VITAL & HEALTH STATISTICS

Blood Lead Levels for Persons Ages 6 Months–74 Years:
United States, 1976–80

This report provides statistics on the distribution of blood lead levels for the civilian noninstitutionalized population, ages 6 months–74 years. Blood lead data collected in the second National Health and Nutrition Examination Survey (NHANES II) are presented by selected demographic and socioeconomic factors, and in association with relevant medical history items and biochemical measures.

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Cooperation of the U.S. Bureau of the Census

Under the legislation establishing the National Health Survey, the Public Health Service is authorized to use, insofar as possible, the services or facilities of other Federal, State, or private agencies.

In accordance with specifications established by the National Center for Health Statistics, the U.S. Bureau of the Census participated in the design and selection of the sample and carried out the household interview stage of the data collection and certain parts of the statistical processing.

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Symbols

- - -	Data not available
. . .	Category not applicable
-	Quantity zero
0.0	Quantity more than zero but less than 0.05
Z	Quantity more than zero but less than 500 where numbers are rounded to thousands
*	Figure does not meet standards of reliability or precision
#	Figure suppressed to comply with confidentiality requirements

Blood Lead Levels for Persons Ages 6 Months–74 Years

by Joseph L. Annest, Ph.D., Division of Health Examination Statistics, National Center for Health Statistics,[a] and Kathryn Mahaffey, Ph.D., Division of Standards Development and Technology Transfer, National Institute for Occupational Safety and Health

Introduction

Human exposure to lead in the U.S. population

The exposure to lead from environmental sources is a public health concern.[1] National estimates of blood lead levels, a common index of lead exposure, for examinees from the second National Health and Nutrition Examination Survey indicated detectable levels of lead throughout the U.S. population.[2–4] A more detailed presentation and comprehensive documentation of national estimates of blood lead levels pertaining to selected demographic and socioeconomic factors, medical history items, and biochemical measures is presented in this report. Quality control and laboratory methods are described and the magnitude of the effect of measurement error on national estimates is evaluated. Details of the statistical analysis of a time trend in national estimates of blood lead levels are presented. Important findings and conclusions from the previous papers[2–4] are highlighted and referenced. Statistics on the distribution of blood lead levels in population subgroups serve (1) to describe the variation in blood lead levels in the U.S. population; (2) to establish baseline estimates for future studies to monitor changes in exposure to lead over time; (3) to provide normative information for use in health policy decisions and for setting standards for the regulation of lead in food, consumer products, gasoline, air, and water; and (4) to determine target populations for lead screening programs.

Public health concerns about exposure to lead

The toxic effects of prolonged exposure to high levels of lead are known.[1] Children with very high blood lead levels (greater than 70 micrograms per deciliter (μg/dl) of whole blood) can suffer permanent renal and neurological damage. In severe cases, lead poisoning can cause encephalopathy, convulsions, coma, and frequently death.[5] Among survivors of severe lead poisoning, significant mental retardation frequently occurs.[6] Health effects of lower levels of lead exposure are more subtle, but significant, including impaired hematopoiesis and neuropsychological deficits in children.[7–9]

Chronic occupational exposure to lead has been shown to cause anemia and peripheral neuropathy; the extent and severity of these effects correlate with observed blood lead levels.[10] However, workers with long-term exposure can have an elevated body-burden of lead, but have blood lead concentrations in the upper portion of the distribution observed among the nonoccupationally exposed population. Occupational lead exposure has also been associated with central nervous system dysfunction and renal impairment, resulting in elevated death rates from kidney disease.[11] Lead may also cause depressed sperm counts.[12]

Lead has toxic effects and serves no apparent useful function in the human body.[13] The detrimental effects of detectable levels of lead in the body are a public health concern, especially in young children during critical periods of physical growth and neurological development. In light of these concerns, this paper focuses on the distribution of blood lead levels in the general population, with emphasis on demographic, socioeconomic, and behavioral associations for young children and other selected population groups.

Factors associated with exposure to lead

The assessment of human variation in blood lead levels depends on an understanding of the environmental sources of lead and the means by which lead enters the body. The most common vehicles for transfer of environmental lead to humans are air, food, water, dust, dirt, and lead-based paint. The most common means of entry into the body are ingestion and inhalation. In the mid-1970's most of the environmental lead in the United States originated from leaded gasoline combustion, industrial emissions, lead-soldered side seam cans for food, and flaking paint chips from houses built before 1950.[1]

Young children are particularly likely to have additional lead exposure from paint, dust, and dirt because of frequent hand-to-mouth activity and the tendency to eat unusual substances (pica).[5] Exposure to lead from paint almost invariably comes from eating lead paint chips or broken, lead-impregnated plaster found in old dilapidated houses. The degree of exposure to lead from dust and dirt depends on the environment in which children reside or play. Sucking dirty fingers, eating with unclean hands, consuming food items dropped on the ground, and mouthing toys or other objects coated with dust or dirt are common practices among toddlers and young children.

Adults also absorb lead from dust and dirt; for example, inhaling lead dust and fumes and ingesting lead dust from fingers, food, and cigarettes.[14,15] The risk of lead toxicity is particularly high for those who work in secondary lead smelters, storage battery plants, scrap metal handling, car repair shops,

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and industries producing lead pigments and lead-soldered stained glass.

Lead usually enters the body by ingestion or inhalation. In young children, clinical studies have shown that approximately 40 to 50 percent of the lead ingested is absorbed from the gastrointestinal tract, while adults absorb about 5 to 10 percent this way.[16] The rate of absorption of airborne lead in relation to age is not as clearly understood. Although percent retention of inhaled lead is influenced greatly by particle size, clinical studies suggest that, in general, 20 to 40 percent of inhaled lead will be deposited in the respiratory tract.[17] However, because of the higher metabolic rates and greater physical activity of children, it is estimated that with comparable exposure, children inhale two to three times as much airborne lead per unit body weight as adults.[18]

Highlights

The following conclusions are derived from data collected by the National Center for Health Statistics:

- Analysis of a chronological trend in the data indicated that average blood lead levels in the United States declined approximately 37 percent (5.4 μg/dl) from February 1976 through February 1980. The results of the trend analysis suggest that the most likely explanation for this decline is a reduction in lead usage in gasoline during the same period.
- National estimates indicated that at the survey midpoint (March 1, 1978) 4.0 percent, or approximately 675,000 of the children 6 months–5 years of age had elevated blood lead levels (30 or more micrograms per deciliter of whole blood). The estimated number of children with elevated blood lead concentrations was two to four times higher than previously predicted by data obtained from the community-based lead poisoning prevention programs.
- The percent of children 6 months–5 years of age with elevated blood lead levels was significantly higher for black children (12.2 percent) than for white children (2.0 percent). Almost one-fifth (18.5 percent) of black children from low-income families had elevated blood lead levels. About one-fifth (18.6 percent) of black children living in the central city areas of large cities with 1 million or more population had elevated blood lead levels.
- A substantial number of children with elevated blood lead levels (30 μg/dl or more) had erythrocyte protoporphyrin (EP) levels less than 50 μg/dl—a finding of importance to lead screening programs.
- Among people 6–17 years of age, mean blood lead levels decreased across successive age groups until late adolescence. Overall, an estimated 0.5 percent of this age group had elevated blood lead levels.
- For people 18–74 years of age, the mean blood lead level of men substantially exceeded that of women.
- Accounting for age, race, smoking, and alcohol consumption, there was a significant positive association between average blood lead level and the degree of potential exposure to lead at the workplace for male and for female workers.
- Approximately 4.1 percent of U.S. children 6 months–5 years of age were reported as having been tested for lead poisoning with more black children (12.5 percent) being tested than white children (2.7 percent). About one-fourth (24.6 percent) of black children living in large urban areas with 1 million or more population were reported as having been tested.
- Some children repeatedly ingest nonfood substances (a behavior called pica) that may be contaminated with lead. During the survey period, 8.1 percent of U.S. children 6 months–5 years of age had a history of eating unusual substances, such as clay, starch, paint or plaster, and dirt. History of pica was more common for black children than white children and for children living in households with annual family incomes less than $10,000 than for children in families with higher incomes.

Survey design and methods

Second National Health and Nutrition Examination Survey (NHANES II) sample design and the lead subsample

The cross-sectional survey was conducted from February 1976–February 1980, with a probability sample of 27,801 persons residing in 64 areas of the United States.[19] National estimates obtained from the data for the 64 areas are representative of the U.S. civilian noninstitutionalized population (including Alaska and Hawaii) 6 months–74 years of age. Certain subgroups of special interest for nutritional assessment were deliberately oversampled. These included children 6 months–5 years, adults 60–74 years, and persons living in low-income areas. The survey included interviews to obtain demographic, medical history, and nutritional information. Medical examinations and numerous laboratory measurements from blood and urine specimens, including blood lead determinations, were also included.

Of the 27,801 sample persons, 16,563 were asked to provide blood specimens for lead level measurements; they included all children 6 months–6 years and a half sample of persons 7–74 years. Blood specimens for assessment of lead concentrations were not obtained on approximately 39 percent of these persons, usually because of nonresponse at various stages of the survey. A detailed description of the investigation of sample persons with missing blood lead data is given in appendix I. The results show no evidence of bias in that nonresponse was distributed uniformly by race, sex, degree of urbanization (of place of residence), and annual family income. The total nonresponse in the lead subsample was greater for young children 6 months–5 years (51.0 percent) than for those 6–17 years (28.6 percent) or 18–74 years (35.7 percent). However, differences in nonresponse by age are taken into consideration in the weighting and poststratification adjustment processes. (See appendix I.)

Blood lead levels were determined for blood specimens from 10,049 examinees. Capillary blood was obtained from 113 children ages 6 months–7 years (mostly from children under 4 years old) by fingerstick (pricking of the fingertip); venous blood was obtained by venipuncture from the remaining 9,936 examinees. Although careful procedures were used during the fingerstick process, the potential for contamination during the capillary blood collection existed.[20] Preliminary analysis of the NHANES II data suggested that including the capillary blood lead data in this analysis would introduce bias in the estimates of mean venous blood lead levels in children. Overall, for children 6 months–5 years, the unweighted mean blood lead level for those receiving fingersticks was observed to be approximately 6.0 μg/dl higher than for those receiving venipunctures. This difference was observed for black and white children. Three additional examinees had elevated venous blood lead levels (76.0, 80.0, and 90.0 μg/dl), which were considered to be the result of atypically high exposure to lead. Therefore, the blood lead values of persons receiving fingersticks and of the three venipuncture cases with extreme lead exposure were excluded from further analysis. (A description of these excluded blood lead values is given in appendix I, table VII.) Thus, in this report, national estimates are based on data obtained on 9,933 NHANES II examinees with venous blood lead levels ranging from 2.0–66.0 μg/dl.

Quality control and laboratory procedures

NHANES II participants were examined at mobile examination centers that were set up at each of the 64 sampling locations so that the environment and equipment would be standard and methods of blood collection and specimen processing would be uniform.

Venous blood specimens were collected in either 5- or 7-milliliter (ml) ethylene diamine tetraacetic acid (EDTA) lavender-top Vacutainer tubes (Vecton-Dickinson Co., Rutherford, NJ). After the blood and anticoagulant were mixed, each sample was placed under a laminar flow hood that provided class-100 air (that is, air containing less than 100 particles per cubic meter of less than 0.5 micrometer diameter) to minimize contamination by dust particles. An aliquot was poured into a Mini-Vial (Packard Instruments, Downers Grove, Ill.). These aliquots (and those in capillary tubes) were then frozen within 1 hour after collection and were shipped in Dry Ice to the Centers for Disease Control (CDC) for analysis. Samples remained frozen until analyzed. All tubes and vials used for collection and storage were from production lots that had been screened previously at CDC for lead contamination.

All laboratory determinations of blood lead levels from these blood samples were performed by the Clinical Chemistry Division, CDC, Atlanta, Georgia, and financed by the Division of Nutrition, Bureau of Foods, Food and Drug Administration, Cincinnati, Ohio. Descriptions of the material, methods, and quality control procedures are presented in detail elsewhere.[21,22] Brief descriptions of the laboratory procedures and quality control methods are presented in the following paragraph to demonstrate the precision and accuracy of the measurements.

Lead concentrations of sample and control specimens were determined with a modified Delves cup atomic absorption micromethod.[23,24] A Perkin-Elmer Model 360 atomic absorption spectrometer equipped with a three-slot burner, microcombustion assembly, lead hollow-cathode lamp, and deuterium arc background corrector was used. The instrument parameters and settings were as follows: wavelength, 283.3 nanometers; lamp current, 10 milliamperes; slit, 0.7 nanometers (normal mode); signal, TC–1 (time constant–1); and operating mode, ABS (absorbance). The reagents and calibration procedures were the same for analysis of all blood samples. Contamination in the laboratory was minimized by handling blood samples under hoods that provided class-100 air.

Blood specimens were analyzed in duplicate, using 10-μl sample volumes. When replicate absorbance values differed by more than 0.025 absorbance or the difference between calculated lead concentrations for duplicates was greater than 7μg/dl, analysis was repeated. The results of the repeat analysis were reported. Out of the approximately 10,000 whole blood lead samples, 458 were repeated because duplicate measurements differed by more than 7 μg/dl. Thirty-six of these 458 had duplicate measurements that differed by more than 25 μg/dl and clearly were cases of a problem such as a microclot, a pitted cup, or a misaligned cup. The remaining 422 samples had a difference between duplicate determinations greater than 7 μg/dl either from one of the previously mentioned sources or from random sources of error typically associated with analytical procedures (for example, pipetting and instrument noise).

Two quality control systems, using bovine whole blood pools, were used. (Blood used in preparation of quality control samples was collected by venipuncture from cows administered lead nitrate capsules orally.) These two systems were (1) "bench" quality control samples that the analyst inserted and measured two–four times in each analytic run for technical evaluation on the day of analysis, and (2) "blind" quality control specimens placed in vials, labeled, and processed to be indistinguishable from regular NHANES II samples. The results were decoded and reviewed by the quality control supervisor. At least one "double blind" sample was randomly inserted and analyzed in duplicate with 20 NHANES II samples (in a given run). If the average of replicate values of either bench or blind quality control samples fell outside their respective previously established 95 percent confidence limits, the run was repeated.

Bench quality control samples were collected in 5-ml lavender-top EDTA Monoject (Sherwood Medical Industries, St. Louis, MO) vacuum collection tubes (from the same production lots as those used to collect NHANES II samples) and stored at -20° C; approximately 100 tubes were drawn at each sampling. Two levels of bench pools were maintained: one containing a lead concentration within the normal range, the other an abnormally high level (greater than 30 μg/dl). New pools were prepared at approximately 6-month intervals, allowing a 2-month overlap between new and old pools to insure standardization of measures over time.

The blind quality control system was based upon two large pools that were prepared in sufficient amounts to cover the entire duration of the survey. Because of initial technical difficulties in labeling the blind specimens (to be indistinguishable from the NHANES II specimens), the two blind pools were analyzed with the NHANES II sample specimens collected from March 1977 through the end of the survey. The lead levels were in the low normal range for one pool and near the decision point at 30 μg/dl for the other pool. Because results showing concentrations equal to or greater than 30 μg/dl were phoned to the personal physician of the examinee, an elevated blind pool could not be used. The blood was collected in sterile 500-ml bottles containing 1.5 milligrams per milliliter (mg/ml) K_3EDTA as the anticoagulant. Blood from several bottles was pooled, mixed, and dispensed into vials identical to those used in the field to process the examinees' blood lead samples. The vials were labeled with pseudopatient numbers corresponding to each geographic location and stored at -20° C.

The normal blind pool with a mean of 13.7 μg/dl had a standard deviation (SD) of 2.2 μg/dl (0.022 parts per million (ppm)) while the high blind pool with a mean of 25.5 μg/dl had an SD of 3.2 μg/dl (0.032 ppm). The coefficient of variation (that is, the standard deviation expressed as a fraction of the mean blood lead level for a given pool) for the bench quality controls having blood lead levels of 30.0 μg/dl or more ranged from 7.0 to 15.0 percent.[21]

Further information on the quality control data and an evaluation of measurement error are presented in appendix II. The potential effects of misclassification error on national prevalence estimates of elevated blood lead levels (30 μg/dl or more) are evaluated. The statistical analysis indicates that accounting for measurement error in blood lead assessments could reduce the estimate of overall prevalence of elevated blood lead levels up to 24 percent.

Statistical methods

Statistics are provided for population subgroups defined by factors previously reported to be associated with blood lead levels in the general population.[2,3,18,25–27] Categories were defined to provide stable population estimates and to test significant differences between population subgroups of importance for public health programs concerned about exposure to environmental lead. Statistics are not presented for further cross-classifications or smaller breakdowns of the data because of unreliability of the estimates or their standard errors.

The statistical analysis of the weighted NHANES II blood lead data took into account the complex survey sample design; that is, the clustering and oversampling of selected groups.[19] The variances were computed using SESUDAAN, a computer program based on a Taylor linearization procedure that provides consistent estimates of standard errors of means and of proportions.[28] The analysis of blood lead levels as a continuous variable was carried out using SURREGR, a regression program that provides consistent estimates of standard errors for regression coefficients.[29] The analysis of blood lead level as a categorical variable was carried out using the variance-covariance matrix produced by SURREGR as input to GENCAT, a program for generalized least-squares categorical data analysis.[30,31]

All differences stated in the text were conducted using the variance-covariance matrix. Tests of significance were con-

ducted using a probability level of 0.05 unless otherwise indicated. Further details on statistical tests and measure of variability are in appendix I.

In the next section, distributions of blood lead levels in the U.S. population are presented by age, race, sex, annual family income, and degree of urbanization (of place of residence) for children, youths, and adults. In addition, statistics on blood lead levels are given by education of the head of the household, history of pica, and history of tests of lead poisoning for children, and by occupation, history of smoking, and alcohol consumption for adults. The definitions of terms and variables and the history questions are in appendix III.

Findings

Blood lead levels by age, race, and sex

Average blood lead level estimates for the U.S. population differ substantially with respect to age, race, and sex (figures 1 and 2 and tables A and 1–4). Differences in blood lead levels associated with these factors in the general population were the result of differences in exposure to environmental sources of lead or in the absorption, retention, or excretion of lead in the blood, or both. However, the relative contributions of these factors to blood lead level in the general population have not been determined.[1]

For young children 6 months–5 years, mean blood lead levels were significantly higher for black children than for white children (figure 1, table 4). The higher blood lead levels in black children than in white children have been noted elsewhere.[32] Accounting for differences associated with race, the differences between means with respect to age and sex were not statistically significant. The lack of association between age and blood lead level for children 6 months–5 years of age is somewhat of a surprise because other studies in the United States[32] and Europe[33,34] indicate a peak in average levels at 2 to 3 years of age.

Overall for children and youths 6–17 years, mean blood lead levels decreased significantly as age increased (figure 2). A similar decline in blood lead levels from 6 years to later adolescence has been noted in the United Kingdom.[33,34] Accounting for the effects of age, race and sex differences were significant. Generally, as age increases, the difference in mean blood lead levels between boys and girls increases, with those of boys consistently higher than those of girls. The average blood lead levels are also significantly higher for black persons than for white persons in this age range.

For adults 18–74 years, there were significant differences in blood lead levels associated with age, race, and sex. The sex difference was the most pronounced, with the mean blood lead level consistently higher for men than for women (figure 2). Accounting for differences related to sex and age using regression analysis, blood lead levels were, on the average, higher for black adults than for white adults (figure 1).

Blood lead levels by income and degree of urbanization

The associations of family income and of the degree of urbanization with blood lead levels were generally consistent across all ages with higher mean blood lead levels among the

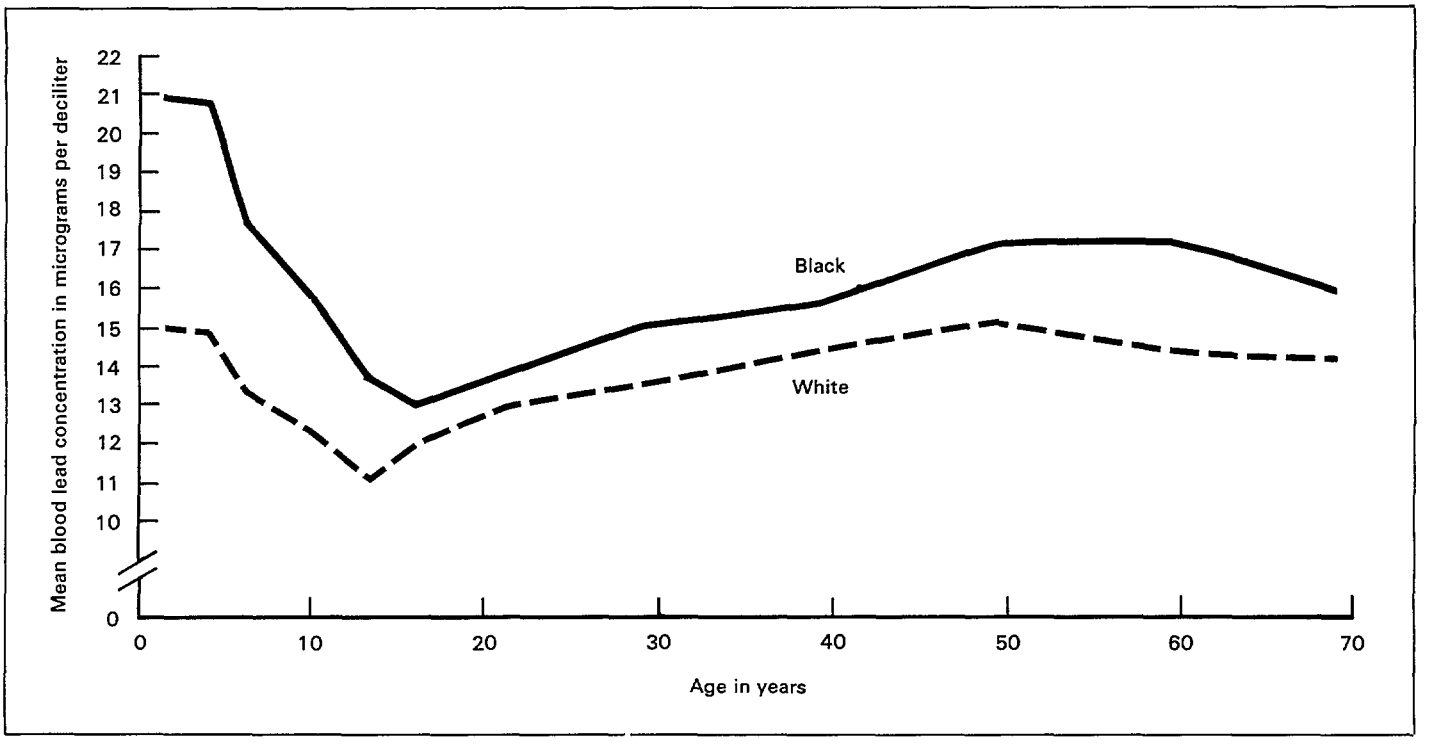


Figure 1. Blood lead levels by race and age: United States, 1976–80

Table A. Number of children examined, geometric mean and standard deviation of blood lead levels and percent of children 1–5 years with values ≥30 μg/dl, by race and age: United States, 1976–80

Race and age	Number examined	Geometric mean[1]	Standard deviation of the mean[1]	Estimated percent with PbB ≥ 30 μg/dl[1]	Standard error of the percent
White					
1 year	211	14.1	1.49	2.8	1.04
2 years	296	14.3	1.45	2.8	1.00
3 years	400	14.3	1.44	2.9	0.68
4 years	434	13.8	1.44	1.8	0.64
5 years	453	13.9	1.40	0.8	0.37
Black					
1 year	55	20.4	1.51	18.2	6.43
2 years	68	20.5	1.45	16.8	3.66
3 years	74	21.3	1.47	18.1	3.82
4 years	103	20.0	1.39	11.1	2.58
5 years	101	18.2	1.41	4.6	1.43

[1]Estimated from the weighted NHANES II data obtained on examinees with blood specimens drawn by venipuncture.

NOTE: ≥ = equal to or greater than.
μg/dl = micrograms per deciliter.

