been exposed to higher levels of cobalt than are found in the general population. These data will also help scientists plan and conduct research about exposure to cobalt and health effects.

Figure 5. Cobalt (creatinine corrected)

Selected percentiles with 95% confidence intervals of urine concentrations (in $\mu\text{g/g}$ of creatinine) for the U.S. population aged 6 years and older, National Health and Nutrition Examination Survey, 1999-2002.



Lead

CAS No. 7439-92-1

General Information

Elemental lead, a malleable, dense, blue-gray metal, is a naturally occurring element found in soils and rocks. It can be combined to form inorganic and organic molecules, such as lead phosphate and tetraethyl lead. Lead has a variety of uses in the manufacture of storage batteries; solders (particularly for electrical components and automobile radiators); metal alloys (including brass, bronze, and certain types of steel); plastics; leaded glass; ceramic glazes; ammunition; antique-molded or cast ornaments; and shielding used as protection from radiation sources. In the past, lead was added to residential paints and gasoline, and it has been used in plumbing for centuries. Small amounts of lead also may

be emitted from the burning of fossil fuels.

Since the elimination of leaded gasoline in the United States and the removal of lead from solder in canned food containers, adult lead exposures tend to be limited to certain occupational and recreational sources. For children, the major sources of exposure are from deteriorated lead-based paint and the resulting dust and soil contamination. However, less common sources of exposure still exist, including lead-glazed ceramic pottery; pewter utensils and drinking vessels; plumbing systems with lead-soldered joints or lead pipes; lead-based painted surfaces undergoing renovation or demolition; lead-containing folk remedies; bullet fragments retained in human tissue; lead-contaminated

Table 16. Lead in blood

Geometric mean and selected percentiles of blood concentrations (in µg/dL) for the U.S. population aged 1 year and older, National Health and Nutrition Examination Survey, 1999-2002.

	Survey years	Geometric mean	Selected percentiles				Sample size
		(95% conf. interval)	(95% confidence interval)				
			50th	75th	90th	95th	
Total, age 1 and older	99-00	1.66 (1.60-1.72)	1.60 (1.50-1.60)	2.40 (2.30-2.60)	3.80 (3.60-3.90)	4.90 (4.60-5.30)	7970
	01-02	1.45 (1.39-1.51)	1.40 (1.30-1.40)	2.20 (2.10-2.20)	3.40 (3.10-3.50)	4.40 (4.20-4.70)	8945
Age group							
1-5 years	99-00	2.23 (1.96-2.53)	2.20 (1.90-2.50)	3.30 (2.80-3.80)	4.80 (4.00-6.60)	7.00 (6.10-8.30)	723
	01-02	1.70 (1.55-1.87)	1.50 (1.40-1.70)	2.50 (2.20-2.80)	4.10 (3.40-5.00)	5.80 (4.70-6.90)	898
6-11 years	99-00	1.51 (1.36-1.66)	1.30 (1.20-1.50)	2.00 (1.70-2.40)	3.30 (2.70-3.60)	4.50 (3.40-6.20)	905
	01-02	1.25 (1.14-1.36)	1.10 (1.00-1.30)	1.60 (1.50-1.80)	2.70 (2.40-3.00)	3.70 (3.00-4.70)	1044
12-19 years	99-00	1.10 (1.04-1.17)	1.00 (.900-1.10)	1.40 (1.30-1.60)	2.30 (2.10-2.30)	2.80 (2.60-3.00)	2135
	01-02	.942 (.899-.986)	.800 (.800-.900)	1.20 (1.20-1.30)	1.90 (1.80-2.00)	2.70 (2.30-2.90)	2231
20 years and older	99-00	1.75 (1.68-1.81)	1.70 (1.60-1.70)	2.50 (2.50-2.60)	3.90 (3.70-4.00)	5.20 (4.80-5.50)	4207
	01-02	1.56 (1.49-1.62)	1.60 (1.50-1.60)	2.20 (2.20-2.30)	3.60 (3.30-3.70)	4.60 (4.20-4.90)	4772
Gender							
Males	99-00	2.01 (1.93-2.09)	1.80 (1.80-1.90)	2.90 (2.80-3.00)	4.40 (4.10-4.80)	6.00 (5.40-6.40)	3913
	01-02	1.78 (1.71-1.86)	1.70 (1.70-1.80)	2.70 (2.50-2.80)	3.90 (3.70-4.10)	5.30 (5.00-5.50)	4339
Females	99-00	1.37 (1.32-1.43)	1.30 (1.20-1.30)	1.90 (1.90-2.10)	3.00 (2.90-3.20)	4.00 (3.70-4.20)	4057
	01-02	1.19 (1.14-1.25)	1.10 (1.10-1.20)	1.80 (1.70-1.80)	2.60 (2.40-2.70)	3.60 (3.00-3.80)	4606
Race/ethnicity							
Mexican Americans	99-00	1.83 (1.75-1.91)	1.80 (1.60-1.80)	2.70 (2.60-2.90)	4.20 (3.90-4.50)	5.80 (5.10-6.60)	2742
	01-02	1.46 (1.34-1.60)	1.50 (1.30-1.60)	2.20 (2.00-2.60)	3.60 (3.30-4.00)	5.40 (4.40-6.60)	2268
Non-Hispanic blacks	99-00	1.87 (1.75-2.00)	1.70 (1.60-1.90)	2.80 (2.50-2.90)	4.20 (4.00-4.60)	5.70 (5.20-6.10)	1842
	01-02	1.65 (1.52-1.80)	1.60 (1.40-1.70)	2.50 (2.30-2.80)	4.20 (3.80-4.60)	5.70 (5.30-6.50)	2219
Non-Hispanic whites	99-00	1.62 (1.55-1.69)	1.60 (1.50-1.60)	2.40 (2.30-2.40)	3.60 (3.40-3.70)	5.00 (4.40-5.70)	2716
	01-02	1.43 (1.37-1.48)	1.40 (1.30-1.40)	2.10 (2.10-2.20)	3.10 (3.00-3.40)	4.10 (3.90-4.50)	3806