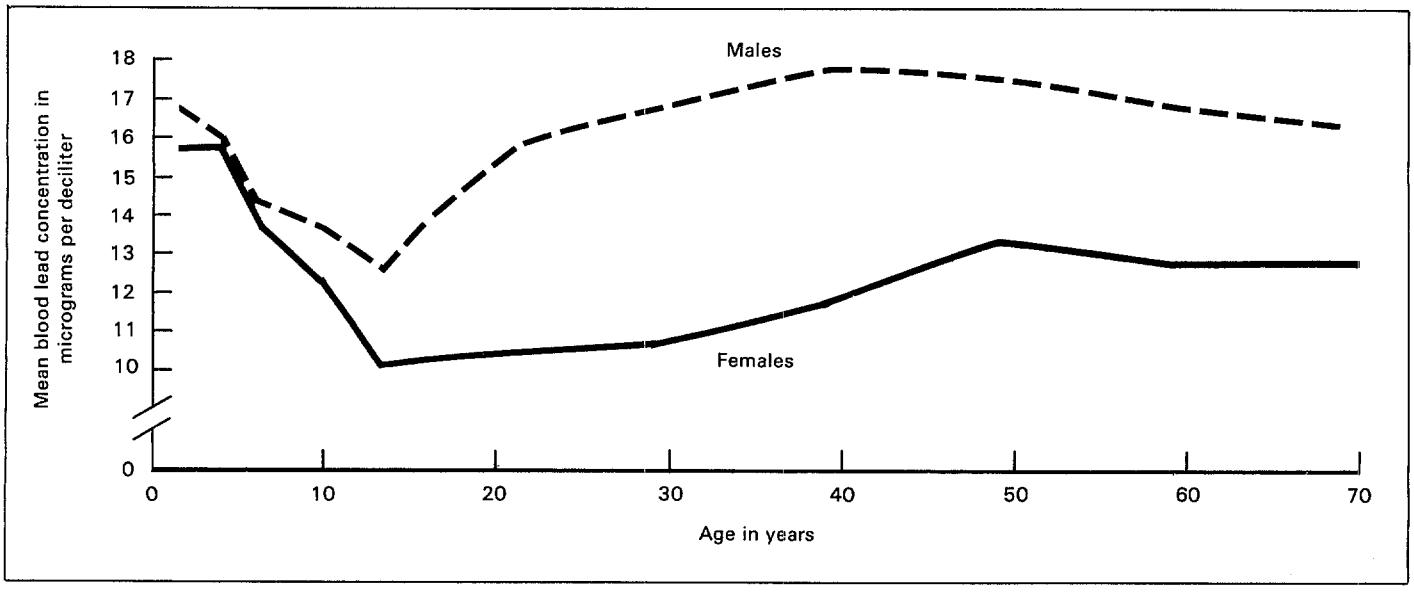


Figure 2. Blood lead levels by sex and age: United States 1976–80

poor than the more affluent and for those in urban than in rural areas (tables 5–7). These associations were most pronounced, however, in children ages 6 months–5 years (figures 3–5). Accounting for variation in blood lead levels associated with age, sex, and race by regression, there was a statistically significant inverse relationship between mean blood lead levels in children and family income. Similar analysis indicates that mean blood lead levels were higher for children living in large urbanized areas than for those living in smaller urban or rural areas.

Mean blood lead levels for black children were significantly higher than those for white children across all three income groups and the two urban groups. A similar racial difference in mean blood lead levels was observed in the rural group (figure 4), but it was not statistically significant, probably because of the relatively small number of rural black children in the sample. This consistent difference between black and white children suggests that higher blood lead levels for black children are not fully explained by exposure to lead-laden sources commonly identified with children living in deteriorating housing in the inner cities (urban lead belt). These findings are consistent with those of other studies regarding this racial difference.[32,35,36]

Within the central cities of large urban areas with 1 million or more population, the mean blood lead level of black children was significantly higher than that of white children. Other studies[18] indicate that exposure to lead in central city children may be associated with socioeconomic factors. It is estimated from the NHANES II data that 43 percent of black children compared with 22 percent of white children living in the central city areas were from households with annual family incomes under $6,000 during the year preceding the time of interview. In 1978 (the midpoint of the survey was March 1, 1978), the income level of $6,000 was near the poverty threshold for a

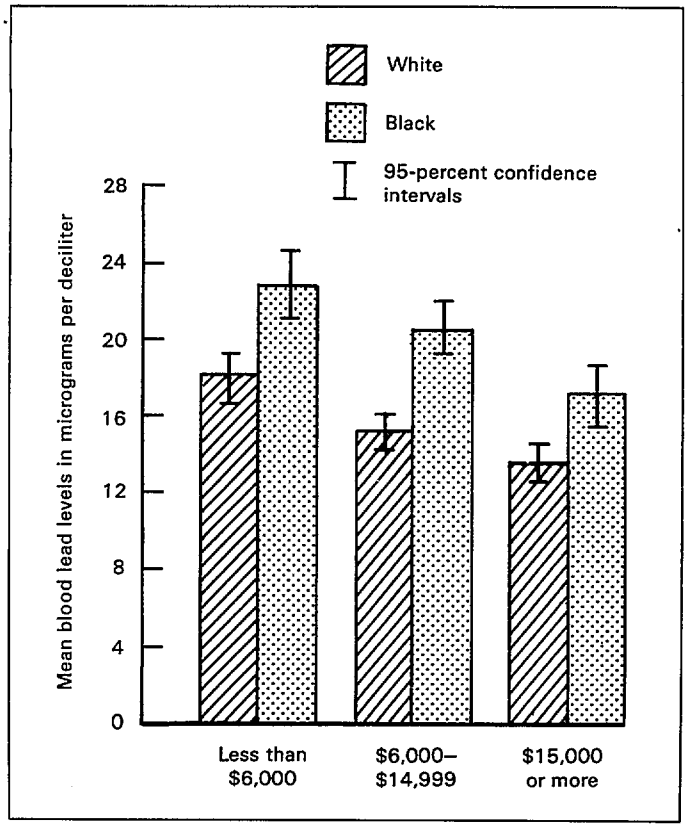


Figure 3. Blood lead levels of children 6 months–5 years by race and annual family income: United States, 1976–80

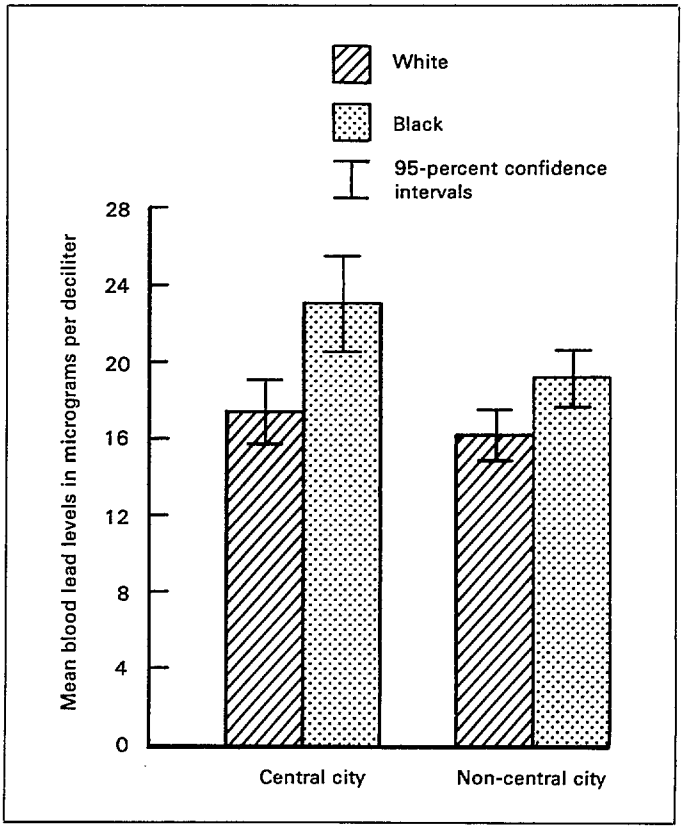


Figure 5. Blood lead levels of children 6 months–5 years in large urban areas by race and location: United States, 1976–80

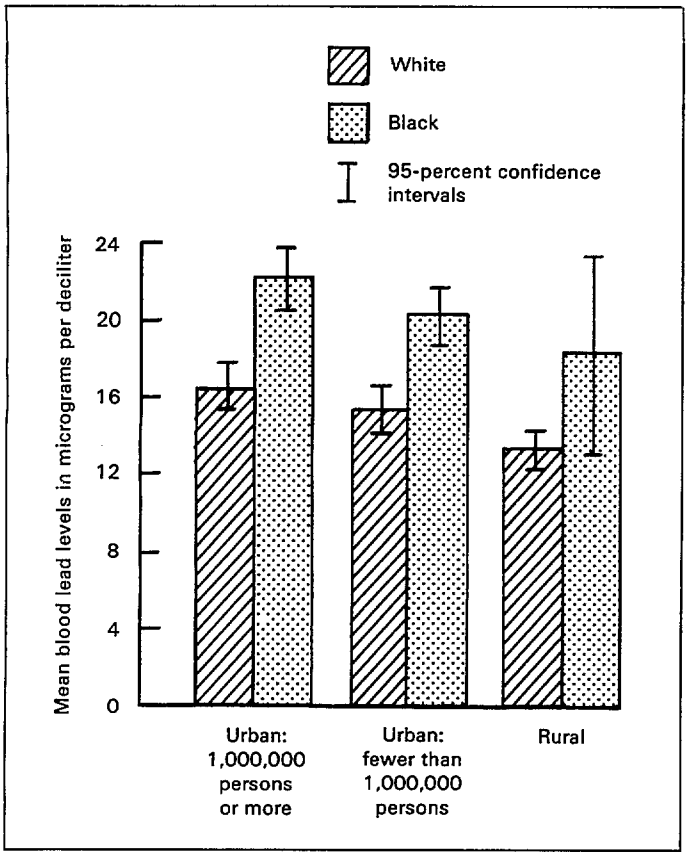


Figure 4. Blood lead levels of children 6 months–5 years by race and degree of urbanization: United States, 1976–80

family of four as determined by the U.S. Bureau of the Census.[37]

Mean blood lead levels of black children living in the central cities were observed to be higher than those of black children living in urban areas outside the central cities and rural areas, respectively. These differences were neither statistically significant nor reliable because of the small number of black children in the sample who were living outside the central city.

Attempts to cross-classify by degree of urbanization and income using the NHANES II data resulted in samples too small to provide reliable estimators. For example, while it would have been of interest to determine whether the association between race and blood lead level differed between various degrees of urbanization by income groups, the number of examinees within such groups was too small.

Elevated blood lead levels in children

The consistent difference in mean blood levels between black and white children ages 6 months–5 years and the higher blood lead levels among children from low-income families or residing in large urban areas was also apparent from the percent of children with elevated blood lead levels (that is, equal to or greater than 30 μg/dl) (table B). In accordance with Centers for Disease Control (CDC) guidelines published in 1978,[38] 30 μg/dl is the cutoff used in many of the community-based lead screening programs for referring children for further clinical diagnostic evaluation.

Based on the CDC guidelines (30 μg/dl or more), an analysis of NHANES II data indicates that 4.0 percent or approx-

Table B. Percent and standard error of the percent of children 6 months–5 years with blood lead levels above selected levels, by race, sex, annual family income, and degree of urbanization: United States, 1976–80

Demographic variable	*All races*[1]			*White*			*Black*		
	20 μg/dl or more	*25 μg/dl or more*	*30 μg/dl or more*	*20 μg/dl or more*	*25 μg/dl or more*	*30 μg/dl or more*	*20 μg/dl or more*	*25 μg/dl or more*	*30 μg/dl or more*
	Percent of children[2,3]								
Both sexes	24.5	9.1	4.0	18.1	5.5	2.0	52.2	24.5	12.2
Boys	25.6	9.7	4.4	19.4	6.0	2.1	48.5	24.8	13.4
Girls	23.4	8.4	3.5	16.6	4.9	1.8	56.2	24.1	10.9
Annual family income									
Under $6,000	45.5	21.8	10.9	33.1	14.0	5.9	61.0	34.2	18.5
$6,000–$14,999	26.4	8.8	4.2	21.1	5.6	2.2	51.1	23.6	12.1
$15,000 or more	13.4	4.4	1.2	10.5	3.3	0.7	36.5	8.0	2.8
Degree of urbanization of place of residence									
Urban, 1 million persons or more	36.2	14.9	7.2	28.3	9.8	4.0	59.1	28.5	15.2
Central city	48.2	22.2	11.6	35.2	12.1	4.5	63.3	32.7	18.6
Non-central city	27.0	9.2	3.7	25.3	8.8	3.8	44.9	*14.1	*3.3
Urban, fewer than 1 million persons	28.0	9.5	3.5	20.6	5.7	1.6	51.4	22.2	10.2
Rural	11.5	4.2	2.1	9.5	2.7	1.2	29.9	*19.4	*10.3
	Standard error of the percent								
Both sexes	2.2	1.0	0.5	2.3	0.8	0.3	3.8	2.9	1.5
Boys	2.6	1.3	0.7	2.6	1.0	0.5	4.7	4.3	2.0
Girls	2.2	0.9	0.5	2.3	1.0	0.4	4.0	4.3	2.4
Annual family income									
Under $6,000	3.2	2.7	1.4	4.0	2.7	1.3	4.3	5.9	3.6
$6,000–$14,999	2.8	1.1	0.7	3.0	1.1	0.5	4.2	2.8	1.9
$15,000 or more	1.9	1.3	0.4	2.0	1.1	0.3	6.4	3.1	1.2
Degree of urbanization of place of residence									
Urban, 1 million persons or more	3.5	2.1	0.7	4.4	2.3	0.7	4.9	3.5	1.5
Central city	4.1	3.4	1.9	6.7	3.4	1.9	5.8	6.2	2.8
Non-central city	3.9	2.0	0.8	4.1	2.4	0.8	8.0	*3.8	*1.4
Urban, fewer than 1 million persons	4.3	1.7	0.6	4.1	1.3	0.4	5.6	4.5	2.4
Rural	2.6	1.4	0.9	2.3	0.8	0.5	11.1	*10.1	*5.3

[1]Includes data for races not shown separately.
[2]The one child (a black male, family income under $6,000, in a rural area) with an excessively high blood lead level (76.0 μg/dl) was excluded. This exclusion has a negligible effect on the national estimates shown here.
[3]Estimated using data on blood lead levels determined from specimens drawn by venipuncture.

NOTE: μg/dl = micrograms per deciliter.

imately 675,000 U.S. children 6 months–5 years of age had elevated blood lead levels in the late 1970's. (This estimate was two to four times higher than previously predicted from data obtained in the CDC lead poisoning prevention programs for children 1–5 years.)[39] Among children of this age, 12.2 percent of black children compared with 2.0 percent of white children had blood lead levels of 30 μg/dl or more. This difference was significant for boys and for girls. The percent with elevated blood lead levels was observed to be slightly higher in boys than girls, but this difference was not statistically significant at the 0.05 level of probability.

The proportion of children with elevated blood lead levels decreased with increased family income. This relationship was significant for black and for white children. The highest percent of elevated blood lead levels (18.5 percent) was found among black children from low-income families. For both white and black children, the percent of persons with elevated blood lead levels was lowest in the highest income group.

For children living in the central cities of large urban areas with 1 million or more population, the percent of children with elevated blood lead levels was significantly higher for black than for white children. Even in the smaller urban (less than 1 million persons) and rural areas, more than 10.0 percent of black children was observed to have elevated blood lead levels compared with less than 2.0 percent for white children. Caution should be exercised in interpreting racial differences in rural areas because of the relatively small number of examined persons (42 cases) in the estimation cell for rural black children.

Recently, concern has been expressed that a cutoff of 30 μg/dl may not provide a reasonable margin of safety for children 5 years of age or younger. Studies suggest that even at blood lead levels between 10 to 20 μg/dl, lead can impair in-

tracellular functions[40,41] and is toxic to the developing brain and nervous system.[42,43] Detection of impairment at these levels has significant public health implications because of the sizable portion (20.5 percent) of children who have blood lead levels between 20 and 30 μg/dl.

The percent of children 6 months–5 years with blood lead levels greater than 25 and 20 μg/dl is shown in table B. If the cutoff used for screening children for lead toxicity were lowered from 30 to 25 μg/dl, approximately 1 out of 4 (24.5 percent) black children and approximately 1 out of 20 (5.5 percent) white children would be considered as having elevated blood lead levels. If it were lowered to 20 μg/dl, about one out of two (52.2 percent) black children and one out of five (18.1 percent) white children would be considered as having elevated blood lead levels.

Blood lead levels of children in association with other selected factors

Mean blood lead levels for those in early childhood differed by the educational attainment of the head of the household (figure 6 and table 8). Regression analysis indicates that there was a significant inverse relationship between mean blood lead level and the education of the head of the household for black and white children 6 months--5 years. This relationship was not present for children 6–17 years.

The reported association of hand-to-mouth activity to blood lead level in early childhood[44–46] was supported by the NHANES II data. Accounting for race using regression analysis, mean blood lead values were significantly higher (approximately 3 μg/dl more) for children 6 months--5 years who were reported as eating unusual substances than for those of the same age for whom no such behavior was reported (table 9). The average blood lead value for these children was much less than that observed among children with overt lead toxicity secondary to eating paint chips high in lead.

The percent of young children with a history of pica was significantly higher for those 6 months--3 years old (11.0 percent) than for those 4--5 years old (3.2 percent) and for children living in households with annual family incomes less than $10,000 (11.9 percent) than for those in households with incomes equal to or greater than $10,000 (6.0 percent) (table 10). Also, the percent of children with pica reported is significantly higher for children with blood lead levels equal to or greater than 20 μg/dl than for those with values less than 20 μg/dl.

Among children 6 months--5 years, 4.1 percent were reported as having been tested for lead poisoning (table 11). Black children (12.5 percent) were more likely to have been tested than white children (2.7 percent). Approximately 1 out of 4 black children (24.6 percent) living in large urban areas (1 million or more population) reported being tested. The mean blood lead level was significantly higher for those tested than for those not tested for lead poisoning (table 9). This latter finding could be expected during the late 1970's because lead screening programs primarily targeted children at high risk of exposure to lead; for example, those living in old, dilapidated households in the inner cities.[38]

The results of tests for lead poisoning are not presented as national estimates because of small sample size. However, a

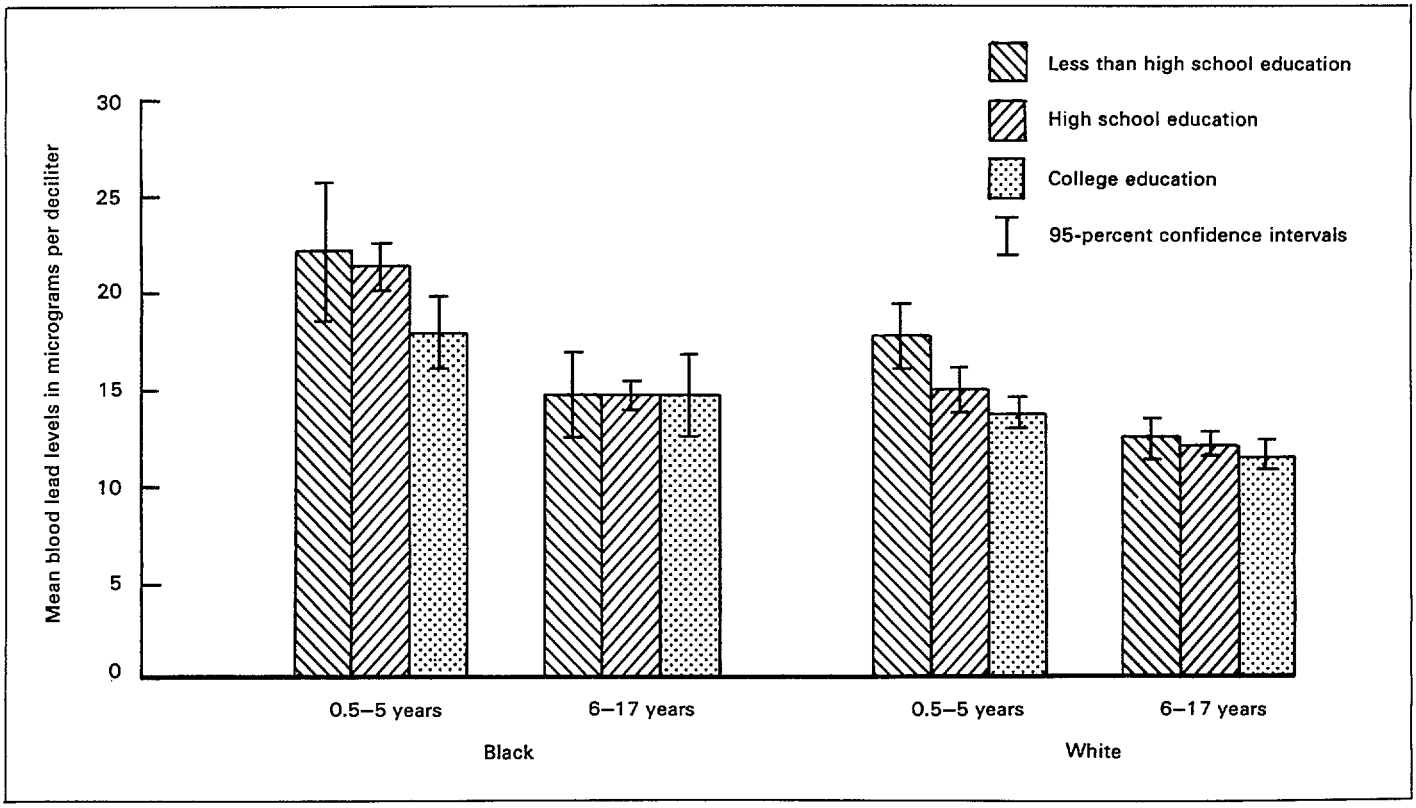


Figure 6. Blood lead levels of children and youths 6 months–17 years by education of the head of household, race, and age: United States, 1976–80

summary of the results of these tests in relation to blood lead level for examinees 6 months--5 years is presented in table VIII of appendix I.

The effects of alcohol consumption, smoking, and occupation on blood lead levels of men and women

Recent studies show that alcohol consumption, cigarette smoking, and occupational exposure to lead can influence the blood lead levels of adults.[25-27] However, there has been concern that the effects attributed to one such factor may be confounded by one of the other factors, making the results of some studies difficult to interpret.

An analysis of the NHANES II data indicated that smoking and certain occupations were associated with higher mean blood lead levels among U.S. workers.[47] Regression analysis suggests that the effects of drinking alcohol, smoking, and occupational exposure to lead can contribute independently and additively to blood lead level (figure 7 and tables 12 and 13).

For persons ages 18--74 years, accounting for differences associated with age and race by regression, the mean blood lead level was significantly greater for men and women who smoke cigarettes than for nonsmokers. The average blood lead level also increased significantly with the number of cigarettes smoked. Mean blood lead level was significantly higher for examinees who reported drinking alcohol (beer, wine, and liquor) than for nondrinkers. There was also a positive association between mean blood lead level and number of alcoholic drinks consumed for men and women (table 12).

For U.S. workers 18--74 years, regression analysis indicated that blood lead levels were significantly associated with

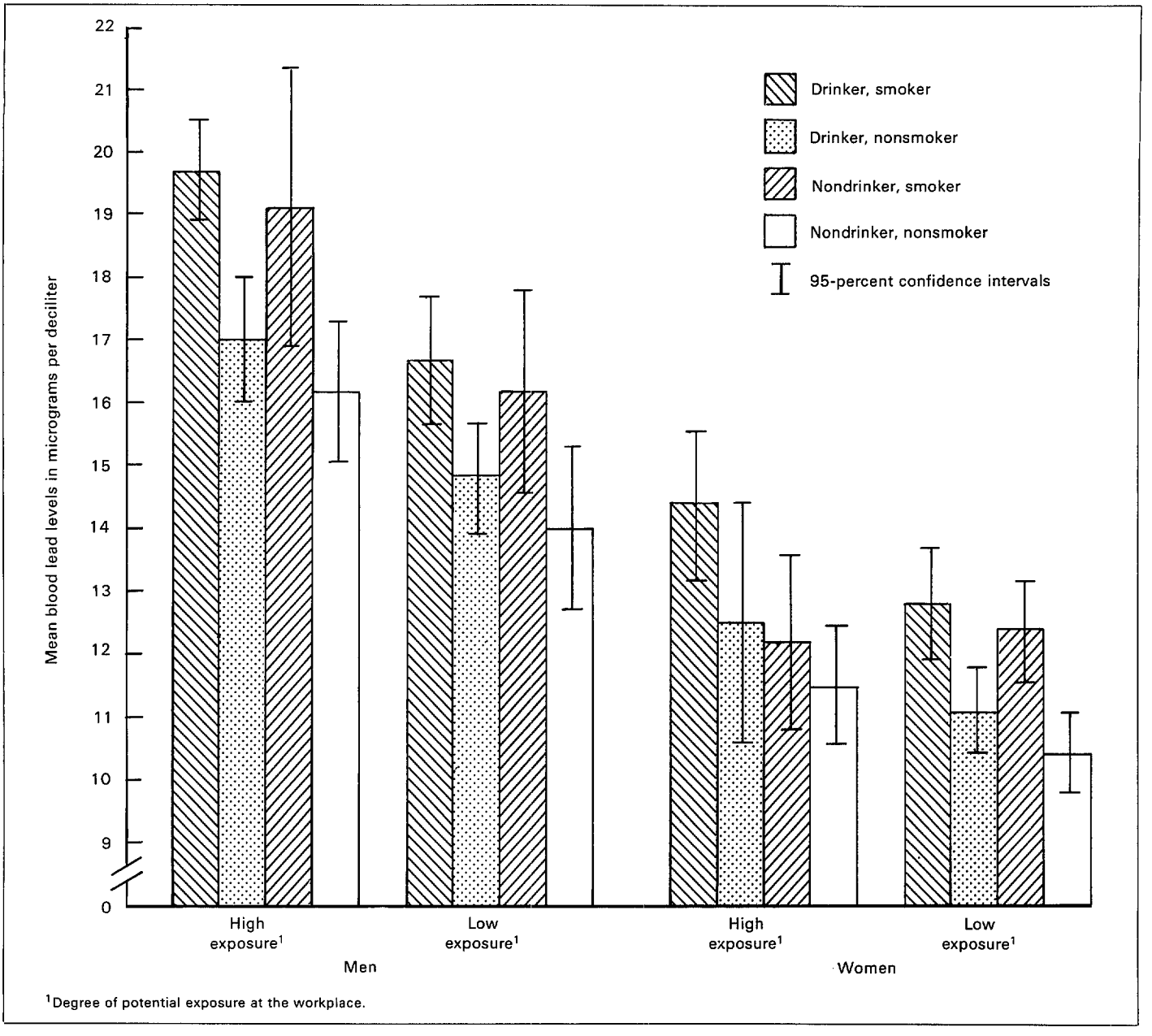


Figure 7. Blood lead levels of persons 18–74 years by sex, degree of potential occupational exposure to lead, alcohol consumption, and smoking status: United States, 1976–80

sex, race, alcohol consumption, smoking, and the degree of potential exposure to lead at the workplace. For the latter, occupational categories were defined using data previously collected during the National Occupational Hazard Survey[48] conducted by the National Institute for Occupational Safety and Health from 1972–1974. (For details on definitions of occupational categories, see appendix III.) Accounting for differences associated with race, age, alcohol consumption, and smoking, the mean blood lead level was significantly higher for the high-potential exposure (to lead at the workplace) group than for the low-potential exposure group for men and for women (table 13). In the regression analysis, there was a significant interaction between occupation and race for men, but no significant interactions between the independent variables for women. Based on the NHANES II data, the analysis indicated that the effects of alcohol consumption, smoking, and exposure to lead at the workplace were additive.

Chronological trend in blood lead levels

Analysis of a chronological trend in the NHANES II data indicates that average blood lead levels in the United States dropped approximately 37 percent from February 1976 through February 1980 (figure 8).[49,50] This trend cannot be attributed to errors in laboratory measurement or the survey sample design. After accounting for differences in race, sex, age, region of the country, season, income, and degree of urbanization, the predicted mean blood lead level from the regression model decreased over the survey period from 14.6 to 9.2 μg/dl of whole blood (a drop of 5.4).[4] Similar analyses were conducted for subgroups defined by race, sex, and age and showed statistically significant reduction in average blood lead levels over the 4-year period ranging from 31 to 42 percent (figure 9).

Changes in general exposure to lead from environmental sources were also investigated in relation to the downward trend in blood lead levels.[4] Only three major factors could have accounted for the downward trend: an increased public awareness of lead sources and lead toxicity, a decreasing amount of lead in the diet, and a reduction in the use of lead in gasoline. There was no evidence that exposure to lead in paint or in the diet, or that factors associated with general public awareness could explain the drop in blood lead levels. However, the correlation of blood lead levels with national estimates of the amount of lead used in gasoline production was highly significant ($p < 0.001$) overall and in population subgroups defined by race, sex, and age (table C). Although strong correlation does not prove cause and effect, the most likely explanation for the decline in blood lead levels is a reduction of lead usage in gasoline during the 4-year period.[4] Details of the statistical analysis are presented in appendix IV.

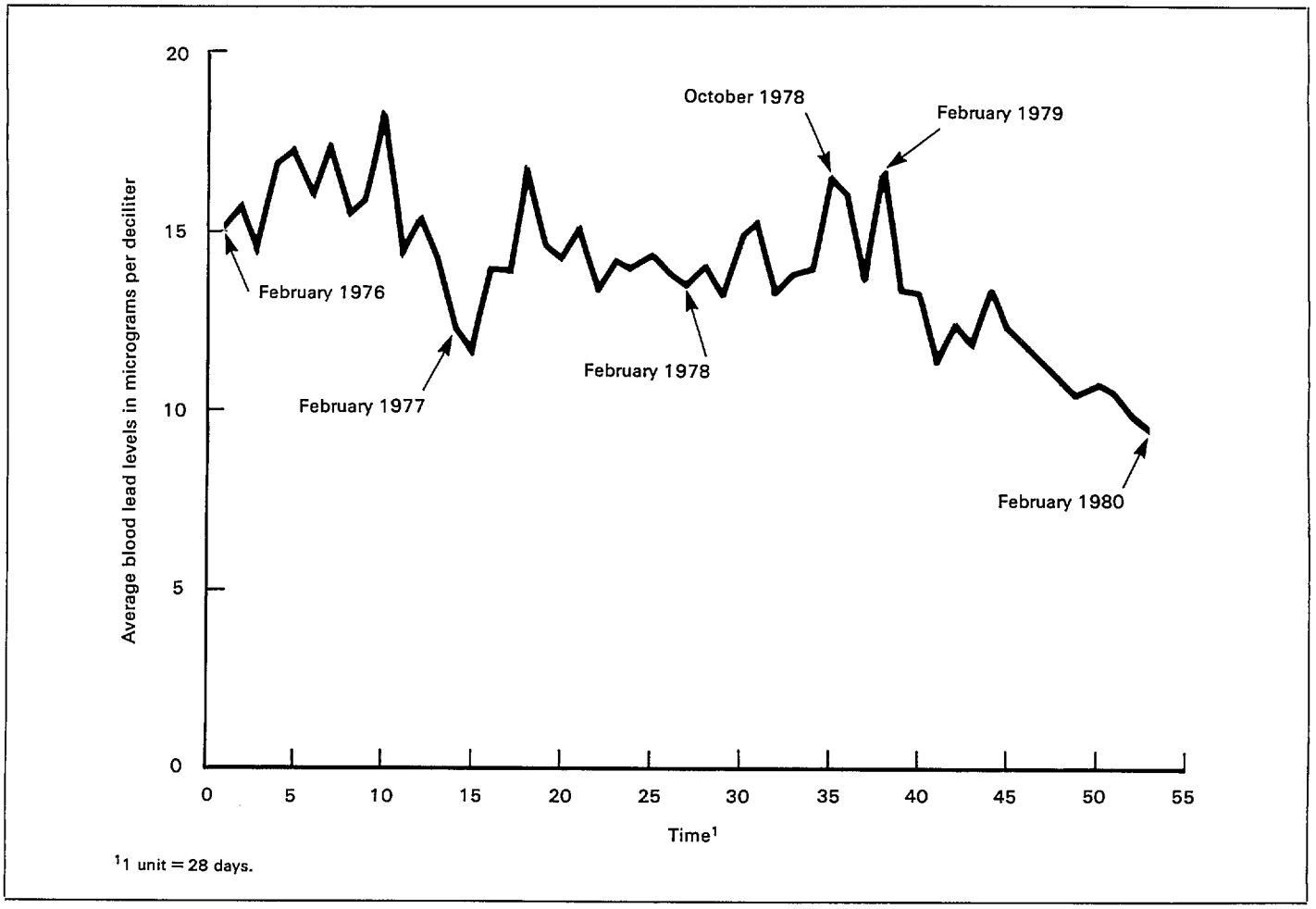


Figure 8. Chronological trend in average blood lead levels for persons 6 months–74 years: United States, 1976–80

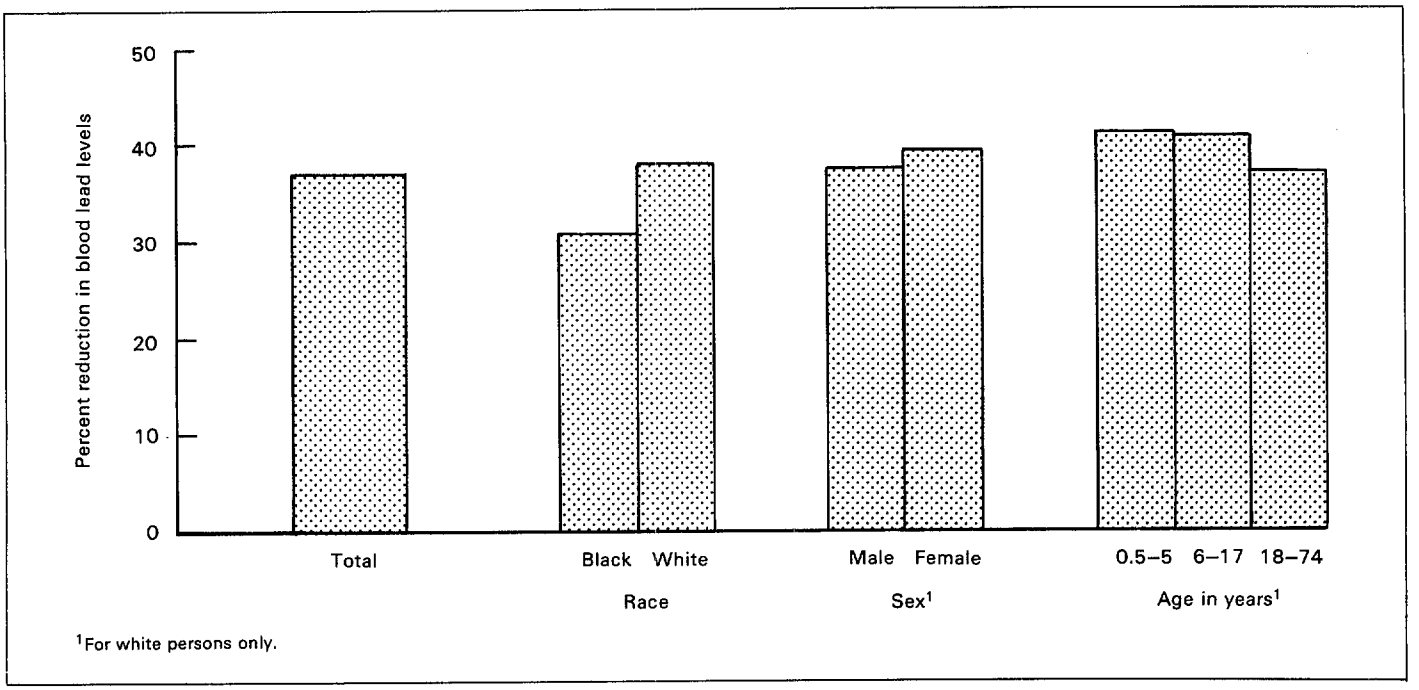


Figure 9. Reduction in mean blood lead levels for persons 6 months–74 years by race, sex, and age: United States, 1976–80

Table C. Pearson correlation coefficients between the average blood lead levels for 6-month periods and the total lead used in gasoline production per 6 months and the averages of the coefficients, by selected characteristics: United States, 1976–80

Characteristic	Coefficient for 6-month periods[1,2]		
	January–June and July–December[3]	April–September and October–March[4]	Average
All races	0.920	0.938	0.929
Blacks[5]	0.678	0.717	0.698
Whites	0.929	0.955	0.942
Sex			
Male	0.944	0.960	0.952
Female	0.912	0.943	0.928
Age			
0.5–5 years	0.955	0.969	0.962
6–17 years	0.908	0.970	0.939
18–74 years	0.920	0.924	0.922

[1]The lead values used to compute the averages were preadjusted by regression analysis to account for the effects of income; degree of urbanization; region of the country; season; and, when appropriate, race, sex, and age.
[2]All correlation coefficients were statistically significant ($P < 0.001$) except those for blacks ($P < 0.05$).
[3]Averages were based on 6-month periods, except for the first and last, which covered only February 1976 through June 1976 and January 1980 through February 1980, respectively.
[4]Averages were based on 6-month periods, except for the last, which covered only October 1979 through February 1980.
[5]Blacks could not be analyzed according to sex and age subgroups because of inadequate sample sizes.

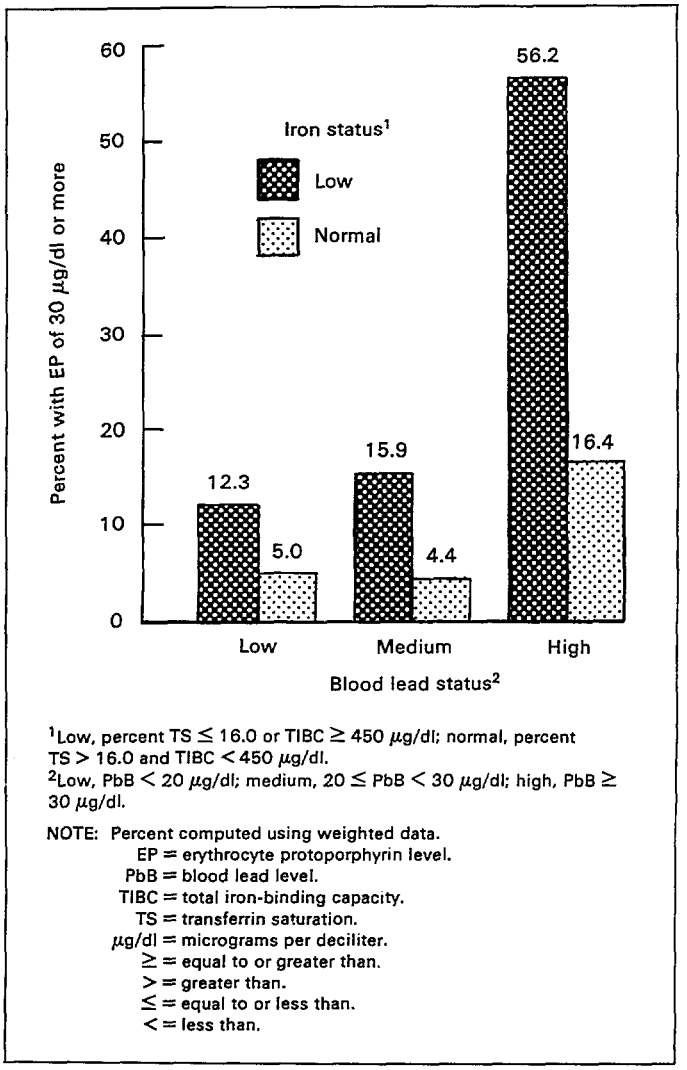
NOTE: < = less than.

An independent study of the chronological trend in the NHANES II blood lead data conducted by Janney et al.[51] reported similar findings. These studies[4,51] and their conclusions have been reviewed for the U.S. Environmental Protection Agency by an expert panel of statisticians.[52] The panel reported the existence of "strong evidence that there was a substantial decline in the average level of blood lead in the U.S. population during the NHANES II survey period" and concluded that there was "a strong correlation between gasoline lead usage and blood lead levels. In the absence of scientifically plausible alternative explanations, the hypothesis that gasoline lead is an important causal factor for blood lead levels must receive serious consideration."

The association of erythrocyte protoporphyrin with blood lead level and iron status

During the last decade, the measurement of erythrocyte protoporphyrin (EP) has been increasingly used in screening young children for lead toxicity and for iron deficiency,[38] and in identifying adults with occupationally related exposure to lead.[53] Erythrocyte protoporphyrin combines with iron to form heme, a constituent of hemoglobin. It is elevated in iron deficiency anemia and lead toxicity. The accumulation of EP can result from the adverse metabolic effects of lead on heme synthesis and from iron deficiency.[54] Individuals who have both iron deficiency and elevated blood lead levels could be particularly susceptible to the biological effects of lead.[55]

Analysis of the NHANES II data suggested that both low iron status (percent transferrin saturation less than 16.0 or total iron-binding capacity greater than or equal to 450 μg/dl serum) and elevated blood lead levels (≥ 30 μg/dl) were associated with increased EP levels in the U.S. population 6 months–74 years. The percent of persons with elevated EP levels (that is, above the 95th percentile, which was 30 μg EP/dl of whole blood in the NHANES II data) was highest among those with low iron status and elevated blood lead levels (figure 10). The cumulative percent distribution of EP by each of six blood lead-iron status categories is shown in figure 11. Comparisons among these groups indicated that the entire distribution of EP was influenced by the effect of blood lead and iron status. The median EP was more than 10 μg/dl higher for the high-blood-lead and low-iron-status group (32.7 μg EP/dl of whole blood) than for any of the other five groups. For the lead screening programs, the interrelated effects of iron status and blood lead level on EP level are an important consideration in evaluating the current criteria for identifying children or adults with excess body burdens of lead using EP tests and blood lead analyses.[55] The relationship of EP and blood lead levels of children 6 months–5 years of age was examined to provide information that may be helpful in evaluating the criteria involved in using EP levels in lead screening programs. As shown in table D, about 20 percent of the examined children with blood lead values of 30 μg/dl or more had EP levels of 50 μg/dl or more.



[1]Low, percent TS ≤ 16.0 or TIBC ≥ 450 μg/dl; normal, percent TS > 16.0 and TIBC < 450 μg/dl.
[2]Low, PbB < 20 μg/dl; medium, 20 ≤ PbB < 30 μg/dl; high, PbB ≥ 30 μg/dl.

NOTE: Percent computed using weighted data.
EP = erythrocyte protoporphyrin level.
PbB = blood lead level.
TIBC = total iron-binding capacity.
TS = transferrin saturation.
μg/dl = micrograms per deciliter.
≥ = equal to or greater than.
> = greater than.
≤ = equal to or less than.
< = less than.

Figure 10. Percent of persons 6 months–74 years with erythrocyte protoporphyrin values of 30 μg/dl or more by blood lead and iron status: United States, 1976–80

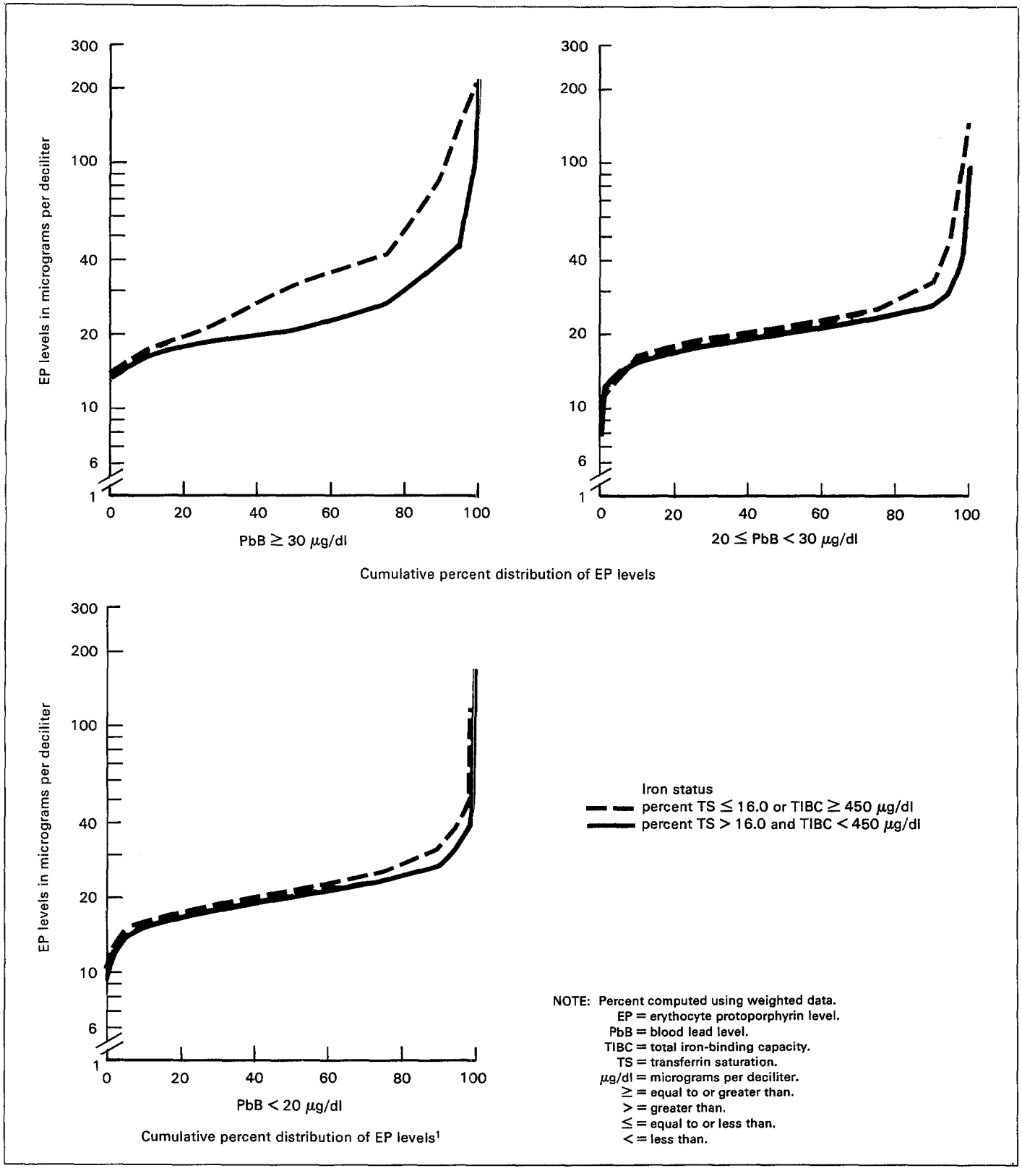


Figure 11. Cumulative percent distribution of erythrocyte protoporphyrin levels for persons 6 months–74 years by blood lead levels and iron status: United States, 1976–80

Table D. Number of examined children 6 months–5 years, estimated population, percent of estimated population, and standard error of the percent by selected erythrocyte protoporphyrin levels, according to selected blood lead levels: United States, 1976–80

Erythrocyte protoporphyrin level in micrograms per deciliter whole blood	*Blood lead level in micrograms per deciliter whole blood*							
	25 or more				*30 or more*			
	Number of examinees[1]	*Estimated population in thousands*	*Percent of estimated population*	*Standard error or the percent*	*Number of examinees*[1]	*Estimated population in thousands*	*Percent of estimated population*	*Standard error of the percent*
Total	256	1,534	100.0	...	117	675	100.0	...
Less than 35	189	1,137	74.1	2.7	69	389	57.7	6.0
35–49	37	216	14.1	2.2	25	152	22.5	3.8
50 or more	30	181	11.8	2.0	23	134	19.8	4.6

[1]The total number of blood samples that were obtained by venipuncture that had both valid blood lead and erythrocyte protoporphyrin values was 2,365 (estimated population in thousands = 16,862). See appendix I for details on the elimination of blood samples from the data.

Summary

National estimates of blood lead levels indicated that exposure to lead in our environment was ubiquitous throughout the U.S. population. For children, blood lead levels were contingent upon age, sex, race, socioeconomic status, and degree of urbanization of place of residence. For children 6 months–5 years, the average blood lead level and the prevalence of elevated blood lead levels (30 μg/dl or more) were substantially higher for black children than for white children during the survey period. An inverse relationship between family income (or education of the head of the household) and average blood lead levels in children ages 6 months–5 years suggests that socioeconomic factors are associated with community and household sources of lead that can influence childhood exposure to lead. There is greater concern about childhood exposure to these sources because of the greater susceptibility of young children to the toxic effects of lead.[5,8,16,56] In addition, young children are more likely to be exposed to lead in dust, dirt, and lead-based paint because of increased hand-to-mouth activity and the tendency to eat unusual substances.

For adults ages 18–74 years, blood lead levels were associated with age, race, sex, occupation, alcohol consumption, and smoking. There was a striking sex difference in average blood lead levels, with significantly higher values for men than for women. Analyses of the NHANES II data indicated that the effects of exposure to lead associated with occupation, alcohol consumption, and smoking are additive.

A 37-percent drop in average blood lead levels of persons 6 months–74 years over the survey period indicated a lessening of exposure to lead in the U.S. population. The most reasonable explanation for the decline in average blood lead levels is the decrease in the use of lead in gasoline over the same period.

Both iron status and blood lead level are associated with erythrocyte protoporphyrin (EP) level. Measurement of EP has been used by many of the lead screening programs to screen individuals initially for high lead levels and for iron deficiency. National estimates from the NHANES II data indicate that the interrelationship of iron status and blood lead level and their combined effects on EP should be considered when the criteria for identifying children or adults with excess body burdens of lead are evaluated.