dust in indoor firing ranges; and contact with soil, dust, or water contaminated by active or inactive mining or smelting operations. Children may also be exposed to lead brought into the home on the work clothes of adults whose work involves lead.

Following inhalation of fine lead particulate or fume or ingestion of soluble lead compounds, absorbed lead is bound to erythrocytes and is distributed initially to multiple soft tissues, including the brain, kidneys, bone marrow, and gonads, and to a slower extent to the subperiosteal surface and matrix of bone. Lead also undergoes transplacental transport and poses a potential hazard to the fetus. The kinetics of lead clearance from the body are characterized by a multi-compartmental model, predominantly composed of the blood and soft tissues, with a half-life of 1 to 2 months, and the skeleton, with a half-life of years to decades. Approximately 70% of lead excretion occurs via the urine, with lesser amounts eliminated via the feces, and scant amounts through sweat, hair, and nails (Leggett,

1993; O'Flaherty, 1993). The fraction of absorbed lead not undergoing prompt excretion, which is approximately half of the absorbed lead, may be incorporated into bone, the site of about 90% of the body lead burden in most adults.

Lead exerts multisystemic toxic effects through a variety of mechanisms, including interference in the function of essential cations such as calcium, zinc, and iron; inhibition of certain enzymes; binding to ion channels and regulatory proteins; generation of reactive oxygen species; and alteration in gene expression. Large amounts of lead in the body can cause clinical anemia, kidney injury, abdominal pain, seizures, encephalopathy, and paralysis. An increased prevalence of anemia has been observed with blood lead levels (BLLs) in excess of 25 µg/dL in children (Schwartz et al., 1990), or higher than 50 µg/dL in adults (Lilis et al., 1977). Kidney toxicity associated with chronic high-dose lead exposure includes interstitial and peritubular fibrosis, but low-level environmental lead exposure also can be associated with

Table 17. Lead in urine

Geometric mean and selected percentiles of urine concentrations (in µg/L) for the U.S. population aged 6 years and older, National Health and Nutrition Examination Survey, 1999-2002.