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List of detailed tables

Table 1. Blood lead levels of persons 6 months–74 years, with arithmetic mean, standard error of the mean, and selected percentiles, by race and age: United States, 1976–80

Race and age	Estimated population in thousands[1]	Number examined[2]	Arithmetic mean	Standard error of the mean	Percentile						
					5th	10th	25th	50th	75th	90th	95th
All races[3]			Blood lead level in micrograms per deciliter								
Total	203,554	9,933	13.9	0.24	7.0	8.0	10.0	13.0	17.0	21.0	25.0
6 months–2 years	7,676	759	16.3	0.57	8.0	9.0	11.0	15.0	20.0	25.0	29.0
3–5 years	9,186	1,613	15.9	0.40	8.0	9.0	11.0	15.0	19.0	24.0	27.0
6–8 years	10,259	451	13.9	0.47	7.0	8.0	11.0	13.0	16.0	20.0	24.0
9–11 years	10,621	377	12.9	0.39	7.0	8.0	10.0	12.0	16.0	19.0	20.0
12–14 years	11,632	448	11.4	0.32	6.0	7.0	8.0	11.0	14.0	16.0	19.0
15–17 years	12,452	444	12.1	0.35	6.0	7.0	9.0	11.0	14.0	18.0	22.0
18–24 years	27,448	985	13.1	0.33	6.0	8.0	9.0	12.0	16.0	21.0	23.0
25–34 years	32,752	1,041	13.7	0.33	6.0	7.0	10.0	13.0	17.0	21.0	24.0
35–44 years	23,651	753	14.6	0.36	7.0	8.0	10.0	13.0	18.0	22.0	27.0
45–54 years	23,032	724	15.3	0.32	7.0	8.0	11.0	14.0	18.0	23.0	26.0
55–64 years	20,350	1,149	14.6	0.32	7.0	8.0	10.0	14.0	18.0	22.0	26.0
65–74 years	14,496	1,189	14.4	0.23	7.0	8.0	10.0	13.0	17.0	21.0	25.0
White											
Total	174,528	8,369	13.7	0.24	6.0	8.0	10.0	13.0	16.0	21.0	24.0
6 months–2 years	6,186	589	15.0	0.56	7.0	8.0	11.0	14.0	18.0	23.0	26.0
3–5 years	7,455	1,287	14.9	0.41	8.0	9.0	11.0	14.0	18.0	22.0	25.0
6–8 years	8,436	374	13.3	0.46	7.0	8.0	10.0	12.0	16.0	20.0	22.0
9–11 years	8,960	315	12.4	0.39	7.0	8.0	9.0	12.0	15.0	18.0	20.0
12–14 years	9,705	367	11.0	0.29	6.0	7.0	8.0	11.0	13.0	16.0	17.0
15–17 years	10,429	368	12.0	0.36	6.0	7.0	9.0	11.0	14.0	18.0	22.0
18–24 years	23,522	849	13.0	0.34	6.0	8.0	9.0	12.0	15.0	21.0	23.0
25–34 years	28,227	885	13.5	0.32	6.0	7.0	9.0	12.0	16.0	21.0	24.0
35–44 years	20,348	648	14.4	0.41	6.0	7.0	10.0	13.0	17.0	22.0	27.0
45–54 years	20,137	625	15.1	0.33	7.0	8.0	11.0	14.0	18.0	23.0	26.0
55–64 years	18,300	1,020	14.4	0.34	7.0	8.0	10.0	13.0	17.0	22.0	26.0
65–74 years	12,824	1,042	14.2	0.28	7.0	8.0	10.0	13.0	17.0	21.0	24.0
Black											
Total	23,853	1,332	15.7	0.48	8.0	9.0	11.0	15.0	19.0	23.0	27.0
6 months–2 years	1,164	141	20.9	0.96	11.0	11.0	15.0	19.0	25.0	33.1	38.0
3–5 years	1,421	278	20.8	0.55	10.0	12.0	16.0	20.0	24.0	30.0	35.0
6–8 years	1,526	65	17.7	1.10	*	11.0	13.0	17.0	21.0	27.0	*
9–11 years	1,567	57	15.8	0.75	*	11.0	13.0	16.0	18.0	20.0	*
12–14 years	1,697	75	13.7	0.71	*	8.0	10.0	14.0	16.0	20.0	*
15–17 years	1,738	66	13.0	0.69	*	8.0	10.0	12.0	15.0	20.0	*
18–24 years	3,406	119	13.8	0.67	7.0	8.0	10.0	13.0	17.0	21.0	23.0
25–34 years	3,499	125	15.1	0.81	7.0	8.0	10.0	14.0	19.0	24.0	28.0
35–44 years	2,527	87	15.6	0.84	*	9.0	11.0	14.0	19.0	21.0	*
45–54 years	2,259	82	17.2	0.90	*	11.0	13.0	16.0	21.0	25.0	*
55–64 years	1,760	116	17.2	1.01	8.0	10.0	12.0	16.0	20.0	25.0	28.0
65–74 years	1,288	121	15.9	0.71	8.0	9.0	11.0	14.0	19.0	22.0	26.0

[1]At the midpoint of the survey, March 1, 1978.
[2]With lead determinations from blood specimens drawn by venipuncture.
[3]Includes data for races not shown separately.

Table 2. Blood lead levels of males 6 months–74 years, with arithmetic mean, standard error of the mean, and selected percentiles, by race and age: United States, 1976–80

Race and age	Estimated population in thousands[1]	Number examined[2]	Arithmetic mean	Standard error of the mean	Percentile						
					5th	10th	25th	50th	75th	90th	95th
All races[3]			Blood lead level in micrograms per deciliter								
Total	99,062	4,945	16.1	0.26	8.0	9.0	12.0	15.0	19.0	24.0	27.0
6 months–2 years	3,926	396	16.8	0.64	8.0	9.0	12.0	15.0	20.0	26.0	31.0
3–5 years	4,695	851	16.0	0.44	8.0	9.0	12.0	15.0	19.0	24.0	28.0
6–8 years	5,345	229	14.4	0.55	7.0	9.0	11.0	13.0	17.0	22.0	27.0
9–11 years	5,301	200	13.6	0.39	8.0	9.0	10.0	13.0	16.0	19.0	22.0
12–14 years	5,920	228	12.6	0.39	7.0	8.0	10.0	13.0	15.0	17.0	20.0
15–17 years	6,320	245	13.9	0.42	7.0	8.0	10.0	13.0	16.0	21.0	22.0
18–24 years	13,275	492	15.9	0.42	8.0	9.0	12.0	15.0	19.0	23.0	26.0
25–34 years	15,895	502	16.8	0.45	9.0	10.0	13.0	16.0	20.0	24.0	27.0
35–44 years	11,367	342	17.7	0.46	9.0	11.0	13.0	17.0	21.0	26.0	32.1
45–54 years	11,114	347	17.5	0.49	9.0	11.0	13.0	17.0	21.0	26.0	28.0
55–64 years	9,607	565	16.7	0.40	8.0	10.0	12.0	15.0	20.0	25.0	29.0
65–74 years	6,297	548	16.3	0.29	9.0	10.0	12.0	15.0	20.0	23.0	28.0
White											
Total	85,112	4,153	15.8	0.27	8.0	9.0	12.0	15.0	19.0	23.0	27.0
6 months–2 years	3,120	293	15.6	0.63	8.0	9.0	11.0	15.0	19.0	24.0	27.0
3–5 years	3,789	676	15.0	0.44	8.0	9.0	11.0	14.0	18.0	22.0	25.0
6–8 years	4,357	199	13.7	0.55	7.0	8.0	10.0	13.0	16.0	20.0	24.0
9–11 years	4,472	164	13.0	0.43	8.0	9.0	10.0	13.0	15.0	18.0	21.0
12–14 years	4,932	190	12.2	0.37	7.0	8.0	9.0	12.0	14.0	17.0	19.0
15–17 years	5,298	200	13.7	0.47	7.0	8.0	10.0	13.0	16.0	22.0	23.0
18–24 years	11,522	435	15.8	0.45	8.0	9.0	12.0	15.0	19.0	23.0	27.0
25–34 years	13,884	423	16.6	0.46	9.0	10.0	12.0	16.0	20.0	24.0	27.0
35–44 years	9,740	295	17.6	0.50	9.0	11.0	13.0	16.0	21.0	26.0	31.0
45–54 years	9,878	306	17.2	0.54	9.0	11.0	13.0	17.0	20.0	26.0	28.0
55–64 years	8,580	500	16.4	0.44	8.0	10.0	12.0	15.0	20.0	25.0	29.0
65–74 years	5,537	472	16.0	0.36	8.0	10.0	12.0	15.0	20.0	23.0	27.0
Black											
Total	11,171	664	18.3	0.52	10.0	11.0	14.0	17.0	21.0	27.0	30.0
6 months–2 years	589	85	20.2	1.12	*	11.0	15.0	18.0	24.0	33.1	*
3–5 years	718	146	21.0	0.78	10.0	12.0	16.0	20.0	25.0	30.0	38.0
6–8 years	804	23	*19.3	*1.59	*	*	16.0	17.0	22.0	*	*
9–11 years	750	32	16.3	0.92	*	*	13.0	15.0	18.0	*	*
12–14 years	851	34	14.9	1.07	*	*	11.0	15.0	18.0	*	*
15–17 years	867	40	14.8	0.85	*	*	12.0	14.0	18.0	*	*
18–24 years	1,533	49	16.8	0.91	*	*	13.0	17.0	20.0	*	*
25–34 years	1,546	65	19.1	0.85	*	12.0	15.0	18.0	22.0	28.0	*
35–44 years	1,112	37	19.1	1.56	*	*	12.0	17.0	21.0	*	*
45–54 years	1,044	35	20.9	1.27	*	*	17.0	21.0	24.0	*	*
55–64 years	801	57	21.2	1.69	*	12.0	16.0	19.0	25.0	28.0	*
65–74 years	555	61	18.7	1.30	*	10.0	12.0	17.0	21.0	27.0	*

[1]At the midpoint of the survey, March 1, 1978.
[2]With lead determinations from blood specimens drawn by venipuncture.
[3]Includes data for races not shown separately.

Table 3. Blood lead levels of females 6 months–74 years, with arithmetic mean, standard error of the mean, and selected percentiles, by race and age: United States, 1976–80

Race and age	Estimated population in thousands[1]	Number examined[2]	Arithmetic mean	Standard error of the mean	Percentile						
					5th	10th	25th	50th	75th	90th	95th
All races[3]			Blood lead level in micrograms per deciliter								
Total	104,492	4,988	11.9	0.23	6.0	7.0	9.0	11.0	14.0	18.0	20.0
6 months–2 years	3,750	363	15.7	0.59	7.0	9.0	11.0	14.0	19.0	24.0	29.0
3–5 years	4,491	762	15.8	0.42	8.0	9.0	11.0	15.0	19.0	24.0	26.0
6–8 years	4,914	222	13.5	0.56	7.0	8.0	10.0	12.0	16.0	20.0	22.0
9–11 years	5,320	177	12.3	0.48	7.0	7.0	9.0	12.0	15.0	19.0	19.0
12–14 years	5,712	220	10.1	0.38	5.0	6.0	8.0	10.0	12.0	15.0	16.0
15–17 years	6,131	199	10.3	0.31	5.0	6.0	8.0	10.0	12.0	15.0	16.0
18–24 years	14,173	493	10.5	0.27	5.0	7.0	8.0	10.0	12.0	15.0	16.0
25–34 years	16,856	539	10.8	0.24	5.0	6.0	8.0	10.0	13.0	16.0	19.0
35–44 years	12,284	411	11.7	0.34	6.0	7.0	9.0	11.0	14.0	17.0	19.0
45–54 years	11,918	377	13.3	0.30	7.0	7.0	10.0	13.0	16.0	19.0	22.0
55–64 years	10,743	584	12.8	0.38	6.0	7.0	9.0	12.0	15.0	19.0	22.0
65–74 years	8,198	641	12.8	0.29	7.0	8.0	9.0	12.0	15.0	19.0	22.0
White											
Total	89,417	4,216	11.7	0.23	6.0	7.0	9.0	11.0	14.0	17.0	20.0
6 months–2 years	3,066	296	14.5	0.58	7.0	8.0	10.0	14.0	18.0	22.0	24.0
3–5 years	3,666	611	14.8	0.44	8.0	9.0	11.0	14.0	18.0	22.0	25.0
6–8 years	4,079	175	13.0	0.59	7.0	8.0	10.0	12.0	15.0	18.0	21.0
9–11 years	4,488	151	11.7	0.43	6.0	7.0	8.0	11.0	14.0	18.0	19.0
12–14 years	4,773	177	9.8	0.38	5.0	6.0	7.0	9.0	12.0	14.0	16.0
15–17 years	5,130	168	10.2	0.31	5.0	6.0	8.0	10.0	12.0	15.0	16.0
18–24 years	11,999	414	10.4	0.29	5.0	6.0	8.0	10.0	12.0	15.0	16.0
25–34 years	14,343	462	10.7	0.25	5.0	6.0	8.0	10.0	13.0	16.0	18.0
35–44 years	10,607	353	11.5	0.38	6.0	7.0	8.0	11.0	14.0	17.0	19.0
45–54 years	10,259	319	13.2	0.33	7.0	7.0	10.0	13.0	15.0	20.0	23.0
55–64 years	9,720	520	12.7	0.40	6.0	7.0	9.0	12.0	15.0	19.0	22.0
65–74 years	7,287	570	12.8	0.31	7.0	8.0	9.0	12.0	15.0	19.0	22.0
Black											
Total	12,682	668	13.4	0.45	7.0	8.0	10.0	13.0	16.0	20.0	22.0
6 months–2 years	575	56	21.7	1.07	*	13.0	15.0	21.0	25.0	34.0	*
3–5 years	703	132	20.6	0.76	11.0	13.0	16.0	20.0	24.0	29.0	32.0
6–8 years	722	42	16.3	1.15	*	*	12.0	15.0	19.0	*	*
9–11 years	817	25	15.4	0.88	*	*	12.0	16.0	18.0	*	*
12–14 years	846	41	12.4	1.13	*	*	9.0	12.0	14.0	*	*
15–17 years	871	26	10.8	0.72	*	*	9.0	10.0	12.0	*	*
18–24 years	1,873	70	11.4	0.46	*	7.0	9.0	11.0	14.0	16.0	*
25–34 years	1,953	60	12.0	0.69	*	7.0	8.0	11.0	14.0	18.0	*
35–44 years	1,415	50	13.0	0.35	*	8.0	10.0	13.0	16.0	19.0	*
45–54 years	1,215	47	13.6	0.59	*	*	11.0	13.0	16.0	*	*
55–64 years	959	59	14.0	0.79	*	9.0	10.0	14.0	17.0	20.0	*
65–74 years	733	60	13.6	0.59	*	9.0	10.0	13.0	16.0	19.0	*

[1]At the midpoint of the survey, March 1, 1978.
[2]With lead determinations from blood specimens drawn by venipuncture.
[3]Includes data for races not shown separately.

Table 4. Blood lead levels of persons 6 months–74 years, with arithmetic mean, standard error of the mean, geometric mean, and geometric standard deviation, and percent distribution by race, age, and sex: United States, 1976–80

Characteristic	Estimated population in thousands[1]	Number examined[2]	Blood lead level in micrograms per deciliter: Arithmetic mean	Standard error of the mean	Geometric mean	Geometric standard deviation	Less than 10	10–19	20–29	30–39	Equal to or greater than 40
All races[3]							Percent distribution				
All ages	203,554	9,933	13.9	0.4	12.8	1.50	22.1	62.9	13.0	1.6	0.4
6 months–5 years	16,862	2,372	16.0	0.42	14.9	1.47	12.2	63.3	20.5	3.5	0.5
6–17 years	44,964	1,720	12.5	0.30	11.7	1.45	27.6	64.8	7.1	0.5	-
18–74 years:											
Men	67,555	2,796	16.8	0.28	15.8	1.45	7.6	64.1	24.2	3.4	0.7
Women	74,173	3,045	11.8	0.22	11.0	1.46	33.7	60.6	5.2	0.3	0.2
White											
All ages	174,528	8,369	13.7	0.24	12.6	1.50	23.3	62.8	12.2	1.5	0.2
6 months–5 years	13,641	1,876	14.9	0.42	14.0	1.44	14.5	67.4	16.1	1.8	0.2
6–17 years	37,530	1,424	12.1	0.30	11.3	1.44	30.4	63.4	5.8	0.4	-
18–74 years:											
Men	59,142	2,431	16.6	0.29	15.6	1.44	8.1	64.8	23.3	3.3	0.5
Women	64,215	2,638	11.7	0.23	10.9	1.47	34.6	59.9	5.0	0.4	-
Black											
All ages	23,853	1,332	15.7	0.48	14.6	1.49	13.3	63.7	20.0	2.3	0.7
6 months–5 years	2,584	419	20.9	0.61	19.6	1.44	2.5	45.3	40.0	10.2	2.0
6–17 years	6,529	263	14.8	0.53	14.0	1.42	12.8	70.9	15.6	0.7	-
18–74 years:											
Men	6,592	304	19.1	0.70	18.1	1.44	2.3	56.4	34.9	4.5	1.9
Women	8,148	346	12.7	0.44	12.0	1.40	24.7	68.1	7.2	-	-

[1]At the midpoint of the survey, March 1, 1978.
[2]With lead determinations from blood specimens drawn by venipuncture.
[3]Includes data for races not shown separately.

Table 5. Blood lead levels of persons 6 months–74 years, with arithmetic mean, standard error of the mean, geometric mean and geometric standard deviation, and percent distribution, by income, race, age, and sex: United States, 1976–80

Characteristic	Estimated population in thousands[1]	Number examined[2]	Blood lead level in micrograms per deciliter: Arithmetic mean	Standard error of the mean	Geometric mean	Geometric standard deviation	Less than 10	10–19	20–29	30–39	Equal to or greater than 40
UNDER $6,000							Percent distribution				
All races[3]	29,410	1,862	14.5	0.40	13.1	1.56	22.1	59.9	15.1	1.9	1.0
6 months–5 years	2,465	448	20.0	0.56	18.6	1.47	3.9	50.6	34.6	9.3	1.6
6–17 years	5,046	230	14.6	0.61	13.5	1.48	16.1	68.5	12.6	2.7	0.1
18–74 years:											
Men	7,945	454	17.5	0.70	16.0	1.55	10.9	54.7	29.1	3.2	2.1
Women	13,954	730	12.1	0.36	11.2	1.49	32.5	61.0	5.9	0.1	0.5
White											
All ages	21,542	1,315	14.0	0.44	12.6	1.56	25.1	59.1	13.1	1.6	1.1
6 months–5 years	1,408	256	18.1	0.61	17.0	1.44	5.4	61.5	27.2	5.3	0.6
6–17 years	3,067	140	14.0	0.69	12.9	1.51	20.1	67.3	9.5	3.0	0.1
18–74 years:											
Men	6,340	353	17.0	0.76	15.4	1.55	12.5	56.9	25.1	3.1	2.4
Women	10,727	566	11.8	0.42	10.9	1.51	35.6	57.8	5.8	0.1	0.7
Black											
All ages	7,355	512	15.8	0.47	14.6	1.53	13.5	63.3	20.3	2.4	0.5
6 months–5 years	917	176	22.9	0.89	21.4	1.47	2.1	36.9	42.5	15.2	3.3
6–17 years	1,927	87	15.7	0.76	14.8	1.42	9.1	70.4	18.2	2.3	-
18–74 years:											
Men	1,451	93	19.5	0.76	18.4	1.51	5.2	46.2	44.6	2.5	1.5
Women	3,061	156	12.9	0.39	12.2	1.44	21.9	72.0	6.1	-	-
$6,000–$14,999											
All races[3]											
All ages	80,416	4,033	14.2	0.25	13.1	1.51	20.5	62.9	14.4	1.9	0.3
6 months–5 years	7,534	1,083	16.2	0.46	15.1	1.46	10.6	63.0	22.2	3.7	0.5
6–17 years	17,533	672	12.9	0.41	12.0	1.46	22.5	68.8	8.5	0.2	-
18–74 years:											
Men	25,436	1,094	17.4	0.32	16.3	1.44	6.5	60.7	27.7	4.3	0.8
Women	29,913	1,184	11.8	0.20	11.0	1.45	32.9	61.5	5.0	0.5	0.1
White											
All ages	68,135	3,413	13.9	0.26	12.8	1.51	21.9	62.5	13.5	1.8	0.3
6 months–5 years	6,252	887	15.3	0.48	14.3	1.44	12.3	66.6	18.9	2.1	0.1
6–17 years	13,936	531	12.4	0.39	11.6	1.47	25.8	66.9	7.1	0.2	-
18–74 years:											
Men	22,162	956	17.1	0.33	16.1	1.43	7.0	61.3	27.0	4.2	0.5
Women	25,784	1,039	11.7	0.21	10.9	1.46	34.2	60.7	4.3	0.5	0.3
Black											
All ages	10,334	533	16.1	0.48	14.9	1.47	11.7	64.2	20.7	2.3	1.1
6 months–5 years	1,037	163	20.7	0.64	19.6	1.41	1.9	47.0	39.0	10.1	2.0
6–17 years	3,159	125	14.9	0.71	14.1	1.37	9.8	75.9	14.3	-	-
18–74 years:											
Men	2,762	121	20.4	0.82	18.9	1.46	1.1	53.3	36.1	5.8	3.7
Women	3,376	124	12.9	0.61	12.3	1.39	23.5	66.1	10.4	-	-
$15,000 OR MORE											
All races[3]											
All ages	87,062	3,718	13.5	0.24	12.6	1.48	23.6	63.9	11.2	1.2	0.1
6 months–5 years	6,428	774	14.1	0.41	13.3	1.43	17.6	69.0	12.2	1.1	0.1
6–17 years	20,814	761	11.7	0.25	11.1	1.41	34.0	61.2	4.6	0.2	-
18–74 years:											
Men	31,674	1,152	16.3	0.29	15.3	1.42	7.7	68.6	20.8	2.7	0.2
Women	28,146	1,031	11.6	0.28	10.9	1.45	35.0	59.8	5.0	0.2	-

See footnotes at end of table.

Table 5. Blood lead levels of persons 6 months–74 years, with arithmetic mean, standard error of the mean, geometric mean and geometric standard deviation, and percent distribution, by income, race, age, and sex: United States, 1976–80—Con.

Characteristic	Estimated population in thousands[1]	Number examined[2]	Blood lead level in micrograms per deciliter: Arithmetic mean	Standard error of the mean	Geometric mean	Geometric standard deviation	Less than 10	10–19	20–29	30–39	Equal to or greater than 40
$15,000 OR MORE—Con.							Percent distribution				
White											
All ages	79,707	3,401	13.4	0.26	12.5	1.48	24.1	63.9	10.8	1.1	0.1
6 months–5 years	5,707	690	13.7	0.44	12.9	1.41	19.4	70.1	9.8	0.6	0.1
6–17 years	19,174	705	11.6	0.28	11.0	1.40	34.9	60.9	4.0	0.2	-
18–74 years:											
Men	28,808	1,052	16.1	0.31	15.2	1.42	8.3	68.6	20.4	2.5	0.2
Women	26,018	954	11.7	0.29	10.9	1.45	34.6	60.1	5.1	0.2	-
Black											
All ages	4,995	224	14.9	0.58	13.9	1.45	15.5	63.7	19.1	1.7	-
6 months–5 years	502	60	17.2	0.83	16.2	1.41	5.1	58.2	33.7	2.7	0.1
6–17 years	1,225	42	13.6	0.79	12.6	1.51	25.6	57.0	17.4	-	-
18–74 years:											
Men	1,949	73	17.5	0.81	16.7	1.36	0.4	67.2	29.0	3.4	-
Women	1,318	49	11.5	0.63	11.0	1.33	33.0	64.8	2.2	-	-

[1]At the midpoint of the survey, March 1, 1978.
[2]With lead determinations from blood specimens drawn by venipuncture.
[3]Includes data for races not shown separately.

Table 6. Blood lead levels of persons 6 months–74 years, with arithmetic mean, standard error of the mean, geometric mean and geometric standard deviation, and percent distribution, by urban or rural residence, race, age, and sex: United States, 1976–80

Characteristic	Estimated population in thousands[1]	Number examined[2]	Blood lead level in micrograms per deciliter: Arithmetic mean	Standard error of the mean	Geometric mean	Geometric standard deviation	Less than 10	10–19	20–29	30–39	Equal to or greater than 40
URBAN, 1 MILLION PERSONS OR MORE							Percent distribution				
All races[3]											
All ages	59,532	2,395	15.0	0.37	14.0	1.45	14.3	66.3	17.3	1.6	0.5
6 months–5 years	4,344	544	18.0	0.53	16.8	1.46	7.1	56.7	29.0	6.2	1.0
6–17 years	12,893	414	13.8	0.53	13.1	1.38	15.8	72.9	10.9	0.4	-
18–74 years:											
Men	19,541	677	17.8	0.34	16.9	1.41	3.3	62.8	29.8	3.2	0.9
Women	22,755	760	12.9	0.40	12.2	1.41	23.8	67.1	8.6	0.4	0.1
White											
All ages	46,407	1,767	15.0	0.31	14.0	1.44	13.6	67.1	17.3	1.5	0.5
6 months–5 years	3,112	358	16.6	0.59	15.6	1.43	8.4	63.3	24.3	3.9	0.1
6–17 years	9,681	294	13.3	0.55	12.6	1.38	17.8	72.2	9.7	0.3	-
18–74 years:											
Men	15,809	531	17.8	0.28	16.9	1.40	3.1	63.0	29.7	3.0	0.2
Women	17,805	584	13.1	0.36	12.4	1.40	21.5	68.5	9.4	0.5	0.1
Black											
All ages	11,687	570	15.5	0.84	14.4	1.46	14.6	63.8	19.0	2.1	0.5
6 months–5 years	1,093	172	22.2	0.83	20.8	1.43	2.9	38.0	43.9	12.0	3.2
6–17 years	3,010	111	15.3	0.83	14.6	1.36	9.4	74.4	15.7	0.5	-
18–74 years:											
Men	3,267	132	18.4	1.22	17.4	1.41	3.0	59.4	32.5	4.3	0.8
Women	4,318	155	12.4	0.78	11.8	1.40	28.5	64.5	7.0	-	-
URBAN, FEWER THAN 1 MILLION PERSONS											
All races[3]											
All ages	79,906	3,869	13.9	0.32	12.8	1.51	22.4	62.9	12.8	1.6	0.3
6 months–5 years	6,891	944	16.5	0.67	15.4	1.46	10.3	61.7	24.5	3.1	0.4
6–17 years	16,988	638	12.6	0.35	11.7	1.47	27.6	64.5	7.4	0.5	-
18–74 years:											
Men	25,672	1,050	16.8	0.37	15.7	1.45	8.3	63.0	24.6	3.6	0.5
Women	30,356	1,237	11.8	0.32	11.0	1.45	33.2	62.2	4.2	0.3	0.1
White											
All ages	67,707	3,144	13.6	0.32	12.5	1.50	24.2	62.4	11.6	1.5	0.3
6 months–5 years	5,297	699	15.4	0.67	14.4	1.44	12.9	66.5	19.0	1.3	0.3
6–17 years	13,871	510	12.2	0.36	11.4	1.46	30.4	63.1	6.1	0.4	-
18–74 years:											
Men	22,369	889	16.5	0.38	15.4	1.45	9.1	63.7	23.2	3.5	0.5
Women	26,171	1,046	11.6	0.32	10.8	1.46	35.2	60.5	3.8	0.3	0.2
Black											
All ages	9,783	612	15.9	0.54	14.8	1.49	11.3	64.6	21.4	2.2	0.5
6 months–5 years	1,246	205	20.3	0.78	19.2	1.39	2.2	46.4	41.2	9.6	0.6
6–17 years	2,717	113	14.5	0.64	13.6	1.46	15.0	69.1	15.3	0.6	-
18–74 years:											
Men	2,551	134	19.6	0.71	18.6	1.44	1.4	53.1	39.7	4.3	1.5
Women	3,268	160	13.0	0.62	12.4	1.39	18.3	74.3	7.4	-	-

See footnotes at end of table.

Table 6. Blood lead levels of persons 6 months–74 years, with arithmetic mean, standard error of the mean, geometric mean and geometric standard deviation, and percent distribution, by urban or rural residence, race, age, and sex: United States, 1976–80—Con.

Characteristic	Estimated population in thousands[1]	Number examined[2]	Blood lead level in micrograms per deciliter: Arithmetic mean	Standard error of the mean	Geometric mean	Geometric standard deviation	Less than 10	10–19	20–29	30–39	Equal to or greater than 40
RURAL							Percent distribution				
All races[3]											
All ages	64,116	3,669	13.0	0.40	11.9	1.53	28.9	59.8	9.5	1.6	0.2
6 months–5 years	5,627	884	13.9	0.64	13.0	1.44	18.2	70.3	9.4	1.9	0.2
6–17 years	15,083	668	11.4	0.52	10.7	1.45	37.2	58.6	3.7	0.5	-
18–74 years:											
Men	22,343	1,069	16.1	0.43	15.1	1.46	10.4	66.5	19.1	3.5	0.5
Women	21,063	1,048	10.7	0.36	9.8	1.49	44.7	51.8	3.0	0.3	0.2
White											
All ages	60,414	3,458	12.8	0.39	11.8	1.52	29.6	59.9	8.9	1.5	0.1
6 months–5 years	5,233	819	13.5	0.57	12.7	1.42	19.7	70.8	8.3	1.2	-
6–17 years	13,978	620	11.2	0.48	10.5	1.44	38.8	57.8	2.9	0.5	-
18–74 years:											
Men	20,963	1,011	15.9	0.44	14.8	1.44	10.6	67.3	18.6	3.2	0.3
Women	20,239	1,008	10.6	0.36	9.8	1.49	44.8	51.9	2.7	0.3	0.3
Black											
All ages	2,383	150	16.2	0.68	14.4	1.61	15.5	60.0	18.7	3.2	2.6
6 months–5 years	245	42	18.3	2.60	16.5	1.65	2.1	68.0	19.6	6.2	4.1
6–17 years	802	39	13.9	1.33	13.0	1.49	18.5	64.0	15.7	1.8	-
18–74 years:											
Men	774	38	20.4	1.47	18.3	1.56	2.9	55.3	29.3	5.9	6.6
Women	562	31	12.4	0.97	11.3	1.59	33.5	59.2	7.3	-	-

[1]At the midpoint of the survey, March 1, 1978.
[2]With lead determinations from blood specimens drawn by venipuncture.
[3]Includes data for races not shown separately.

Table 7. Blood lead levels of persons 6 months–74 years, with arithmetic mean, standard error of the mean, geometric mean and geometric standard deviation, and percent distribution, by central city or non-central city residence, race, age, and sex: United States, 1976–80

Characteristic	Estimated population in thousands[1]	Number examined[2]	Blood lead level in micrograms per deciliter: Arithmetic mean	Standard error of the mean	Geometric mean	Geometric standard deviation	Percent distribution: Less than 20	20–29	Equal to or greater than 30
CENTRAL CITY									
All races[3]									
All ages	24,560	1,123	14.9	0.67	13.9	1.46	81.5	16.6	1.9
6 months–5 years	1,822	286	20.0	0.71	18.5	1.50	51.8	36.6	11.6
6–17 years	5,124	177	14.6	0.87	13.9	1.37	85.0	15.0	-
18–74 years:									
Men	7,661	297	17.3	0.70	16.3	1.41	71.2	25.2	3.6
Women	9,953	363	12.6	0.70	11.8	1.42	91.4	8.4	0.2
White									
All ages	14,602	625	14.8	0.56	13.9	1.43	82.7	16.1	1.2
6 months–5 years	885	133	17.4	0.84	16.3	1.45	64.8	30.7	4.5
6–17 years	2,710	86	14.3	0.93	13.6	1.37	85.2	14.8	-
18–74 years:									
Men	4,778	183	17.1	0.56	16.1	1.40	73.2	24.3	2.5
Women	6,229	223	13.0	0.66	12.3	1.41	90.8	8.9	0.3
Black									
All ages	8,856	452	15.4	0.94	14.2	1.48	78.2	18.8	3.0
6 months–5 years	855	143	23.1	1.30	21.6	1.46	36.7	44.7	18.6
6–17 years	2,259	84	15.0	1.00	14.3	1.37	83.6	16.4	-
18–74 years:									
Men	2,514	103	18.0	1.42	17.0	1.41	65.7	28.2	6.1
Women	3,228	122	12.2	0.98	11.5	1.43	91.3	8.7	-
NON-CENTRAL CITY									
All races[3]									
All ages	34,908	1,268	15.1	0.30	14.1	1.44	80.0	17.8	2.2
6 months–5 years	2,519	257	16.5	0.60	15.6	1.41	73.0	23.3	3.7
6–17 years	7,746	236	13.3	0.59	12.6	1.39	90.9	8.5	0.6
18–74 years:									
Men	11,880	380	18.2	0.31	17.2	1.40	62.7	32.8	4.5
Women	12,763	395	13.2	0.32	12.5	1.39	90.4	8.9	0.7
White									
All ages	31,741	1,138	15.1	0.32	14.1	1.45	79.9	17.9	2.2
6 months–5 years	2,223	224	16.2	0.65	15.3	1.42	74.7	21.5	3.8
6–17 years	6,949	207	13.0	0.63	12.3	1.38	91.6	7.9	0.5
18–74 years:									
Men	11,032	348	18.1	0.30	17.2	1.40	63.1	32.1	4.8
Women	11,538	359	13.2	0.35	12.5	1.41	89.6	9.6	0.8
Black									
All ages	2,831	118	16.0	0.60	15.2	1.39	79.2	19.8	1.0
6 months–5 years	238	29	19.2	0.74	18.5	1.33	55.1	41.6	3.3
6–17 years	751	27	16.0	0.67	15.4	1.34	84.1	14.1	1.8
18–74 years:									
Men	753	29	19.7	1.39	18.7	1.42	50.6	48.1	1.3
Women	1,090	33	13.1	0.58	12.7	1.29	98.1	1.9	-

[1]At the midpoint of the survey, March 1, 1978.
[2]With lead determinations from blood specimens drawn by venipuncture.
[3]Includes data for races not shown separately.

Table 8. Blood lead levels of persons 6 months–17 years, with arithmetic mean, standard error of the mean, and percent distribution, by education of the head of household, race, and age: United States, 1976–80

Education of head of household, race, and age	*Number examined*[1]	*Blood lead level in micrograms per deciliter*				
		Arithmetic mean	*Standard error of the mean*	*Less than 20*	*20–29*	*Equal to or greater than 30*
				Percent distribution		
LESS THAN HIGH SCHOOL						
All races[2]						
6 months–17 years	666	14.0	0.62	83.3	14.3	2.4
6 months–5 years	345	19.1	0.88	58.2	32.0	9.8
6–17 years	321	13.1	0.61	87.6	11.3	1.1
White						
6 months–17 years	506	13.3	0.57	87.6	10.2	2.2
6 months–5 years	262	17.9	0.80	65.3	28.4	6.3
6–17 years	244	12.5	0.57	91.3	7.2	1.5
Black						
6 months–17 years	135	15.8	1.04	70.5	27.1	2.4
6 months–5 years	69	22.2	1.86	37.2	45.8	17.0
6–17 years	66	14.8	1.12	75.9	24.1	-
HIGH SCHOOL						
All races[2]						
6 months–17 years	2,199	13.3	0.33	89.3	9.5	1.2
6 months–5 years	1,273	16.4	0.49	73.4	22.5	4.1
6–17 years	926	12.6	0.31	93.0	6.5	0.5
White						
6 months–17 years	1,715	12.7	0.35	91.5	7.9	0.6
6 months–5 years	966	15.1	0.63	80.6	17.4	2.0
6–17 years	749	12.2	0.34	93.8	5.8	0.4
Black						
6 months–17 years	440	16.2	0.38	79.2	17.0	3.8
6 months–5 years	274	21.4	0.63	47.4	39.5	13.1
6–17 years	166	14.8	0.43	88.3	10.6	1.1
COLLEGE LEVEL OR ABOVE						
All races[2]						
6 months–17 years	1,192	12.5	0.28	92.0	7.7	0.3
6 months–5 years	734	14.4	0.40	84.7	14.0	1.3
6–17 years	458	12.0	0.31	94.0	6.0	-
White						
6 months–17 years	1,054	12.3	0.30	93.6	6.3	0.1
6 months–5 years	636	13.9	0.43	88.4	10.9	0.7
6–17 years	418	11.8	0.32	94.9	5.1	-
Black						
6 months–17 years	98	15.8	1.01	72.4	26.3	1.3
6 months–5 years	69	18.0	0.96	58.6	37.1	4.3
6–17 years	29	14.8	1.12	78.3	21.7	-

[1]With lead determinations from blood specimens drawn by venipuncture.
[2]Includes data for races not shown separately.

Table 9. Blood lead levels of children 6 months–5 years, with arithmetic mean, standard error of the mean, and percent above selected levels, by selected medical history items and race of child: United States, 1976–80

Selected medical history items and race of child	Number examined[1]	Blood lead level in micrograms per deciliter: Arithmetic mean	Standard error of the mean	20 or more: Percent	20 or more: Standard error of the percent	25 or more: Percent	25 or more: Standard error of the percent	30 or more: Percent	30 or more: Standard error of the percent
PRIOR TEST FOR LEAD POISONING (REPORTED)									
All races[2]									
Yes	122	21.2	0.70	48.4	3.15	26.1	3.81	14.3	2.53
No	2,226	15.8	0.43	23.2	2.32	8.3	0.99	3.5	0.47
White									
Yes	58	17.2	0.47	28.8	4.42	8.9	3.48	2.2	1.35
No	1,800	14.8	0.44	17.5	2.35	5.4	0.82	2.0	0.34
Black									
Yes	61	25.3	1.28	68.5	5.62	42.4	6.19	26.0	4.26
No	352	20.3	0.62	50.0	4.07	22.2	3.12	10.3	1.48
EATING UNUSUAL SUBSTANCES (REPORTED)									
All races[2]									
Yes	182	19.2	1.02	47.5	6.72	19.3	4.06	8.7	2.23
No	2,192	15.8	0.38	22.6	2.01	8.2	0.85	3.6	0.50
White									
Yes	123	17.5	1.11	37.5	7.42	14.9	4.23	5.6	1.84
No	1,753	14.7	0.39	16.7	2.06	4.8	0.67	1.7	0.34
Black									
Yes	53	23.7	1.61	70.4	7.58	33.6	7.85	19.3	7.14
No	366	20.5	0.59	49.8	3.89	23.3	2.63	11.3	1.64

[1]With lead determinations from blood specimens drawn by venipuncture.
[2]Includes data for races not shown separately.

Table 10. Percent of children 6 months–5 years with a history of eating unusual substances by selected characteristics: United States, 1976–80

Characteristic	*All races*[1]			*White*			*Black*		
	Number examined[2]	*Percent*	*Standard error of the percent*	*Number examined*[2]	*Percent*	*Standard error of the percent*	*Number examined*[2]	*Percent*	*Standard error of the percent*
Age									
6 months–5 years	4,117	8.1	0.44	3,263	7.5	0.55	723	11.4	1.28
6 months–3 years	2,556	11.0	0.58	2,022	10.3	0.73	443	15.1	1.63
4–5 years	1,561	3.2	0.42	1,241	2.8	0.42	280	5.2	1.40
Sex									
Boys	2,142	7.6	0.64	1,662	7.1	0.74	399	10.5	1.75
Girls	1,975	8.7	0.48	1,601	8.0	0.63	324	12.4	2.30
Annual family income									
Less than $10,000	1,699	11.9	1.06	1,149	11.4	1.27	484	13.1	1.48
$10,000 or more	2,296	6.0	0.48	2,041	5.8	0.61	196	9.5	1.96
Degree of urbanization									
Urban, 1 million persons or more	948	8.3	0.75	618	7.8	1.38	298	8.7	1.75
Urban with less than 1 million persons and rural	3,169	8.1	0.57	2,645	7.4	0.58	425	13.4	1.92
Prior test for lead poisoning (reported)									
Yes	199	10.0	2.36	95	9.0	3.01	101	11.3	3.79
No	3,879	8.0	0.43	3,143	7.4	0.53	608	11.3	1.42
Blood lead level in micrograms per deciliter									
30 or more	117	16.4	4.36	42	18.7	6.77	67	18.2	6.30
20–29	503	14.1	2.22	306	13.2	2.38	173	14.8	3.69
Less than 20	1,752	5.2	0.64	1,528	5.1	0.74	179	7.1	1.65

[1]Includes data for races not shown separately.
[2]Number examined in the lead subsample; except for blood lead level, number with lead determinations from blood specimens drawn by venipuncture.

Table 11. Percent of children 6 months–5 years with a history of being tested for lead poisoning by selected characteristics: United States, 1976–80

Characteristic	*All races*[1]			*White*			*Black*		
	Number examined[2]	*Percent*	*Standard error of the percent*	*Number examined*[2]	*Percent*	*Standard error of the percent*	*Number examined*[2]	*Percent*	*Standard error of the percent*
Age									
6 months–5 years	4,078	4.1	0.60	3,238	2.7	0.50	709	12.5	2.17
6 months–3 years	2,535	3.0	0.54	2,009	1.7	0.40	435	11.0	2.40
4–5 years	1,543	6.0	0.83	1,229	4.4	0.80	274	15.2	2.40
Sex									
Boys	2,124	3.5	0.71	1,651	2.3	0.61	392	11.0	2.63
Girls	1,954	4.7	0.60	1,587	3.1	0.51	317	14.1	2.39
Annual family income									
Less than $10,000	1,686	6.7	0.98	1,142	3.5	0.64	478	15.9	3.00
$10,000 or more	2,271	2.6	0.62	2,024	2.3	0.67	188	7.3	1.88
Degree of urbanization									
Urban, 1 million persons or more	932	8.2	1.91	608	2.8	0.97	292	24.6	4.78
Urban with less than 1 million persons and rural	3,146	2.7	0.61	2,630	2.7	0.62	417	3.8	1.56
Eating unusual substances (reported)									
Yes	333	5.1	1.29	237	3.2	1.16	86	12.6	4.15
No	3,745	4.0	0.61	3,001	2.6	0.50	623	12.5	2.31
Blood lead level in micrograms per deciliter				Micrograms per deciliter					
30 or more	115	15.5	2.89	41	3.1	2.40	66	26.3	3.48
20–29	495	7.2	1.43	300	4.7	1.41	171	13.2	2.07
Less than 20	1,738	2.9	0.53	1,517	2.4	0.45	176	8.2	2.35

[1]Includes data for races not shown separately.
[2]Number examined in the lead subsample; except for blood lead level, number with lead determinations from blood specimens drawn by venipuncture.

Table 12. Blood lead levels of persons 18–74 years, with arithmetic mean and standard error of the mean, by sex, degree of cigarette smoking, and degree of alcohol consumption: United States, 1976–80

Smoking and drinking status	Men			Women		
	Number examined[1]	*Arithmetic mean*	*Standard error of the mean*	*Number examined*[1]	*Arithmetic mean*	*Standard error of the mean*
Nonsmokers[2]	1,453	15.6	0.28	2,079	11.2	0.23
Cigarette smokers[2]	1,095	18.5	0.35	960	12.9	0.27
Number of cigarettes smoked per day						
Less than 15	267	17.4	0.48	365	12.3	0.34
15–29	492	18.5	0.45	401	12.9	0.40
Greater than 29	329	19.2	0.48	190	13.9	0.40
Drinking status						
Nondrinkers[3]	844	15.8	0.50	1,491	11.2	0.28
Drinkers[3]	1,937	17.2	0.26	1,546	12.2	0.25
Number of alcoholic drinks consumed per day						
Less than 1	1,420	16.5	0.26	1,375	12.0	0.27
1 or more	517	19.2	0.42	171	14.0	0.43

[1]With lead determinations from blood specimens drawn by venipuncture.
[2]Nonsmokers include those not currently smoking cigarettes, a pipe, or cigars at the time of examination. Cigarette smokers include those currently smoking cigarettes at the time of examination whether or not they smoke cigars or a pipe.
[3]Nondrinkers and drinkers refer to consumption of alcoholic beverages, including beer, wine, or liquor.

Table 13. Blood lead levels of U.S. workers 18–74 years, with arithmetic mean and standard error of the mean, by selected characteristics: United States, 1976–80

Characteristic	Degree of potential exposure to lead at the workplace[1]								
	Total			High			Low		
	Number examined[2]	Arithmetic mean	Standard error of the mean	Number examined[2]	Arithmetic mean	Standard error of the mean	Number examined[2]	Arithmetic mean	Standard error of the mean
MALE WORKERS, 18–74 YEARS									
All races[3]	1,622	17.3	0.32	1,045	18.3	0.39	577	15.5	0.36
White	1,408	17.1	0.34	893	18.1	0.42	515	15.5	0.37
Black	183	19.4	0.81	133	21.0	0.75	50	15.9	1.14
Drinking and smoking status[4]									
Nondrinker, nonsmoker	259	15.4	0.43	160	16.2	0.57	99	14.0	0.67
Nondrinker, smoker	157	18.2	0.87	110	19.1	1.10	47	16.2	0.82
Drinker, nonsmoker	540	16.1	0.41	320	17.0	0.51	220	14.8	0.46
Drinker, smoker	655	18.7	0.33	449	19.7	0.40	206	16.7	0.53
FEMALE WORKERS, 18–74 YEARS									
All races[3]	1,310	11.8	0.25	303	12.9	0.38	1,007	11.5	0.28
White	1,107	11.7	0.25	252	13.2	0.39	855	11.3	0.28
Black	183	12.2	0.48	47	11.8	0.68	136	12.4	0.60
Drinking and smoking status[4]									
Nondrinker, nonsmoker	378	10.7	0.35	79	11.5	0.48	299	10.4	0.37
Nondrinker, smoker	132	12.3	0.34	42	12.2	0.70	90	12.4	0.42
Drinker, nonsmoker	442	11.3	0.32	75	12.5	0.98	367	11.1	0.36
Drinker, smoker	357	13.2	0.39	107	14.4	0.60	250	12.8	0.45

[1]High and low potential exposure groups were defined using information on types of occupations with observed potential exposure to lead from the National Occupational Hazards Survey (NOHS) conducted from 1972 through 1974. Those reporting agricultural occupations and those reporting a selected group of professional and semiprofessional occupations were excluded. The former were excluded because farm-related occupations are not covered under the provisions of the Occupational Safety and Health Act of 1970 and thus were not part of the NOHS. The latter were excluded because of the difficulty in classifying them as either high or low potential exposure to lead.
[2]With lead determinations from blood specimens drawn by venipuncture.
[3]Includes data for races not shown separately.
[4]Drinkers and nondrinkers refer to consumption of alcoholic beverages, including beer, wine, or liquor; smokers include those currently smoking cigarettes, a pipe, or cigars at the time of examination; nonsmokers include exsmokers.

Appendixes

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Appendix I
Statistical notes

This report is based on data collected in the second National Health and Nutrition Examination Survey (NHANES II) from February 1976 through February 1980. NHANES II, conducted by the National Center for Health Statistics, was a survey of the civilian noninstitutionalized U.S. population (including Alaska and Hawaii) 6 months–74 years of age. Both interview and examination procedures were used to collect a broad spectrum of demographic, socioeconomic, and morbidity data and related medical and nutritional information. During household interviews, demographic, socioeconomic, and some of the medical history data were obtained from sample persons. Specially designed mobile examination centers (MEC's), transported to each sample location, provided standardized conditions and equipment for the dietary interview, medical examination, and related clinical tests and procedures.

Survey design

NHANES II utilized a stratified, multistage design that provided for the selection of samples at each stage with known probability. In hierarchical order, the stages of selection were primary sampling units (PSU's—a PSU is a county or a small group of contiguous counties); census enumeration districts (ED's); segments (a segment is a cluster of households); households; eligible persons; and, finally, sample persons.

The first-stage sampling units selected in the previous National Health Examination Survey and NHANES I surveys were subsets of the sample PSU's in the National Health Interview Survey (NHIS), another major data collection program of the National Center for Health Statistics. In NHIS the United States is divided into 1,924 PSU's, with 376 of the PSU's being selected for the sample. Sixty-five of these 376 sample PSU's were selected as the NHANES I sample. The PSU's used in previous examination surveys were defined either as a single county or as a group of contiguous counties (except in certain parts of New England). Many of the larger PSU's were defined as standard metropolitan statistical areas (SMSA's) and often contained several counties. The PSU's that contained several counties and covered a large area were not ideally suited for an examination survey. Attempting to survey large geographic areas from a centrally located examination center created a number of logistical problems. Some examinees had been asked to travel more than 50 miles to be examined, while others had been asked to travel through very congested areas. Many respondents were reluctant to travel under such conditions. The cost of followup visits to the households was also a function of the distance or time from the examination center. An analysis of the response rates for several stands in NHANES I lent further support to these assumptions. The use of smaller areas as PSU's would reduce both the average distance traveled to the examination center by examinees and the cost of the fieldwork. These considerations were the basis for redefining and restratifying the PSU's in NHANES II.

In NHIS, 156 of the 376 PSU's are self-representing SMSA's. It was these 156 self-representing SMSA's in the NHIS design that were redefined and restratified for the NHANES II design.

For NHANES II, the self-representing PSU's in NHIS were first split along county boundaries. Within each region, each of the counties was classified as being either a self-representing or a non-self-representing PSU. The PSU's that were non-self-representing were further combined into homogeneous classes or strata equal in size to the NHIS strata containing non-self-representing PSU's.

The effect of dividing the 156 self-representing PSU's in NHIS and redefining the PSU's by using county boundaries resulted in a total of 397 PSU's: 198 of which were defined as self-representing and 199 of which were defined as non-self-representing and subsequently were used to form an additional 43 non-self-representing strata, which were combined with the other 220 non-self-representing PSU's in NHIS. The average population of a self-representing PSU was reduced from 838,000 to 584,000. In area, the average size of these PSU's was reduced more than 60 percent, from 2,185 square miles to 855 square miles.

These 461 first-stage units (NHIS strata) were further stratified into a total of 64 superstrata, and one PSU was selected from each of the superstrata using a modified Goodman-Kish controlled selection technique.[57] These 64 PSU's represented the geographic locations visited by the MEC's during the survey period.