	Survey years	Geometric mean	Selected percentiles				Sample size
		(95% conf. interval)	(95% confidence interval)				
			50th	75th	90th	95th	
Total, age 6 and older	99-00	.766 (.708-.828)	.800 (.700-.800)	1.30 (1.30-1.40)	2.10 (2.00-2.30)	2.90 (2.60-3.20)	2465
	01-02	.677 (.637-.718)	.600 (.600-.700)	1.20 (1.10-1.20)	2.00 (1.80-2.00)	2.60 (2.50-2.80)	2690
Age group							
6-11 years	99-00	1.07 (.955-1.20)	1.00 (.900-1.20)	1.50 (1.40-1.60)	2.40 (1.80-3.00)	3.40 (2.40-5.00)	340
	01-02	.753 (.661-.857)	.800 (.600-.800)	1.20 (1.00-1.30)	2.00 (1.60-2.40)	2.60 (2.10-3.60)	368
12-19 years	99-00	.659 (.579-.749)	.600 (.500-.800)	1.10 (.900-1.20)	1.70 (1.40-2.10)	2.20 (1.90-2.50)	719
	01-02	.564 (.526-.605)	.600 (.500-.600)	.900 (.800-1.10)	1.50 (1.40-1.70)	1.90 (1.70-2.10)	762
20 years and older	99-00	.752 (.691-.818)	.700 (.700-.800)	1.40 (1.20-1.50)	2.10 (1.90-2.30)	2.90 (2.50-3.20)	1406
	01-02	.688 (.641-.738)	.700 (.600-.700)	1.20 (1.10-1.20)	1.90 (1.80-2.10)	2.80 (2.50-2.90)	1560
Gender							
Males	99-00	.923 (.822-1.04)	.900 (.800-.900)	1.60 (1.40-1.80)	2.40 (2.20-2.90)	3.40 (2.90-3.70)	1227
	01-02	.808 (.757-.862)	.700 (.700-.800)	1.30 (1.30-1.50)	2.40 (2.20-2.70)	3.20 (2.90-3.50)	1335
Females	99-00	.642 (.589-.701)	.600 (.600-.700)	1.20 (1.10-1.30)	1.90 (1.60-2.10)	2.40 (2.00-2.90)	1238
	01-02	.573 (.535-.613)	.500 (.500-.600)	1.00 (1.00-1.10)	1.50 (1.40-1.70)	2.20 (1.90-2.40)	1355
Race/ethnicity							
Mexican Americans	99-00	1.02 (.915-1.13)	1.00 (.900-1.20)	1.70 (1.60-1.90)	2.80 (2.50-3.40)	4.10 (3.10-5.40)	884
	01-02	.833 (.745-.931)	.900 (.700-1.00)	1.50 (1.20-1.70)	2.40 (2.00-2.90)	3.20 (2.70-3.70)	683
Non-Hispanic blacks	99-00	1.11 (1.00-1.23)	1.10 (1.00-1.20)	1.90 (1.50-2.00)	2.90 (2.40-3.50)	4.20 (3.30-5.70)	568
	01-02	.940 (.833-1.06)	.900 (.800-.900)	1.50 (1.30-1.80)	2.60 (2.00-3.20)	3.70 (2.90-4.80)	667
Non-Hispanic whites	99-00	.695 (.625-.773)	.700 (.600-.700)	1.30 (1.10-1.40)	1.90 (1.70-2.20)	2.60 (2.30-3.00)	822
	01-02	.610 (.572-.651)	.600 (.600-.700)	1.00 (1.00-1.10)	1.80 (1.70-2.00)	2.40 (2.10-2.50)	1132

small decrements in renal function (Payton et al., 1994; Kim et al., 1996; Muntner et al., 2003).

Low-level environmental lead exposure has been associated with subclinical decrements in neurocognitive function in young children and elevated blood pressure in adults. Although in 1991 the Centers for Disease Control and Prevention (CDC) established 10 µg/dL as a blood lead concentration of concern in children, no threshold for lead's effects has yet been identified (National Research Council, 1993). Recent studies have suggested possible neurodevelopmental effects at blood lead concentrations of less than 10 µg/dL (Lanphear et al., 2000; Canfield et al., 2003); further assessment is ongoing. In adults, subtle, nonspecific neurocognitive effects may occur at BLLs as low as 20-60 µg/dL (Mantere et al., 1984; Schwartz et al., 2001), with overt encephalopathy, seizures, and peripheral neuropathy at higher levels (e.g., levels greater than 100 µg/dL). Results of studies of adults with occupational or environmental lead exposure have shown consistent

associations between increased BLLs and increased blood pressure (Schwartz, 1995; Staessen et al., 1995; Nash et al., 2003) and associations between increased bone lead concentrations and blood pressure (Hu et al., 1996; Korrick et al., 1999).

The potential adverse effects of lead on reproduction are an area of ongoing research and may include increased spontaneous abortion in women (Borja-Aburto et al., 1999) and problems with sperm formation in men (Alexander et al., 1996; Telisman et al., 2000). The International Agency for Research on Cancer (IARC) considers lead as a possible human carcinogen, and the National Toxicology Program (NTP) considers lead and its compounds as reasonably anticipated to be human carcinogens (NTP, 2005), but further study is needed on the relation between lead exposure and cancer in people (Jemal et al., 2002).

Table 18. Lead in urine (creatinine corrected)

Geometric mean and selected percentiles of urine concentrations (in µg/g of creatinine) for the U.S. population aged 6 years and older, National Health and Nutrition Examination Survey, 1999-2002.