The U.S. Bureau of the Census had the major responsibility for selecting households and sample persons within each of the PSU's. Three sampling frames of housing units were used to select the sample within each of the PSU's. The list frame consisted of all housing units based on the 1970 Census of the Population. An area frame was used in areas with "rapid" growth (housing units built prior to 1970) and in areas with "slow" growth (all housing units regardless of year built). A new construction frame was used to supplement the list frame

NOTE: A list of references follows the text.

for all places built since 1970 and in about half of the places in the area sample that were experiencing rapid growth.

The second stage of the design consisted of the selection of clusters of households (segments) within ED's. An ED is a geographical area that contains approximately 300 housing units. To oversample persons with low incomes, the ED's were stratified into a poverty stratum and a nonpoverty stratum. The poverty strata contained ED's with 13 percent or more of persons below the poverty level, and the nonpoverty strata contained ED's with less than 13 percent of persons below the poverty level as determined by the 1970 Census. ED's within each stratum were selected proportionally to their measures of size. To insure sampling reliability, clusters of 16 listed addresses were drawn from the sampling frames and then systematically subsampled at a rate of 1 out of 2 to produce a final segment of 8 household address listings.

At the third stage of sampling, a list of all eligible sample persons was made within each selected segment. Using the following sampling rates, the sample of persons to be examined was selected so that the younger and older age groups were oversampled and so that approximately one person per sample household was selected:

Age	*Rate*
6 months–5 years	¾
6–59 years	¼
60–74 years	¾

Of the 27,801 persons included in the NHANES II sample, 20,322 (73.1 percent) were interviewed and examined. The NHANES II sample size and response data by age, sex, and race are shown in table I. Table II shows the number of examined persons and population estimates at the midpoint of the survey by race according to sex and age.

A more complete description of the survey design is included in *Vital and Health Statistics,* Series 1, No. 15.[14]

Estimation procedures

Because the design of NHANES II is a complex, multistage probability sample, national estimates are derived through a multistage estimation procedure. The procedure has three basic components: (1) inflation by the reciprocal of the probability of selection, (2) adjustment for nonresponse, and (3) poststratification by age, sex, and race. A brief description of each component follows:

- *Inflation by the reciprocal of the probability of selection.* The probability of selection is the product of the probabilities of selection from each stage of selection in the design—PSU, segment, household, and sample person.
- *Adjustment for nonresponse.* The estimates are inflated by a multiplication factor that brings estimates based on examined persons up to a level that would have been achieved if all sample persons had been examined. The nonresponse adjustment factor was calculated by dividing the sum of the reciprocals of the probability of selection for all selected sample persons within each of five income groups (under $6,000, $6,000–$9,999, $10,000–$14,999, $15,000–$24,999, and $25,000 and over), three age groups (6 months–5 years, 6–59 years, and 60–74 years), four

NOTE: A list of references follows the text.

Table I. Sample size and response rates by age, sex, and race: National Health and Nutrition Examination Survey, 1976–80

Age, sex, and race	*Total sample size*	*Interview and examination status*			
		Interviewed[1]		*Examined*	
		Number	*Percent*	*Number*	*Percent*
Total	27,801	25,286	90.95	20,322	73.10
Age					
6 months–11 months	444	431	97.1	356	80.2
1–5 years	4,625	4,445	96.1	3,762	81.3
6–11 years	2,085	1,963	94.2	1,725	82.7
12–17 years	2,438	2,304	94.5	1,975	81.0
18–24 years	2,713	2,537	93.5	2,054	75.7
25–34 years	3,031	2,773	91.5	2,237	73.8
35–44 years	2,236	2,005	89.7	1,589	71.1
45–54 years	2,149	1,866	86.8	1,453	67.6
55–64 years	3,868	3,330	86.1	2,556	66.1
65–74 years	4,212	3,632	86.2	2,615	62.1
Sex					
Female	14,395	13,122	91.2	10,339	71.8
Male	13,406	12,164	90.7	9,983	74.5
Race					
White	23,537	21,350	90.7	17,105	72.7
Black	3,653	3,389	92.8	2,763	75.6
Other	611	547	89.5	454	74.3

[1]Completed medical history interview.

Table II. Number of examined persons and estimated population, by race, age, and sex of the examinee: United States, 1976–80

Age and sex	All races[1]		White		Black	
	Examined persons	Population in thousands[2]	Examined persons	Population in thousands[2]	Examined persons	Population in thousands[2]
Both sexes						
6 months–74 years	20,322	203,554	17,105	174,426	2,763	23,853
6–11 months	356	1,599	285	1,300	62	238
1–5 years	3,762	15,263	1,979	12,342	661	2,346
6–11 years	1,725	20,880	1,397	17,264	288	3,094
12–17 years	1,975	24,084	1,610	20,027	321	3,435
18–24 years	2,054	27,448	1,738	23,362	268	3,406
25–34 years	2,237	32,752	1,901	28,357	284	3,499
35–44 years	1,589	23,651	1,379	20,392	173	2,527
45–54 years	1,453	23,032	1,264	20,235	162	2,259
55–64 years	2,556	20,350	2,262	18,243	264	1,760
65–74 years	2,615	14,496	2,290	12,906	280	1,288
Male						
6 months–74 years	9,983	99,062	8,389	85,009	1,341	11,171
6–11 months	179	819	130	646	42	121
1–5 years	1,964	7,802	1,533	6,263	357	1,186
6–11 years	885	10,646	725	8,768	136	1,554
12–17 years	1,039	12,241	853	10,133	157	1,718
18–24 years	988	13,275	846	11,442	121	1,533
25–34 years	1,067	15,895	901	13,864	139	1,546
35–44 years	745	11,367	653	9,808	70	1,112
45–54 years	690	11,114	617	9,865	62	1,044
55–64 years	1,227	9,607	1,086	8,642	129	801
65–74 years	1,199	6,297	1,045	5,576	128	555
Female						
6 months–74 years	10,339	104,492	8,716	89,418	1,422	12,682
6–11 months	117	780	155	654	20	117
1–5 years	1,798	7,461	1,446	6,078	304	1,160
6–11 years	840	10,234	672	8,496	152	1,540
12–17 years	936	11,843	757	9,893	164	1,717
18–24 years	1,066	14,173	892	11,919	147	1,873
25–34 years	1,170	16,856	1,000	14,494	145	1,953
35–44 years	844	12,284	726	10,584	103	1,415
45–54 years	763	11,918	647	10,369	100	1,215
55–64 years	1,329	10,743	1,176	9,601	135	959
65–74 years	1,416	8,198	1,245	7,329	152	733

[1]Includes data for races not shown separately.
[2]At the midpoint of the survey, March 1, 1978.

geographic regions, and within or outside SMSA's by the sum of the reciprocals of the probability of selection for *examined* sample persons in the same income, age, region, and SMSA groups. The percent distribution of the nonresponse adjustment factors is shown in table III.

- *Poststratification by age, sex, and race.* The estimates were ratio adjusted within each of 76 age-sex-race cells to independent estimates, provided by the U.S. Bureau of the Census, of the population as of March 1, 1978, the approximate midpoint of the survey. The ratio adjustment used a multiplication factor in which the numerator was the U.S. population and the denominator was the sum of the weights adjusted for nonresponse for examined persons. This ratio estimation process brings the population estimates into agreement with the U.S. Bureau of the Census estimates of the civilian noninstitutionalized population of the United States, and, in general, reduces sampling errors of NHANES II estimates.

Table III. Percent distribution of the nonresponse adjustment factors: National Health and Nutrition Examination Survey, 1976–80

Size of factor	Percent distribution
Total	100.0
1.00–1.24	26.8
1.25–1.49	54.8
1.50–1.74	10.9
1.75–1.99	4.4
2.00–2.49	2.2
2.50–2.99	0.9

Nonresponse bias

In any health examination survey there exists the potential for three levels of nonresponse: (1) household interview nonresponse, (2) examination nonresponse, and (3) item nonresponse. Household interview nonresponse is defined as those sample

persons who do not complete the household medical history questionnaire. Examination nonresponse is those sample persons who initially respond to the household demographic questions and some or all of the medical history questionnaire but who subsequently do not come to the examination center for an examination. Item nonresponse results from sample persons who do not complete some portion of either the household interview questionnaires or the examination protocol, or, to a small degree, results from loss of blood specimens during shipment and processing. Intense efforts were undertaken during NHANES II to develop and implement procedures and inducements that would reduce all types of nonresponse and thereby reduce the potential for bias in the survey estimates. These procedures are discussed in "Plan and operation of the second National Health and Nutrition Examination Survey, 1976–80, *"Vital and Health Statistics,* Series 1, No. 15.[14]

In NHANES II, 9 percent (table I) of sample persons refused to give medical history interviews and, despite intense efforts, 20 percent gave medical history interviews but refused to be examined. Overall, 27 percent (table I) of the 27,801 persons selected for NHANES II were not examined. However, a comparison of the 1976 NHIS[58] and NHANES II[59] suggests that there is not a large nonresponse bias in some health-related variables because of the close agreement on selected interview items in NHANES II data with comparable items in the 1976 NHIS data. The 1976 NHIS data were used for the comparison because that survey included questions on diabetes (of interest in NHANES II) and because the nonresponse was 4 percent, assumed to be randomly distributed throughout the population.

Evidence from earlier studies also suggests no substantial nonresponse bias. An analysis of data on examined and nonexamined (but interviewed) persons was done using the first 35 stands of NHANES I.[60] It was found that the two groups were quite similar with respect to the health characteristics that were being compared. In another study of examined and nonexamined persons selected for participation in NHANES I, no differences were found between the two groups with respect to health-related variables.[61] In another study[62] factors relating to response in cycle I of the National Health Examination Survey of 1960–62 were investigated. It was found that 36 percent of the nonexamined persons in that survey viewed themselves as being in excellent health compared with 31 percent of examined persons. A self-appraisal of being in poor health was made by 5 percent of nonexamined persons and by 6 percent of those who were examined.

In a different study of cycle I[63] comparisons between two extreme groups—those who participated in the survey with no persuasive effort and those who participated only after a great deal of persuasive effort—differences between the two groups generally had little effect on estimates based on numerous selected examination and questionnaire items. This was interpreted as evidence that no large bias exists between the two groups for the items investigated and was offered as further support for the belief that there is little bias introduced to the findings because of differences in health characteristics between examined and nonexamined persons.

All NHANES II sample persons ages 6 months–6 years and a half-sample of those ages 7–74 years were to have had blood lead determinations. However, 39.3 percent of these sample persons had missing lead values due to nonresponse at various stages of participation in the survey. The rate of nonresponse was greater among preschool-aged children than among youths or adults (table IV). About half (51.0 percent) of the children ages 6 months–5 years compared with 28.6 percent of persons ages 6–17 years and 35.7 percent of adults ages 18–74 years had no blood lead determinations. Among medically examined persons in the lead subsample (table V), those with missing blood lead values were randomly distributed by demographic (other than age) and socioeconomic categories.

Description of exclusions and of respondents pertaining to tests for lead poisoning

Blood lead data from blood specimens drawn by fingerstick (pricking of the fingertip) and from extreme cases of lead exposure (blood lead values of 70 micrograms per deciliter (μg/dl) or more) were excluded from computations of national estimates. A description of blood lead levels for persons receiving fingersticks is given in table VI. Seventy-five percent of the 113 children with blood lead values who received fingersticks were

NOTE: A list of references follows the text.

Table IV. Nonresponse among sample persons 6 months–74 years in the lead subsample by age: National Health and Nutrition Examination Survey, 1976–80

Age	Number of sample persons								Percent of sample persons in the lead subsample without lead values	Percent of examinees in the lead subsample without lead values
						Examined but missing blood lead values				
	In lead subsample	Interviewed	Not interviewed	Examined	Not examined	Total	Refused to give blood specimen	Blood specimen drawn[1]		
All ages	16,563	15,179	1,384	12,288	2,891	2,239	1,197	1,042	39.3	18.2
6 months–5 years	5,069	4,876	193	4,118	758	1,634	988	646	51.0	39.7
6–17 years	2,413	2,261	152	1,967	294	245	122	123	28.6	12.5
18–74 years	9,081	8,042	1,039	6,203	1,839	360	87	273	35.7	5.8

[1]By venipuncture or fingerstick.

Table V. Nonresponse among examined persons 6 months–74 years in the lead subsample by age, race, sex, income, and degree of urbanization: National Health and Nutrition Examination Survey, 1976–80

Demographic variable	Age											
	6 months–74 years			6 months–5 years			6–17 years			18–74 years		
	Number of persons examined	Examined persons with missing lead values		Number of persons examined	Examined persons with missing lead values		Number of persons examined	Examined persons with missing lead values		Number of persons examined	Examined persons with missing lead values	
		Number	Percent		Number	Percent		Number	Percent		Number	Percent
Total	12,288	2,239	18.2	4,118	1,634	39.7	1,967	245	12.5	6,203	360	5.8
Race												
White	10,253	1,806	17.6	3,264	1,311	40.2	1,616	192	11.9	5,373	303	5.6
Black	1,737	367	21.1	723	269	37.2	313	48	15.3	701	50	7.1
Other	298	66	22.1	131	54	41.2	38	5	13.2	129	7	5.4
Sex												
Male	6,123	1,119	18.3	2,143	840	39.2	1,022	119	11.6	2,958	160	5.4
Female	6,165	1,120	18.2	1,975	794	40.2	945	126	13.3	3,245	200	6.2
Annual family income												
Under $6,000	2,291	404	17.6	752	281	37.4	268	37	13.8	1,271	86	6.8
$6,000–$14,999	5,082	994	19.6	1,876	739	39.4	780	107	13.7	2,426	148	6.1
$15,000 or more	4,509	758	16.8	1,368	562	41.1	852	91	10.7	2,289	105	4.6
Unknown	406	83	20.4	122	52	42.6	67	10	14.9	217	21	9.7
Degree of urbanization												
Large urban[1]	2,993	583	19.5	949	391	41.2	483	69	14.3	1,561	123	7.9
Smaller urban[2]	4,805	869	18.1	1,647	639	38.8	721	81	11.2	2,437	149	6.1
Rural	4,490	787	17.5	1,522	604	39.7	763	95	12.5	2,205	88	4.0

[1]1 million or more persons.
[2]Fewer than 1 million persons.

Table VI. Blood lead determinations from specimens collected by fingersticks in children 6 months–7 years, by race: National Health and Nutrition Examination Survey, 1976–80

Race	Number examined	Mean	Standard deviation	Mode	Median	Minimum	Maximum	Skewness
		Blood lead level in micrograms per deciliter						
All races[1]	113	24.8	15.4	18.0	22.2	7.0	116.0	3.5
White	77	23.2	17.6	18.0	19.0	7.0	116.0	3.7
Black	36	28.3	8.0	27.0	28.0	12.0	47.0	0.1

[1]Includes data for races not shown separately.

under 3 years of age. Table VII shows the characteristics of three persons receiving venipunctures who had blood lead values greater than 70 μg/dl. Each of these three individuals was referred to his personal physician for medical attention.

The distribution of blood lead levels for examinees ages 6 months–5 years according to responses to medical history questions about previous tests for lead poisoning is presented in table VIII. Approximately 16 percent of 123 examinees who reported being tested for lead poisoning had blood lead levels equal to or geater than 30 μg/dl. Of these 20 examinees, only 6 reported that the test indicated they had lead poisoning or high lead levels. Five of these six examinees were treated for lead poisoning. The blood lead levels of the respondents at the time they were previously tested for lead poisoning were not reported.

Subsequent to constructing tables 1–11 and the release of a public use data tape, codes on 7 of the 2,372 examined children 6 months–5 years of age were changed—3 from venipuncture to fingerstick and 4 from fingerstick to venipuncture. The overall findings and conclusions were not affected by these coding changes. The data listed below are those of the 7 cases and are offered to help users of the data tape should they wish

Table VII. Characteristics of three persons with blood lead values greater than 70.0 μg/dl who received venipuncture: National Health and Nutrition Examination Survey, 1976–80

Blood lead value	Demographic factors				
	Age in years	Sex	Race	Family income	Degree of urbanization
76.0 μg/dl	1	Male	Black	Under $6,000	Rural
80.0 μg/dl	42	Male	White	$15,000 or more	Large urban[1]
90.0 μg/dl	18	Male	Black	Under $6,000	Smaller urban[2]

[1]1 million or more persons.
[2]Fewer than 1 million persons.

NOTE: μg/dl = micrograms per deciliter.

Table VIII. Summary of history of being tested and treated for lead poisoning for children 6 months–5 years, by blood lead level: National Health and Nutrition Examination Survey, 1976–80

Medical history items	Total	PbB ≥ 30		20 ≤ PbB < 30		PbB < 20	
		Number	Percent	Number	Percent	Number	Percent
Not tested for lead poisoning	2,226	96	4.3	453	20.4	1,677	75.3
Tested for lead poisoning	123	20	15.6	42	34.4	61	50.0
How long ago tested:							
0–3 months	26	7		7		12	
4–6 months	12	1		7		4	
7–9 months	12	4		4		4	
10–12 months	2	-		2		-	
1–4 years	71	8		22		41	
Test results positive[1]	19	6		5		8	
Treated for lead poisoning	9	5		2		2	
How long ago treated:							
0–3 months	1	1		-		-	
4–6 months	2	2		-		-	
1–3 years	6	2		2		2	

[1]Indicated child had lead poisoning or high lead levels.

NOTE: ≥ = equal to or greater than.
≤ = equal to or less than.
< = less than.

to generate similar tables:

- **Codes changed from venipuncture to fingerstick:**
  Sequence no. 02618: blood lead level = 20 μg/dl
  Sequence no. 03678: blood lead level = 20 μg/dl
  Sequence no. 05053: blood lead level = 20 μg/dl
- **Codes changed from fingerstick to venipuncture:**
  Sequence no. 02025: blood lead level = 24 μg/dl
  Sequence no. 08295: blood lead level = 20 μg/dl
  Sequence no. 08310: blood lead level = 11 μg/dl
  Sequence no. 08312: blood lead level = 8 μg/dl

Limitations of the data

Rigorous quality control methods were implemented throughout specimen collection and processing and in data processing to insure validity and accuracy of the results reported. However, there are some factors that might affect the data. Foremost is the relative imprecision of a measurement or measurement error. Based on an analysis of the quality control pools,[17] the coefficients of variation for the laboratory methods used are approximately 15.0 and 12.0 percent for control pools with low (less than 30 μg/dl) and high (30 μg/dl or more) mean lead levels, respectively. The potential effect of measurement error on population estimates is discussed in appendix II.

A possible logistical factor indirectly influencing the blood lead data is the itinerary of the MEC's. To minimize the effects of adverse weather conditions on response rates, MEC's were set up in the Northern States during the summer and more Southern States during the winter. The potential environmental effects on blood lead levels associated with seasonality[64] and geographic location may be confounded, to some undetermined degree, with those associated with degree of urbanization of place of residence. For this reason, the effects associated with seasonality were taken into account in the regression analysis of the chronological trend in blood lead levels. (See appendix IV.)

Measures of variability

Because the statistics presented in this report are based on a sample, they will differ somewhat from the figures that would have been obtained if a complete census had been taken using the same survey instruments, instructions, interview and examination personnel, and procedures. The probability design of this survey permits the estimation of standard errors and standard deviations that are appropriate for the design and weighted estimates shown in this report.

Standard errors and standard deviations are distinct concepts. The standard error is primarily a measure of the variation inherent in the process of estimating a population mean from a sample mean. As calculated for this report, the standard error also reflects part of the variation that arises in the measurement process. It does not include estimates of any bias that might be contained in the data. The chances are about 68 out of 100 that an estimate based on a sample using the same procedures and instruments would differ from the value obtained from a complete census by less than the standard error. The chances are about 95 out of 100 that the difference would be less than twice the standard error and about 99 out of 100 that it would be less than 2½ times as large.

The estimates of standard errors in the text and detailed tables were calculated using a Taylor series linearization method.[24] This process approximates the variance of statistics, for example, means and proportions, using the first two terms of a Taylor series expansion. If the higher order terms of the expansion are negligible and the sample is of a reasonable size for the domains of interest, then this approximation provides variance estimates as reliable as those from the pseudoreplication method adapted for use in the analyses of NHANES II data.[65,66] It should be noted that the estimates of standard errors are themselves subject to errors that may be large if the number of cases or the number of PSU's involved in the calculation of variances are small.

The standard deviation, on the other hand, is a measure of the dispersion of the observations in a sample, and is useful in describing how an individual observation compares with the mean of the sample. As calculated for this report, it also reflects part of the variation that arises in the measurement process. If the data are normally distributed (that is, Gaussian), then one standard deviation from the mean (in either direction) encompasses approximately 68 percent of the distribution; two standard deviations, about 95 percent; and 2½ standard deviations, about 99 percent.

The estimates of standard deviations presented in the tables were calculated by using the pseudoreplication method, a balanced half-sample replication technique that is based on variability among random subsamples of the total sample.[65,66]

Data reliability

The criteria for reliability of estimates shown in this report consisted of the following: (1) that the sample size, on which the estimate is based, is at least 25 persons and (2) that the estimated coefficient of variation (i.e., the standard error of the mean divided by the mean) is less than 30 percent. Thus if the sample size was too small or if the variation regarding the mean was too large, an asterisk was placed next to the value on the table. This estimate is considered neither precise nor stable enough to meet reliability standards; however, the values are shown to give an impression of the observed distribution and to permit users to combine data into useful categories.

Tests of significance

Hypothesis testing and tests of significance were conducted using two computer programs, SURREGR[25] and GENCAT.[26,27] The former was used for regression analysis and the latter for generalized categorical data analysis. All tests accounted for the complex survey sample design. Unless otherwise specified, tests were conducted using a probability level of 0.05.

NOTE: A list of references follows the text.

Appendix II
Statistical analysis of the effects of analytic error on national estimates

The effect of analytic error (in blood lead determination) on national prevalence estimates of elevated blood lead values (30 μg/dl or more) from the second National Health and Nutrition Examination Survey (NHANES II) is the topic of this appendix. The predicted effect of analytic error (due to sampling of blood and laboratory measurement error) on NHANES II national estimates is compared with that predicted from computations presented by Lucas.[67] Also, potential bias in estimating analytic error associated with systematic deviations in the NHANES II quality control blood lead measures is evaluated and discussed.

Background

Following the lognormal model and definitions of parameters in the model used by Lucas,[67] the total population variance of blood lead levels can be partitioned into two major components: the population variance of the true blood lead values σ_t^2 and the analytic variance σ_a^2. The variance of the true blood lead values is composed of individual-to-individual variation in blood lead levels and variation in an individual's blood lead level over time. The analytic variance includes variation in blood lead levels attributable to the sampling of an individual's blood at a fixed point in time σ_s^2 and to laboratory measurement error σ_m^2. Lucas[67] gives a more detailed description of these variance components.

The degree to which analytic error influences national estimates of the percent of persons with blood lead levels of 30.0 μg/dl or more from the NHANES data was examined using two different statistical techniques. The first technique (referred to as analysis no. 1) was a semiparametric approach using a discriminant function to estimate the probability of misclassification.[68] This procedure was used to estimate classification probabilities above and below the threshold (30.0 μg/dl WB) for each measured blood lead level from venous blood specimens collected in NHANES II. The second technique (referred to as analysis no. 2) was the parametric approach described by Lucas.[67] This latter procedure involved computing Z values and the correlation between measured and true blood lead values. Estimates of classification probabilities above and below the threshold were then obtained from tables of the bivariate normal distribution.

For both analyses, it was assumed that the relationship between measured blood lead levels and true blood lead values can be described by a bivariate lognormal distribution (a bivariate normal distribution using the natural log blood lead values).

Model used for analysis

Following the lognormal model used by Lucas,[67] it is assumed that the relationship between measured blood lead values and true blood lead values can be described as a bivariate lognormal distribution. Thus, if

$$Y = \ln(\text{true blood lead})$$
$$X = \ln(\text{measured blood lead})$$
$$\varepsilon = \ln(\text{analytical error})$$

then

$$X = Y + \varepsilon$$

where Y and ε are independent; with $Y \sim N(\mu_t, \sigma_t^2)$; where μ_t and σ_t^2 are the population mean and variance of the true blood lead values, respectively, and $\varepsilon \sim N(0, \sigma_a^2)$. It follows immediately that

$$X \sim N(\mu_t, \sigma_t^2 + \sigma_a^2)$$

Estimation of means and variance components from NHANES II blood lead data

Estimates of population mean and the total population variance (for example, $\sigma_t^2 + \sigma_a^2$) for selected groups are presented in table IX. These estimates were computed using weighted natural log transforms of the measured blood lead values. Population variances were estimated using a variant developed by Ron Forthofer and Robert Casady[69] of the balanced repeated replication (BRR) strategy described by McCarthy[70] and Kish and Frankel.[71] This procedure yields unbiased estimates of population variance.