	Survey years	Geometric mean	Selected percentiles				Sample size
		(95% conf. interval)	(95% confidence interval)				
			50th	75th	90th	95th	
Total, age 6 and older	99-00	.721 (.700-.742)	.700 (.677-.725)	1.11 (1.06-1.15)	1.70 (1.62-1.85)	2.37 (2.21-2.76)	2465
	01-02	.639 (.603-.677)	.634 (.586-.676)	1.03 (.962-1.08)	1.52 (1.42-1.60)	2.03 (1.89-2.22)	2689
Age group							
6-11 years	99-00	1.17 (.975-1.41)	1.06 (.918-1.22)	1.55 (1.22-1.97)	2.71 (1.67-4.66)	4.66 (1.97-18.0)	340
	01-02	.918 (.841-1.00)	.870 (.798-.933)	1.26 (1.12-1.43)	2.33 (1.59-3.64)	3.64 (1.83-5.56)	368
12-19 years	99-00	.496 (.460-.535)	.469 (.408-.508)	.702 (.655-.828)	1.10 (.981-1.28)	1.65 (1.15-2.78)	719
	01-02	.404 (.380-.428)	.373 (.342-.400)	.602 (.541-.702)	.990 (.882-1.18)	1.41 (1.07-1.63)	762
20 years and older	99-00	.720 (.683-.758)	.712 (.667-.739)	1.10 (1.02-1.18)	1.69 (1.53-1.87)	2.31 (2.11-2.62)	1406
	01-02	.658 (.617-.703)	.649 (.608-.702)	1.04 (.992-1.11)	1.51 (1.40-1.61)	2.00 (1.85-2.19)	1559
Gender							
Males	99-00	.720 (.679-.763)	.693 (.645-.734)	1.10 (.991-1.22)	1.68 (1.50-2.09)	2.43 (2.15-3.03)	1227
	01-02	.639 (.607-.673)	.638 (.586-.684)	1.01 (.957-1.08)	1.55 (1.41-1.61)	2.06 (1.88-2.43)	1334
Females	99-00	.722 (.681-.765)	.706 (.667-.746)	1.11 (1.05-1.16)	1.74 (1.50-2.02)	2.38 (2.03-2.88)	1238
	01-02	.639 (.594-.688)	.625 (.571-.670)	1.03 (.938-1.11)	1.50 (1.39-1.61)	1.98 (1.85-2.15)	1355
Race/ethnicity							
Mexican Americans	99-00	.940 (.876-1.01)	.882 (.796-1.02)	1.43 (1.36-1.56)	2.38 (2.08-2.77)	3.31 (2.78-4.18)	884
	01-02	.810 (.731-.898)	.769 (.702-.893)	1.28 (1.09-1.44)	2.05 (1.75-2.50)	2.78 (2.55-3.33)	682
Non-Hispanic blacks	99-00	.722 (.659-.790)	.667 (.583-.753)	1.11 (.988-1.20)	1.98 (1.56-2.51)	2.83 (2.20-3.88)	568
	01-02	.644 (.559-.742)	.606 (.510-.710)	.962 (.853-1.19)	1.79 (1.36-2.33)	2.75 (2.04-3.98)	667
Non-Hispanic whites	99-00	.696 (.668-.725)	.677 (.645-.718)	1.07 (.997-1.14)	1.66 (1.50-1.83)	2.31 (1.94-2.82)	822
	01-02	.615 (.579-.654)	.621 (.571-.667)	1.00 (.930-1.06)	1.46 (1.37-1.52)	1.88 (1.61-2.00)	1132

Interpreting Levels of Lead in Blood and Urine Reported in the Tables

Levels of lead in blood were measured in all participants aged 1 year and older and urine lead levels were measured in a sample of people aged 6 years and older. Participants were selected to be a representative sample of the U.S. population. Blood lead measurement is the preferred method of evaluating lead exposure and its health effects in people. BLLs are contributed to by both recent intake and an equilibration with stored lead in other tissues, particularly in the skeleton. Urinary lead measurements are more variable than blood lead levels for a given individual.

The U.S. adult population has similar or slightly lower BLLs than adults in other developed nations. A general population survey of 4,646 adults in Germany in 1998 reported a geometric mean blood lead concentration of 3.07 $\mu\text{g}/\text{dL}$ (Becker et al., 2002), a value nearly twice that found for U.S. adults in the 2001-2002 NHANES sample. A general population survey of 1,164 adults in Italy in 2000 found blood lead values slightly more than double those reported for U.S. adults in the 2001-2002 NHANES sample (Apostoli et al., 2002a).

In 1991, CDC designated 10 $\mu\text{g}/\text{dL}$ as the blood lead level of concern in children, a level associated with the risk for subtle neurodevelopmental impairments. For children 1- 5 years old sampled over the four year period 1999-2002, the geometric mean BLL was 1.9 $\mu\text{g}/\text{dL}$ (1.8-2.1), with 1.6% (1.1-2.3) of the children having BLLs greater than or equal to 10 $\mu\text{g}/\text{dL}$. Data from NHANES III, (phase 2, 1991-1994) showed that 4.4% of children aged 1-5 years had BLLs greater than or equal to 10 $\mu\text{g}/\text{dL}$, and the geometric mean BLL for children aged 1-5 years was 2.7 $\mu\text{g}/\text{dL}$ (Pirkle et al., 1998). State childhood blood lead surveillance systems reported blood lead results for 2.4 million children to CDC in 2001. Of these children, 3.09% had a confirmed BLL of greater than or equal to 10 $\mu\text{g}/\text{dL}$ (CDC, 2003a). Among a predominantly non-white population of U.S. children aged 0 to 17 years who were screened at an urban medical center in Washington, D.C. in 2001 and 2002, the geometric mean BLL in males was 3.2 $\mu\text{g}/\text{dL}$ ($n = 5,584$) and 3.0 $\mu\text{g}/\text{dL}$ in females ($n = 5,562$) (Soldin et al., 2003). These levels are higher than levels in similar age groups in the 2001-2002 NHANES sample and may reflect a higher prevalence of elevated BLLs that occur among children who 1) are non-Hispanic black and Mexican American; 2) live in urban settings; 3) are from lower socioeconomic groups; 4) are immigrants, refugees, or 5) reside in housing built before 1950 (CDC, 2003a; CDC, 2002; Geltman et al., 2001). In places where leaded gasoline is still used, such as in

Bangladesh, BLLs among school children are similar to BLLs measured in the United States before lead was removed from gasoline (i.e., a mean BLL of 15.0 $\mu\text{g}/\text{dL}$ and 87.4% of children with levels in excess of 10 $\mu\text{g}/\text{dL}$ [Kaiser et al., 2001]).

The U.S. Department of Labor, Occupational Safety and Health Administration (OSHA) requires monitoring of blood lead levels when occupational exposure to airborne levels of lead exceeds the established action level of greater than 30 micrograms per cubic meter of air (OSHA, 29 CFR 1910.1025). First established in the late 1970s, OSHA regulations have required medical removal of workers from workplace lead exposure when blood lead concentrations exceed 50 $\mu\text{g}/\text{dL}$ or at lower levels per a physician's discretion. The American Conference of Governmental Industrial Hygienists (ACGIH, 2001) established a Biological Exposure Index (BEI) for inorganic lead in 1995 which recommended that BLL in workers remain less than 30 $\mu\text{g}/\text{dL}$. Levels for adults in the NHANES 1999-2000 and 2001-2002 samples are generally below these worker thresholds (four adult NHANES participants were above 30 $\mu\text{g}/\text{dL}$).

Comparing Adjusted Geometric Means

Geometric mean BLLs for the demographic groups were compared after adjusting for the covariates of race/ethnicity, age, gender, and log serum cotinine (data not shown). In NHANES 2001-2002, adjusted geometric mean BLLs were higher in children aged 1-5 years than in children aged 6-11 years, and both these age groups had higher levels than did those aged 12-19 years. BLLs in adults aged 20 years and older were higher than BLLs in the group aged 12-19 years. BLLs for males were higher than those for females. Non-Hispanic whites had lower BLLs than non-Hispanic blacks.

For urinary lead in NHANES 2001-2002, adjusted geometric mean levels of urinary lead in the group aged 6-11 years were higher than either the group aged 12-19 years or the group aged 20 years and older. The group aged 20 years and older had higher levels than the group aged 12-19 years. Mexican Americans had higher urinary levels than either non-Hispanic blacks or whites. Non-Hispanic blacks had higher levels than non-Hispanic whites. Males had slightly higher levels than females.

It is unknown whether these differences associated with age, gender or race/ethnicity represent differences in exposure, pharmacokinetics, or the relationship of dose per body weight. For instance, to account for the decreasing BLLs observed with increasing ages during childhood, several explanations are possible, including

decreasing exposure, dilution of lead by growth of body mass, or changing equilibria with bone turnover.

These blood and urine levels of lead provide physicians with a reference range so that they can determine whether or not people have been exposed to higher levels of lead than are found in the general population. These data will also help scientists plan and conduct research about exposure to lead and health effects.