For his computations, Lucas[67] partitioned the analytic variance σ_a^2 into two components: a sampling component denoted by σ_s^2 and a measurement component denoted by σ_m^2. For this study, the measurement error σ_m^2 was further partitioned into two components. The first, which we denote σ_r^2, measures variation between mean levels of measured blood lead on a day-to-day basis, while the second, denoted $\sigma_{d.r}^2$, measures the variation

NOTE: A list of references follows the text.

Table IX. Estimates used in analyses nos. 1 and 2: United States, 1976–80

Selected race, sex, and age groups	Estimated population parameters		
	$\hat{\mu}_t$	$\hat{\sigma}_t^2 + \hat{\sigma}_s^2$	$\hat{\sigma}_t^2$
All persons 6 months–74 years[1]	2.553	0.1670	0.1462
All children 6 months–5 years[1]	2.699	0.1509	0.1301
White	2.636	0.1334	0.1126
Black	2.974	0.1350	0.1142
All persons 6–17 years[1]	2.460	0.1368	0.1160
Men, 18–74 years[1]	2.759	0.1361	0.1153
Women, 18–74 years[1]	2.397	0.1439	0.1231

[1]Includes races other than white or black.

NOTE: $\hat{\mu}_t$ = population mean of the true blood lead values in units of the natural logarithm of the blood lead level.
$\hat{\sigma}_t^2$ = population variance of the true blood lead values in units of the natural logarithm of the blood lead level.
$\hat{\sigma}_a^2$ = analytic variance in units of the natural logarithm of the blood lead level.

of determinations within a day. As the measured blood level reported for NHANES II is the average of two determinations made within a day, the appropriate model is

$$\sigma_m^2 = \sigma_r^2 + \frac{\sigma_{d:r}^2}{2}$$

Using the lead data from the high and low blind quality control blood pools, our estimates of the above component parameters are $\hat{\sigma}_r^2 = 0.01012$ and $\hat{\sigma}_{d:r}^2 = 0.01385$; the estimate of σ_m^2 is, therefore, $\hat{\sigma}_m^2 = 0.01704$.

Unfortunately, only one sample of blood was taken from each NHANES II respondent so it was impossible to estimate the parameter σ_s^2 directly from the NHANES II data. However, the blood sampling methodology used by Griffin et al.[72] was similar to the venipuncture method used in the NHANES II. An analysis of the Griffin data, using the methodology developed by Snee and Smith[73] provided an overestimate (the expected value of the estimate contained both the time component and the sampling component) of the sampling variance component. The overestimate of σ_s^2 based on Griffin's data is $\hat{\sigma}_s^2 = 0.00379$.

Assuming the variance component resulting from sampling for NHANES II is similar to that for the Griffin study,[72] the estimate of analytic variance $\hat{\sigma}_a^2$ for NHANES II is

$$\begin{aligned}\hat{\sigma}_a^2 &= \hat{\sigma}_s^2 + \hat{\sigma}_m^2 \\ &= 0.00379 + 0.01704 \\ &= 0.02083\end{aligned}$$

Subtracting the above estimate from the total population variance estimates yields the estimates of σ_t^2 shown in table IX.

NOTE: A list of references follows the text.

Computational methods of analysis no. 1

Given the assumptions of the model, the true and measured values, Y and X, respectively, are bivariate lognormal with a correlation of $\sigma_t/\sqrt{\sigma_t^2 + \sigma_a^2}$. Therefore, the expected true blood lead value given the measured value X can be expressed as

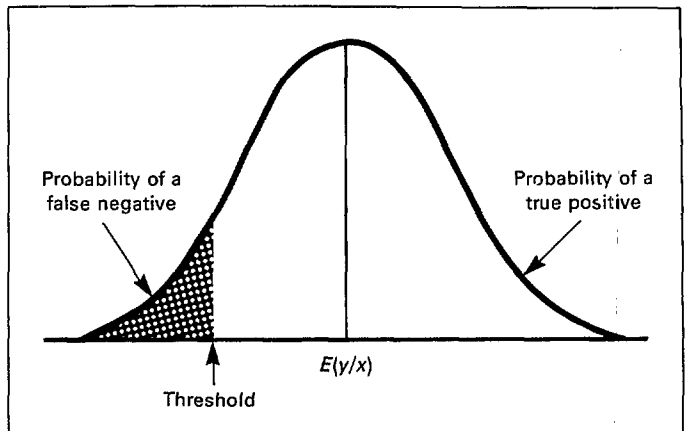
$$E(Y/X) = \mu_t + \frac{\sigma_t^2}{\sigma_t^2 + \sigma_a^2}(X - \mu_t)$$

and the conditional variance is

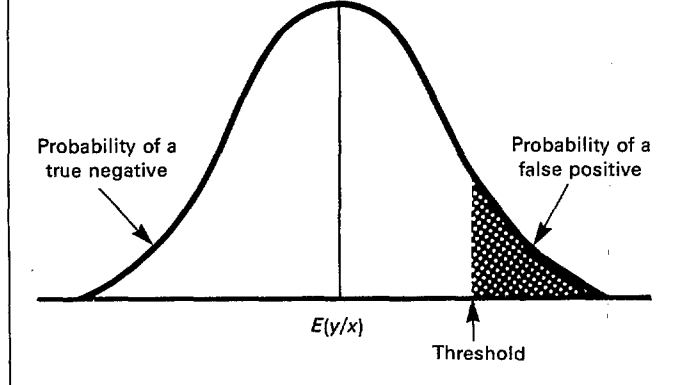
$$\text{Var}\,(Y/X) = \sigma_t^2\left(1 - \frac{\sigma_t^2}{\sigma_t^2 + \sigma_a^2}\right)$$

Using these formulas the true probability that a measured blood lead level exceeds the given threshold of 30.0 μg/dl of whole blood can be computed as the area under the normal curve with mean $E(Y/X)$ and variance Var (Y/X).

In figure I the correction for misclassification error caused by analytic variance is illustrated. Case 1 represents the probability of misclassification of a conditional expected blood lead level that lies above the threshold value. With the assumption that the distribution of analytic error about the conditional mean is normal, and using the conditional estimate of the variance (Var (Y/X)), then the area under that normal curve that



Case 1: Computing probability of true exceedence with expected positive



Case 2: Computing probability of true exceedence with expected negative

Figure I. Estimating probabilities of elevated blood lead levels from observed levels

lies to the left of the threshold is an estimate of the probability that the examinee's blood lead value is observed to be less but is truly greater (OLTG) than the threshold. This is often referred to as the probability of a false negative. The area under the normal curve that lies to the right of the threshold is an estimate of the probability that the examinee's blood lead value is observed to be greater and is truly greater (OGTG).

The opposite occurs when the conditional expected blood lead level lies below the selected threshold (case 2, figure I). The area under the normal curve to the left of the given threshold is an estimate of the probability that the examinee's blood lead value is observed to be less and is truly less (OLTL) than the selected threshold. The area under the curve to the right of the threshold is the probability that the examinee's blood lead value is observed to be greater but is truly less (OGTL) than the threshold; this is commonly referred to as a false positive.

After these probabilities were computed for each measured blood lead value, they were multiplied by the NHANES II sample weights and summed within each of the four possible categories: OLTG, OLTL, OGTG, and OGTL. Subsequently, an estimate of the percent of persons in the population with blood lead levels truly greater than the threshold of 30.0 μg/dl (after accounting for the effects of analytic error) was obtained by

$$\frac{\text{OLTG} + \text{OGTG}}{\text{OLTG} + \text{OLTL} + \text{OGTG} + \text{OGTL}} \times 100$$

Computational methods of analysis no. 2

Using the assumptions of the model and the estimated values of the parameters, estimates of the percent truly greater than a threshold of 30 μg/dl were obtained using the method proposed by Lucas.[67]

Results

The results of analyses nos. 1 and 2 were very similar. These results are presented as the estimated percent truly greater (than a threshold of 30 μg/dl) in table X. In general, the percent truly greater than this threshold was approximately 24 percent less than the prevalence of blood lead levels of 30 μg/dl or more estimated from the weighted NHANES II data. This effect is substantially less than that predicted by Lucas.[67] For a population with a geometric mean of 15 μg/dl, Lucas predicted 95 percent false positives (observed greater but truly less than a threshold of 30 μg/dl) and 0.04 percent false negatives (observed less but truly greater than a threshold of 30 μg/dl). Overall, this study predicted about the same percent of false positives but substantially more (1.4 percent) false negatives.

While the lognormal model proposed by Lucas[67] may have general application for studies involving blood lead determination, the various parameters in the model depend upon factors inherent to the particular study of interest. For example, the sampling variance components are expected to differ using the fingerstick versus the venipuncture method for blood collection. Likewise, the measurement variance component might be quite different depending on the laboratory method of blood lead determination; that is, atomic absorption spectrophotometry, dithizone, anodic stripping voltammetry or isotopic dilution mass spectrometry.

NOTE: A list of references follows the text.

Table X. Blood lead levels of persons 6 months–74 years, with mean and standard deviation of the mean, percent of persons with levels of 30 μg/dl or more, and estimated percents of persons with levels above the threshold of 30 μg/dl after accounting for analytic error, by selected characteristics: United States, 1976–80

Characteristic	Estimated population in thousands[1]	Number examined[2]	Arithmetic		Geometric		Estimated[3] prevalence of PbB ≥ 30.0 μg/dl	Analysis no. 1: estimated percent truly greater	Analysis no. 2: estimated percent truly greater
			Mean	Standard deviation	Mean	Standard deviation			
			Micrograms per deciliter				Percent ± SEP[4]		
All persons, 6 months–74 years[5]	203,554	9,936	13.9	6.05	12.8	1.51	1.9 ± 0.2	1.4	1.5
All children, 6 months–5 years[5]	16,862	2,376	16.0	6.56	14.9	1.48	4.0 ± 0.5	2.9	2.9
White	13,641	1,876	14.9	5.60	14.0	1.44	2.0 ± 0.3	1.3	1.3
Black	2,584	420	20.9	8.18	19.6	1.44	12.2 ± 1.5	10.6	11.3
All persons, 6–17 years[5]	44,964	1,720	12.5	4.68	11.7	1.45	0.5 ± 0.2	0.3	0.3
Men, 18–74 years[5]	67,555	2,798	16.9	6.76	15.8	1.45	4.2 ± 0.5	3.1	3.3
Women, 18–74 years[5]	74,173	3,045	11.8	4.64	11.0	1.46	0.5 ± 0.2	0.3	0.3

[1]At the midpoint of the survey, March 1, 1978.
[2]With lead determinations from blood specimens drawn by venipuncture.
[3]From the weighted NHANES II blood lead data.
[4]Standard error of the percent.
[5]Includes data for races other than white and black.

NOTE: μg/dl = micrograms per deciliter.

It is inappropriate to apply the computations of Lucas[67] to estimate the effect of analytic error on NHANES II national estimates. The classification probabilities above and below given thresholds were computed using variance components estimated from the NHANES II data, whereas Lucas used variance components estimated from studies[73,74] in which the protocols differed from one another and from that of NHANES II.

The results of analyses nos. 1 and 2 agreed with Lucas[67] concerning the effects of analytic error in relation to the magnitude of the geometric mean. Analytic error had less effect on the prevalence of blood lead levels of 30 μg/dl or more for the population subgroups with higher geometric mean values. For instance, for black children (with a geometric mean of 19.6 μg/dl) and for white children (with a geometric mean of 14.0 μg/dl) (table X), the estimated percents truly greater than 30 μg/dl from analysis no. 1 were approximately 87 and 65 percent of the prevalence of blood lead levels of 30 μg/dl or more, respectively.

NOTE: A list of references follows the text.

Examination of potential bias in estimating analytic error

Analytic error can result from lack of quality control at four different stages of lead analysis: (1) collection and preparation of the blood sample prior to laboratory assessment; (2) laboratory manipulation of the blood sample, physically and chemically, prior to delivery to a given instrumentation system; (3) instrumentation, quantitation, and calibration methods used to determine lead levels; and (4) establishment of relevant criteria for accuracy and precision through internal or external quality assurance checks, or both. Sources of contamination and error in determining lead concentrations in blood specimens at each of these stages are discussed elsewhere.[75] The NHANES II lead analysis was tightly controlled in each of these areas according to a very strict protocol.[21] If there were systematic errors caused by deviation in the protocol they would show up in an examination of the blind quality control blood samples.

To investigate the potential for systematic changes due to analytic error over the course of the NHANES II blood lead determinations, the blind quality control samples from the low

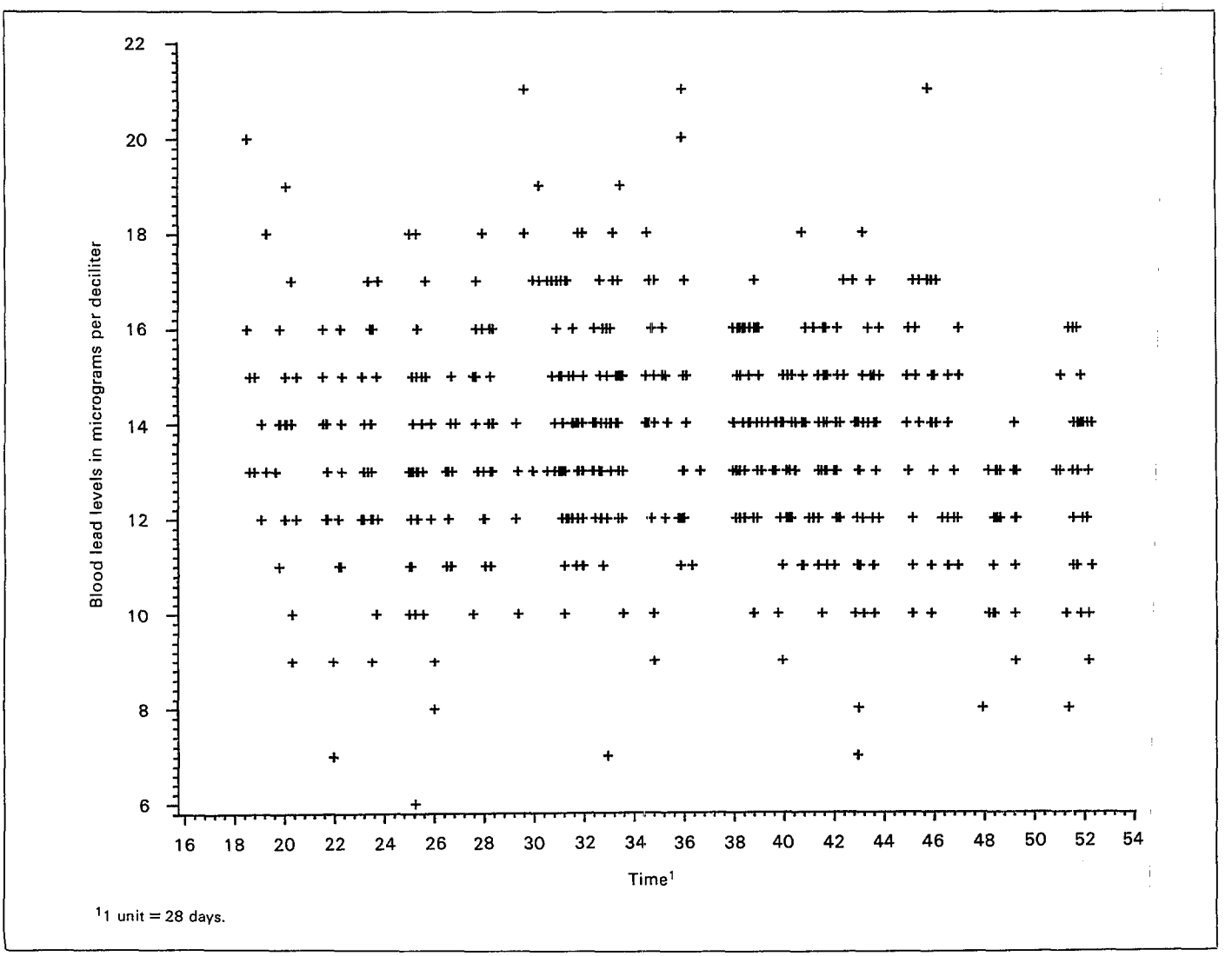


Figure II. Scattergram of blood lead levels determined from the low quality control blood pool by chronological time: National Health and Nutrition Examination Survey, 1976–80

and high bovine blood pools were examined with respect to a time variable. The time variable was defined to correspond to the chronology of collection of NHANES II blood samples analyzed in the same analytical (laboratory) run as the respective blind control sample. Figures II and III show scattergrams of the blood lead values by time for the low and high blind pools, respectively. (Note that blind quality control data from approximately 12 months of the NHANES II are not presented because of initial technical difficulties in labeling the blind specimens to be indistinguishable from the NHANES II specimens.) Estimates of the effect of analytic error on national estimates presented in the previous section could be biased if the average blind pool values changed significantly with time or if the measurement variance was heterogeneous over time. A statistical analysis of the blind control data within each pool suggested that neither the mean log blood lead values nor the estimates of measurement variance were significantly different among three selected time periods at the 0.05 level of probability. Table XI shows the mean log blood lead values, standard deviations of the means, and measurement variance estimates for three chronological time periods for the low and high blind pools.

In addition, the blind quality control lead values (untransformed) were examined with respect to time using regression analysis. Because the observed blood lead data from the low and high pools were approximately normally distributed, lead values in each pool were z transformed; that is (lead value − lead pool mean)/(lead pool standard deviation). The resulting data from the two pools, each having a mean of zero and a variance of one, were combined, and regressions of z on time alone and z on time and time-squared together were performed. When the linear term time was fit alone, there was no significant trend of transformed lead values (table XII).

Regression of z on the linear and quadratic time variables suggested a statistically significant curvilinear relationship between z and the chronological time variable (table XII). However, the regression coefficients associated with the linear and quadratic time variables were relatively small in magnitude. Also, less than 2 percent ($R^2 = 0.0186$) of the total variability of the transformed lead values was explained by the model.

The association between time and the blind quality control lead values in relation to the chronological downward trend in the NHANES II blood lead data are discussed elsewhere.[4] Findings reported in that paper indicate that the statistically significant 37 percent overall reduction in mean NHANES II blood lead levels from February 1976 through February 1980

NOTE: A list of references follows the text.

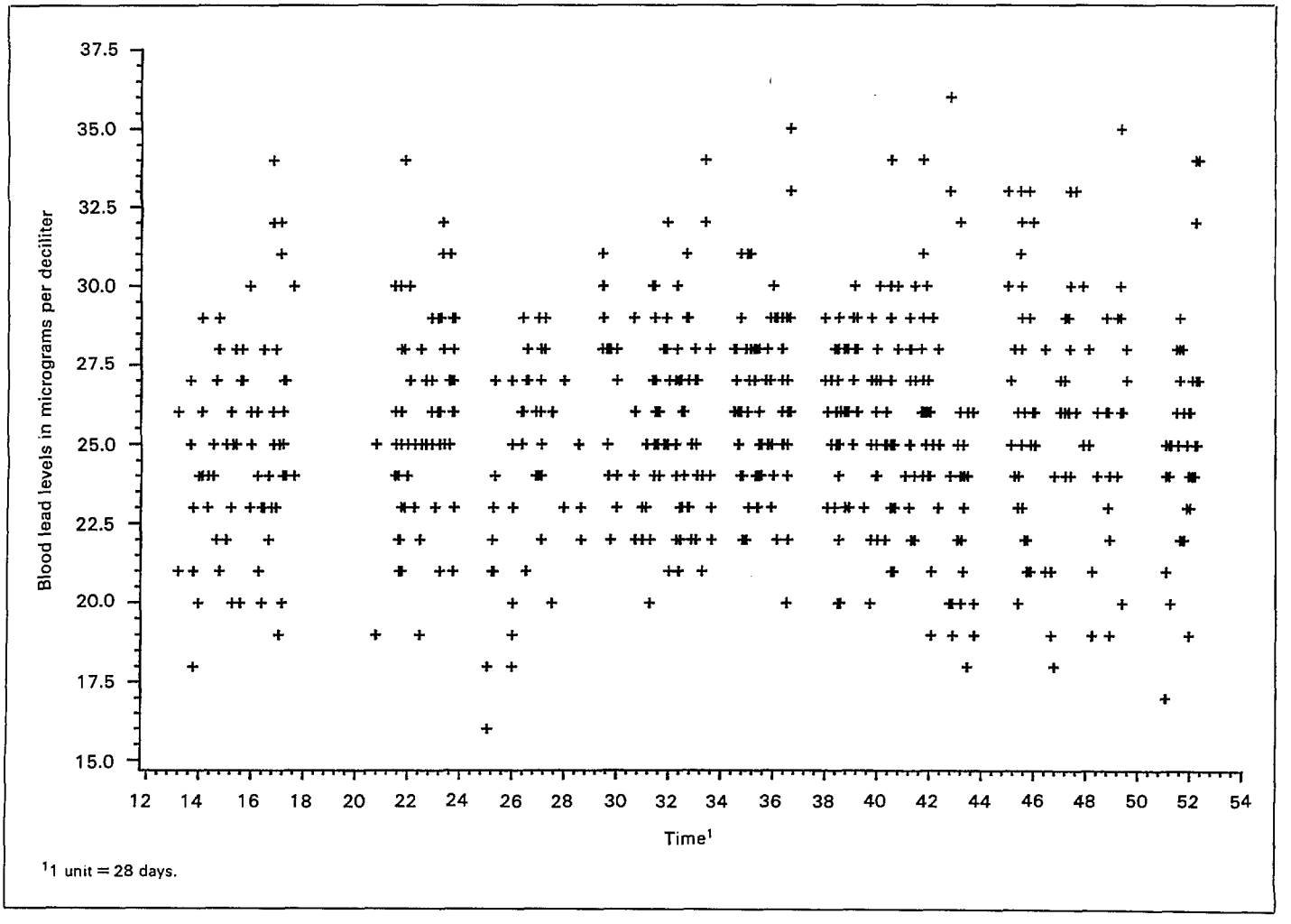


Figure III. Scattergram of blood lead levels determined from the high quality control blood pool by chronological time: National Heath and Nutrition Examination Survey, 1976–80

Table XI. Natural logarithm of blood lead levels for the blind quality control pools with mean, standard deviation of the mean, and estimated measurement variance by chronological time periods: National Health and Nutrition Examination Survey, 1976–80

Time period[1]	Low blind pool				High blind pool			
	Number	Mean	Standard deviation	σ_m^2	Number	Mean	Standard deviation	σ_m^2
		ln PbB in μg/dl				ln PbB in μg/dl		
First	72	2.60	0.178	0.0215	144	3.22	0.132	0.0118
Second	266	2.61	0.169	0.0208	246	3.24	0.118	0.0103
Third	188	2.56	0.177	0.0207	210	3.24	0.146	0.0162

[1]In 28-day periods from the beginning (time = 1) to the end (time = 52.4) of NHANES II.
13 ≤ first < 25
25 ≤ second < 40
40 ≤ third ≤ 52.4

NOTE: $\sigma_m^2 = \sigma_r^2 + \sigma_d^2/2$, where m = measurement; r = run; and d = determination.

ln = natural logarithm.
PbB = blood lead level.
μg/dl = micrograms per deciliter.
≤ = equal to or less than.
< = less than.

Table XII. Results of regression analysis of z on a chronological time variable: National Health and Nutrition Examination Survey, 1976–80

A. Dependent variable = z; independent variable = time; R-square = 0.0000

Source	Degrees of freedom	Sum of squares	Mean square	F value	Probability > F
Model	1	0.000	0.000	0.000	0.9954
Error	1,124	1,124.000	1.000	...	...
C total	1,125	1,124.000	...	...	...
Variable	**Degrees of freedom**	**Parameter estimate**	**Standard error**	**t for H_0: parameter = 0**	**Probability > |t|**
Intercept	1	0.000	0.1070	0.005	0.9956
Time	1	−0.000	0.0029	−0.006	0.9954

B. Dependent variable = z; independent variables = time and time squared; R-square = 0.0186

Source	Degrees of freedom	Sum of squares	Mean square	F value	Probability > F
Model	2	20.933	10.462	10.656	0.0001
Error	1,123	1,103.067	0.982	...	...
C total	1,125	1,124.000	...	...	...
Variable	**Degrees of freedom**	**Parameter estimate**	**Standard error**	**t for H_0: parameter = 0**	**Probability > |t|**
Intercept	1	−1.291	0.2992	−4.315	0.0001
Time	1	0.084	0.0185	4.558	0.0001
Time squared	1	−0.001	0.0003	−4.616	0.0001

NOTES: z = (blood lead value minus pool mean)/standard deviation of the pool mean.
Time corresponds to the chronology of collection of blood specimens analyzed in the same analytical laboratory runs.
> = greater than.
$|t|$ = absolute value of t.

is not attributable to the nominal systematic error in the blind quality control blood lead determinations.

Summary

While the lognormal model assumed by Lucas[67] could have general application to studies involving blood lead determinations, estimation of the various parameters in the model depends upon the sampling, measurement, and quality control protocols inherent in the study of interest. Therefore, to determine the possible effects of analytic error (due to sampling of blood and laboratory measurement error) on NHANES II national estimates, it was imperative to estimate these parameters using the NHANES II data. The misclassification probabilities of blood lead values above and below given thresholds reported by Lucas[67] were based on parameters in the model estimated from blood lead data obtained in studies with protocols notably different from NHANES II. Thus, the direct application of the misclassification probabilities presented by Lucas[67] to NHANES II national estimates is inappropriate.

The Lucas[67] model was applied using appropriate parameter estimates computed from the NHANES II data. The results show that accounting for analytic error could reduce the overall prevalence of elevated blood lead levels from NHANES II by 24 percent, as opposed to the 90-percent reduction predicted using Lucas' computations.

One source of analytic error not explicitly parameterized in the Lucas model is systematic deviation in laboratory de-

NOTE: A list of references follows the text.

terminations. Most protocols do not provide for the specific assessment of this component of analytic error. However, in the NHANES II, evidence of systematic error could be found by examining the blind quality control blood samples that were subjected to the survey's blood lead laboratory determination procedures. The application of statistical regression techniques to these NHANES II quality control data resulted in a finding that systematic errors were minimal. No significant trends in either mean log blood lead values or estimates of measurement variance were found across time in either the high or the low lead values from blind quality control blood pools.

Appendix III
Demographic and socioeconomic terms and dietary and medical history items

Age—Age was defined as age at last birthday at the time of the household interview.

Race—The race of each respondent was determined during the household interview. Race was observed and recorded as "white," "black," or "other." Other includes Japanese, Chinese, American Indian, Korean, Eskimo, and all races other than white and black. Persons of Mexican descent were included with "white" unless definitely known to be American Indian or of another race. Black persons and persons of mixed black and other parentage were recorded as black. When a person of mixed racial background was uncertain about his or her race, the race of the father was recorded. Data on other races are not presented separately in this report but are included in the "all races" category.

Sex—Sex was recorded by the interviewers and examiners.

Annual family income—The respondent was given a card listing 12 income categories and was instructed to select the one that represented his or her total combined family income for the past 12 months. Respondents were asked to include income from all sources such as wages, salaries, social security or retirement benefits, help from relatives, rent from property, and so forth.

Degree of urbanization of place of residence—Four urbanization classes were defined, based on the population of the place in which the examinee resided and, in some cases, on whether the examinee resided in the central city of a standard metropolitan statistical area (SMSA). These classes were (1) urbanized area with a population of 1,000,000 or more, in the central city of the SMSA; (2) urbanized area with a population of 1,000,000 or more, but not in the central city of the SMSA; (3) urbanized area under 1,000,000 population or urban place with a population of 2,500 or more but outside the urbanized area, either in or out of the SMSA; and (4) rural, including rural areas in extended cities and all incorporated or unincorporated areas with a population of less than 2,500.

Season—The four seasons were defined as follows:

Winter	December 21–March 20
Spring	March 21–June 20
Summer	June 21–September 20
Fall	September 21–December 20

Geographic region—The 48 contiguous States, the District of Columbia, Alaska, and Hawaii were stratified into four broad geographic regions, each of about the same population size. The compositions of the regions are as follows:

Region	*States included*
Northeast	Connecticut, Maine, Massachusetts, New Hampshire, Rhode Island, Vermont, New York, New Jersey, and Pennsylvania
Midwest	Ohio, Indiana, Michigan, Wisconsin, Illinois, Minnesota, Iowa, and Missouri
South	Delaware, District of Columbia, Maryland, Virginia, Kentucky, Tennessee, West Virginia, Alabama, Arkansas, Louisiana, Mississippi, Georgia, North Carolina, South Carolina, and Florida
West	California, Oregon, Washington, Texas, Arizona, Colorado, Idaho, Montana, Nevada, New Mexico, Oklahoma, Utah, Wyoming, Alaska, Hawaii, Kansas, Nebraska, North Dakota, and South Dakota

Education of the head of household—For each sample person interviewed, questions were asked pertaining to the head of the household. One such item was the highest grade or year of regular school that the head of the household attended. For presentation of blood lead data of children ages 6 months–17 years, three levels of educational status of the household head were defined. These categories are less than high school, high school, and college level or equivalent.

Occupation—Occupation was reported by the respondent during the household interview. These data were later converted to numeric codes by the U.S. Bureau of the Census, based on the *1970 Census of Population. Alphabetical Index of Industries and Occupations.*[76]

For analysis of blood lead level in relation to occupation, U.S. workers ages 18–74 years were partitioned into two groups based on their potential exposure to lead at the workplace. High and low potential exposure groups were defined using information on types of occupations with observed potential exposure to lead from the National Occupational Hazard Survey (NOHS) conducted from 1972 through 1974.[68] Examinees in the second National Health and Nutrition Examination Survey reporting agricultural occupations and those reporting a selected group of professional and semiprofessional occupations were excluded from analysis and tables of blood lead data presented by these occupational exposure categories. The former were excluded because farm-related occupations are not covered under the provisions of the Occupational Safety and Health

NOTE: A list of references follows the text.

Act of 1970 and thus were not part of the National Occupational Hazard Survey. The latter (professionals and semiprofessionals) were excluded because of difficulty in classifying them as either high or low potential exposure to lead in their work setting.

Smoking status—Smoking status was derived from questionnaire data reported by respondents, ages 18–74 years, during the household interview. Nonsmokers included those currently smoking neither cigarettes, pipe tobacco, nor cigars at the time of examination. Ex-smokers are included as nonsmokers. Smokers include those currently smoking cigarettes, pipe tobacco, or cigars at the time of examination. Cigarette smokers include those currently smoking cigarettes at the time of examination, whether or not they also smoke cigars or pipe tobacco.

Consumption of alcoholic beverages—Information on consumption of alcoholic beverages was obtained during the dietary interview at the time of examination. Nondrinkers were defined as those who responded as not having had a drink of beer, wine, or liquor within 3 months of the time of exam. All other respondents were classified as drinkers.

Pica and lead poisoning tests—Data on eating unusual substances and on being tested for lead poisoning were obtained during the household interview. The questions responded to are shown in figure IV.

19a. Some children eat unusual substances. Does -- eat clay, starch, paint, plaster, dirt, or any material that might be considered unusual?	(146)	1 ☐ Yes 2 ☐ No (20)	
b. Is it –		**Yes**	**No**
Clay?	(147)	1 ☐	2 ☐
Starch?	(148)	1 ☐	2 ☐
Paint or plaster?	(149)	1 ☐	2 ☐
Dirt?	(150)	1 ☐	2 ☐
Any other material? – Specify _____	(151)	1 ☐	2 ☐

26a. Has -- ever been tested for lead poisoning?	(217)	1 ☐ Yes 2 ☐ No 9 ☐ DK } (27)
b. How long ago was -- tested?	(218)	_____ Years
	(219)	_____ Months 0 ☐ Less than one month
c. Did the results indicate that he had lead poisoning or high lead?	(220)	1 ☐ Yes 2 ☐ No (27)
d. Has -- ever been treated for lead poisoning?	(221)	1 ☐ Yes 2 ☐ No (27)
e. How long ago was -- treated?	(222)	_____ Years
	(223)	_____ Months 0 ☐ Less than one month

Figure IV. Questions asked to obtain data on eating unusual substances and on testing for lead poisoning: National Health and Nutrition Examination Survey, 1976–80.

Appendix IV
Statistical analysis of the chronological trend in the NHANES II blood lead levels

Linear regression analysis of the time trend

The computer program SURREGR,[29] which accounts for the complex survey design, was used for regression analyses. The blood lead values were weighted to represent population estimates, and those weights were included in the regression calculations. Regression analysis of NHANES II blood lead levels on time and the demographic covariates were performed for all races and in the selected population subgroups defined by race, sex, and age.

A plot of the mean blood lead levels of all races versus time (figure 8) suggested that a piecewise (segmental) linear regression model[78] would be appropriate. Mean blood lead values were computed as national estimates from data obtained on 9,933 examinees with blood lead measurements from specimens drawn by venipuncture over the 4-year period of NHANES II. The time variable was defined as the number of days from the beginning of the survey (February 20, 1976) to the date of sample collection divided by 28 (28 days was arbitrarily chosen).

Regression model

The regression model was constructed to fit a line from time = 0 through time = 34 and a second line from time = 34 to the end of the survey. The time value of 34 (that is, the 34th 28-day period) was chosen from visual inspection. The statistical formulation of the piecewise regression model was as follows:

$$Y_i = \beta_0 + \beta_1 X_1 + \beta_2 (X_1 - 34) X_2 + \ldots + \beta_n (X_n) + \varepsilon_i \quad (1)$$

where Y_i = natural logarithm (ln) of an examinee's blood lead value

X_1 = number of days from February 20, 1976, to sample collection date divided by 28

X_2 = indicator variable: $X_2 = 0$ if $X_1 \leq 34$ and $X_2 = 1$ if $X_1 > 34$

X_n = indicator variable for the n^{th} demographic covariate

When $X_1 \leq 34$ (those examined from February 1976–September 1978), the expected value of Y given equation (1) reduces to

$$E(Y) = \beta_0 + \beta_1 X_1 + \ldots + \beta_n (\overline{X}_n) \quad (2)$$

When $X_1 > 34$ (those examined from October 1978–February 1980), the expected value of Y given equation (1) reduces to

$$E(Y) = (\beta_0 - 34\beta_2) + (\beta_1 + \beta_2) X_1 + \ldots + \beta_n (\overline{X}_n) \quad (3)$$

In the regression analyses based on equation (1), the coding for the time variables was as follows:

$$\text{ORDER} = X_1$$

$$\text{ORDER1} = (X_1 - 34) X_2$$

The coding of the demographic covariates was as follows:

- Race:
  RACE1 = 1 Blacks
  RACE1 = 0 All other races
- Sex:
  SEX1 = 1 Male
  SEX1 = 0 Female
- Age:
  CHILD = 1 TEEN = 0 6 months–5 years
  CHILD = 0 TEEN = 1 6 years–17 years
  CHILD = 0 TEEN = 0 18 years–74 years
- Income:
  INC1 = 1 Annual family income less than $10,000
  INC1 = 0 Annual family income greater than or equal or $10,000
- Urbanization:
  RURAL = 1 Area of less than 1 million population
  RURAL = 0 Area of greater than or equal to 1 million population
- Season:
  WINTER = 1 Winter or spring
  WINTER = 0 Summer or fall
- Region of the country:
  SOUTH = 1 South and West
  SOUTH = 0 Northeast and Midwest

Two-variable interaction terms were selected for the model by prescreening all the possible two-variable interaction terms using ordinary least-squares stepwise regression (SAS, MAXR

NOTE: A list of references follows the text.

option).[79] Stepwise regression using backward elimination was also performed retaining variables that remained significant at the 0.10 level. Both approaches indicated that the following interaction terms were significant at the 0.10 level:

- AS1: CHILD × SEX1
- AS2: TEEN × SEX1
- AR1: CHILD × RACE1
- AI1: CHILD × INC1
- AI2: TEEN × INC1
- RU1: RACE1 × RURAL
- UN1: RURAL × SOUTH
- RW1: RACE1 × WINTER

The main effects, plus these interaction terms, were referred to as the demographic covariates.

The ordinary least-squares prescreening procedure gave a starting set of variables for regression analysis with SURREGR.[29] When the starting set of variables were examined using SURREGR, the significance of some of the variables decreased, because the variance estimates were generally higher after accounting for the sample design. Therefore, a "manual" backward elimination procedure was performed by running SURREGR multiple times, dropping the least significant variable from the model on each run until all variables were significant at the 0.05 level. In the process, ORDER and ORDER1 variables and any main effect demographic variables associated with a significant two-variable interaction term were kept in the model. The "manual" backward elimination procedure was applied separately to all races together, blacks, whites, white males, white females, whites 6 months–5 years, whites 6–17 years, and whites 18–74 years. Blacks were not analyzed by sex and age subgroups due to inadequate sample sizes.

Results from the time trend analysis

The results of the regression analyses are summarized by population group in table XIII. For each population group, the model was highly significant and the decrease in blood lead levels as measured by ORDER and ORDER1 was also highly significant.

The magnitude of the decrease in blood lead levels from the beginning to the end of the 4-year survey period was estimated in the following manner. First, the average value of each indicator variable (over the respective population group) was multiplied by its regression coefficient and then added to the intercept to form an adjusted intercept. The resulting equation then consisted of an adjusted intercept, ORDER and ORDER1. This equation was evaluated at values of ORDER and ORDER1 that represented the start and end of the survey as natural logarithms. The antilogs of these lead values are presented in table XIV along with a calculated percent drop over the survey period. The numbers given in the last column of table XIV were used to graph the bars in figure 9.

NOTE: A list of references follows the text.

The results shown in table XIV indicate that after accounting for the demographic covariates with indicator variables, a downward trend in blood lead values is still present and ranges from 31 to 42 percent.

Linear regression analysis of NHANES II blood lead levels on a gasoline lead variable

The regression analysis using the national estimates of lead used in gasoline production was conducted in the same manner as the time trend analysis, except GASQ (the U.S. Environmental Protection Agency quarterly national gasoline estimate)[80] replaced ORDER and ORDER1 in the model. Preliminary stepwise regression analysis indicated that GASQ was preferred over the quadratic term GASQ-squared. Separate regression analyses were performed for all races, blacks, whites, white males, white females, whites 6 months–5 years, whites 6–17 years, and whites 18–74 years. As previously noted, blacks were not analyzed by sex and age subgroups because of inadequate sample sizes.

The results of the regression analyses are summarized by population group in table XV. For each population group, the coefficient for the gasoline lead variable (GASQ) was highly significant.

The amount of lead used in gasoline production decreased by more than 50 percent from the beginning to the end of the survey. The magnitude of the change in blood lead levels that can be accounted for by the gasoline lead variable can be calculated in the same manner as was done for ORDER and ORDER1. An adjusted intercept is determined and the model equation is then evaluated at the gasoline lead values present at the start and end of the survey. The results of these calculations are presented in table XVI. The entire analysis was repeated using 6 months' national gasoline estimates with very similar results.

Correlation analysis between blood lead level and lead used in gasoline production

To further evaluate the relationship of lead in gasoline production to NHANES II blood lead levels, the following correlation analysis was done. First, a regression model consisting only of the demographic covariates was fit to the natural logarithm of the blood lead data. Then, the mean of all the natural logarithm blood lead levels used in the regression was added to the residuals from the fit. This procedure gave blood lead levels adjusted for the effects of the demographic covariates. These adjusted natural logarithm blood lead levels were then averaged by 6-month periods. Pearson correlation coefficients were computed between these average natural logarithm blood lead levels by 6-month periods and the total lead used in gasoline per 6 months. This correlation varied slightly by whether the 6-month periods were chosen as January–June and July–December or April–September and October–March. Results for both selections and for an average of the two were shown in text table C for all races and for selected population subgroups defined by race, sex, and age.

Table XIII. Results of regression of the natural logarithm of the blood lead level on time and the demographic covariates: National Health and Nutrition Examination Survey, 1976–80

Race, sex, age, and variable	F statistic	Degrees of freedom	Probability	Coefficient
All races				
ORDER	5.78	1	0.0222	−0.00337
ORDER1	22.34	1	0.0000	−0.01563
CHILD	319.62	1	0.0000	0.3543
TEEN	0.26	1	0.6148	0.0123
RURAL	4.77	1	0.0364	−0.0511
INC1	1.41	1	0.2434	−0.0129
RACE1	12.13	1	0.0015	0.0948
SOUTH	10.77	1	0.0025	−0.0701
AS1	290.58	1	0.0000	−0.3336
SEX1	1,585.72	1	0.0000	0.3656
AS2	77.92	1	0.0000	−0.1805
AR1	19.69	1	0.0001	0.1110
AI1	72.77	1	0.0000	−0.1611
AI2	10.35	1	0.0030	−0.0835
RU1	4.66	1	0.0385	0.0740
Intercept	...	...	...	2.5933
Overall	168.41	15	0.0000	...
Black				
ORDER	7.21	1	0.0114	−0.004519
ORDER1	2.73	1	0.1083	−0.007368
CHILD	129.26	1	0.0000	0.5216
TEEN	2.93	1	0.0965	0.0548
INC1	0.88	1	0.3555	−0.0298
WINTER	10.61	1	0.0027	−0.1141
AS1	42.69	1	0.0000	−0.4387
SEX1	132.84	1	0.0000	0.4176
AS2	27.65	1	0.0000	−0.2514
AI1	13.05	1	0.0010	−0.1314
Intercept	...	...	...	2.6924
Overall	40.41	10	0.0000	...
White				
ORDER	5.12	1	0.0306	−0.003467
ORDER1	21.37	1	0.0001	−0.01612
CHILD	230.72	1	0.0000	0.332204
TEEN	0.05	1	0.8224	0.006612
RURAL	5.25	1	0.0287	−0.05389
INC1	2.26	1	0.1424	−0.01652
SOUTH	12.36	1	0.0013	−0.07817
AS1	207.86	1	0.0000	−0.3215
SEX1	1,195.37	1	0.0000	0.3597
AI1	53.83	1	0.0000	−0.1503
AS2	51.46	1	0.0000	−0.1787
AI2	7.82	1	0.0087	−0.8685
Intercept	...	...	...	2.6092
Overall	136.93	12	0.0000	...
White, male				
ORDER	5.07	1	0.0314	−0.003379
ORDER1	19.16	1	0.0001	−0.01592
CHILD	2.04	1	0.1631	−0.03030
TEEN	22.48	1	0.0000	−0.1740
INC1	6.71	1	0.0143	−0.0481
SOUTH	13.06	1	0.0010	−0.0861
AI1	10.37	1	0.0029	−0.0939
AI2	3.06	1	0.0897	−0.0816
Intercept	...	...	...	2.9524
Overall	39.17	8	0.0000	...
White, female				
ORDER	4.61	1	0.0395	−0.003724
ORDER1	17.80	1	0.0002	−0.01655
CHILD	161.33	1	0.0000	0.36834
TEEN	0.08	1	0.7751	0.00761
RURAL	7.16	1	0.0117	−0.0729
INC1	0.48	1	0.4944	0.0106
SOUTH	10.25	1	0.0031	−0.0776
AI1	32.13	1	0.0000	−0.2055
AI2	6.49	1	0.0158	−0.0908
Intercept	...	...	...	2.6137
Overall	46.67	9	0.0000	...
White, 6 months–5 years old				
ORDER	8.82	1	0.0056	−0.005682
ORDER1	8.45	1	0.0066	−0.01325
INC1	73.76	1	0.0000	−0.1602
SOUTH	3.66	1	0.0646	−0.0670
SEX1	4.46	1	0.0426	0.0367
Intercept	...	...	...	2.9341
Overall	39.01	5	0.0000	...
White, 6–17 years old				
ORDER	2.43	1	0.1288	−0.002974
ORDER1	22.16	1	0.0000	−0.02028
SEX1	72.15	1	0.0000	−0.1797
INC1	7.89	1	0.0084	−0.0889
Intercept	...	...	...	2.5234
Overall	32.91	4	0.0000	...
White, 18–74 years old				
ORDER	4.29	1	0.0465	−0.003226
ORDER1	20.16	1	0.0001	−0.01584
SEX1	1,193.67	1	0.0000	0.3588
RURAL	5.12	1	0.0306	−0.0525
SOUTH	14.69	1	0.0006	−0.0882
Intercept	...	...	...	2.5963
Overall	295.14	5	0.0000	...

Table XIV. Percent decrease in blood lead levels over the survey period, after accounting for the effects of the demographic covariates, by selected group characteristics: United States, 1976–80

Group characteristic	*Beginning of the survey*	*End of the survey*	*Difference*	*Percent difference*
	Blood lead levels in micrograms per deciliter			
All races	14.59	9.17	5.42	37.1
Black	16.70	11.51	5.19	31.1
White	14.38	8.91	5.46	38.0
Male	16.73	10.46	6.27	37.5
Female	12.51	7.59	4.92	39.3
0.5–5 years	16.51	9.61	6.90	41.8
6–17 years	12.82	7.56	5.27	41.1
18–74 years	14.66	9.25	5.41	36.9

Table XV. Results of regression of the natural logarithm of the blood lead level on the gasoline lead variable and the demographic covariates: United States, 1976–80

Race, sex, age, and variable	F *statistic*	*Degrees of freedom*	*Probability*	*Coefficient*
All races				
GASQ	96.31	1	0.0000	1.5159
CHILD	287.93	1	0.0000	0.3597
TEEN	0.19	1	0.6657	0.0106
RURAL	20.20	1	0.0001	−0.1031
INC1	2.05	1	0.1618	−0.0162
RACE1	5.96	1	0.0203	0.0714
SOUTH	0.84	1	0.3663	0.0211
AS1	289.11	1	0.0000	−0.3354
SEX1	1,495.38	1	0.0000	0.3668
AS2	82.36	1	0.0000	−0.1750
AR1	24.69	1	0.0000	0.1174
AI1	65.95	1	0.0000	−0.1684
AI2	10.31	1	0.0030	−0.0844
RU1	5.69	1	0.0232	0.0771
Intercept	...	...	...	1.7999
Overall	174.53	14	0.0000	...
Black				
GASQ	42.28	1	0.0000	1.1121
CHILD	174.48	1	0.0000	0.5298
TEEN	2.56	1	0.1194	0.0505
INC1	2.93	1	0.0968	−0.0488
WINTER	6.32	1	0.0172	−0.0959
AS1	53.77	1	0.0000	−0.4544
SEX1	136.37	1	0.0000	0.4223
AS2	25.24	1	0.0000	−0.2391
AI1	11.91	1	0.0016	−0.1252
Intercept	...	...	...	2.0702
Overall	62.96	9	0.0000	...
White				
GASQ	94.50	1	0.0000	1.5944
CHILD	229.73	1	0.0000	0.3368
TEEN	0.02	1	0.8779	0.00453
RURAL	22.32	1	0.0000	−0.1082
INC1	2.79	1	0.1044	−0.0192
SOUTH	0.53	1	0.4713	0.0165
AS1	224.67	1	0.0000	−0.3247
SEX1	1,119.97	1	0.0000	0.3606
AS2	52.62	1	0.0000	−0.1740
AI1	57.11	1	0.0000	−0.1574
AI2	7.86	1	0.0085	−0.0875
Intercept	...	...	...	1.7761
Overall	135.18	11	0.0000	...
White, male				
GASQ	92.08	1	0.0000	1.5675
CHILD	1.86	1	0.1818	−0.0288
TEEN	19.25	1	0.0001	−0.1676
INC1	5.82	1	0.0218	−0.0472
SOUTH	0.02	1	0.8997	−0.00307
AI1	11.21	1	0.0021	−0.1039
AI2	3.16	1	0.0848	−0.0872
Intercept	...	...	...	2.0981
Overall	34.18	7	0.0000	...
White, female				
GASQ	81.12	1	0.0000	1.6721
CHILD	169.76	1	0.0000	0.3735
TEEN	0.02	1	0.8888	0.00374
RURAL	26.51	1	0.0000	−0.1337
INC1	0.18	1	0.6724	0.00643
SOUTH	0.90	1	0.3509	0.0237
AI1	38.90	1	0.0000	−0.2131
AI2	6.67	1	0.0146	−0.0887
Intercept	...	...	...	1.7415
Overall	38.93	8	0.0000	...
White, 6 months–5 years old				
GASQ	81.13	1	0.0000	1.7876
INC1	76.98	1	0.0000	−0.1711
SOUTH	1.05	1	0.3121	0.0314
SEX1	3.62	1	0.0661	0.0334
Intercept	...	...	...	1.9330
Overall	47.34	4	0.0000	...
White, 6–17 years old				
GASQ	38.75	1	0.0000	1.6086
SEX1	89.82	1	0.0000	0.1902
INC1	10.27	1	0.0031	−0.1041
Intercept	...	...	...	1.6978
Overall	39.35	3	0.0000	...
White, 18–74 years old				
GASQ	86.33	1	0.0000	1.5454
SEX1	1,108.31	1	0.0000	0.3602
RURAL	23.39	1	0.0000	−0.1037
SOUTH	0.03	1	0.8563	0.00465
Intercept	...	...	...	1.7880
Overall	290.39	4	0.0000	...

Table XVI. Percent decrease in blood lead levels over the survey period that can be explained by a decline in the use of lead in gasoline, after accounting for the effects of the demographic covariates by selected group characteristics: United States, 1976–80

Group characteristic	*Beginning of the survey*	*End of the survey*	*Difference*	*Percent difference*
	Blood lead levels in micrograms per deciliter			
All races	14.82	9.48	5.35	36.1
Black	16.24	11.70	4.54	28.0
White	14.63	9.14	5.49	37.5
Male	17.04	10.73	6.31	37.0
Female	12.71	7.76	4.95	38.9
0.5–5 years	16.40	9.68	6.72	41.0
6–17 years	13.08	8.14	4.94	37.8
18–74 years	14.95	9.48	5.47	36.6

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