SUMMARY OF ANALYSES OF THE NHANES II

BLOOD LEAD DATA

Ethyl Corporation
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SUMMARY

Since the peak year of 1970, gasoline lead usage has declined 75% by 1981. The Second National Health and Nutrition Examination Survey (NHANES II) carried out over the 4-year period of 1976-80 found that overall mean blood-lead levels decreased from 15.8 μg/dl to 10.0 μg/dl. Dr. Houk (CDC) and EPA have either alleged or implied that this decrease is primarily due to the reduction in gasoline lead usage. However, the NHANES II program was not designed to show trends within the 4-year period and Houk and EPA could equally well have concluded that the reduced blood lead was due to the decrease in percent of children less than 6 years old and percent of non-rural residents in the latter part of the survey.

Ethyl's in-depth statistical analysis of the NHANES II data has used a regression model to separate the contributions to blood lead into factors related to personal attributes and those related to the person's environment. This analysis showed that other factors varying with time were the major causes of the 1976 to 1980 decrease in blood lead and not gasoline lead usage.

The validity of Ethyl's statistical analysis has been confirmed by two outstanding statistical experts--Dr. Ralph Bradley of the University of Georgia and Dr. Norman R. Draper of the University of Wisconsin.

There are eight other cited studies in this report that also indicate that the public's blood-lead concentrations are not materially affected by the level of gasoline lead usage.

Acceptance of the incorrect alleged cause-effect relationship between blood-lead levels and gasoline lead usage ignores the beneficial impact of the nationwide blood-lead screening programs, the lead-paint control and educational programs, and the lead-content control of processed food and beverages in reducing blood-lead levels in recent years.

INTRODUCTION

There have been two main factors reducing gasoline lead usage in the United States since 1970, the peak usage year. One has been the increased demand for unleaded gasoline. The catalyst-equipped car, requiring unleaded gasoline, was introduced in the U.S. in the mid-1970's. Today, because of very restrictive car gaseous emission standards, all new cars are catalyst equipped. Thus, there is an ever-increasing percentage of unleaded gasoline demand, with unleaded gasoline claiming about 54% of total gasoline demand in 1982. The other factor has been the lead phase-down regulations initiated by the Environmental Protection Agency.

These two factors, combined with smaller cars with reduced fuel consumption and higher gasoline prices, resulted by 1981 in a 75 percent reduction in gasoline lead usage since 1970.

It has been claimed by at least two investigators that this large reduction in gasoline lead usage has been primarily responsible for the decided reduction in blood-lead concentrations of population groups observed in several studies. This paper analyzes the data on which these allegations are based and how the results relate to other studies, and concludes that the effect of gasoline lead usage on the public's blood-lead concentration is minimal and of no health-related significance.

NHANES II BLOOD-LEAD DATA

The Second National Health and Nutrition Examination Survey (NHANES II) [1] was carried out by the U.S. National Center for Health Statistics over the 4-year period of 1976-80. This survey was designed to obtain health and nutrition data of a sampled population representing the U.S. population ranging in age from 6 months to 74 years.

Reliable venipuncture blood-lead concentration data were obtained for 9,933 persons in 64 sampling areas, including preschool children ages 6 months - 5 years, youths ages 6-17 years, and adults ages 18-74 years[2].

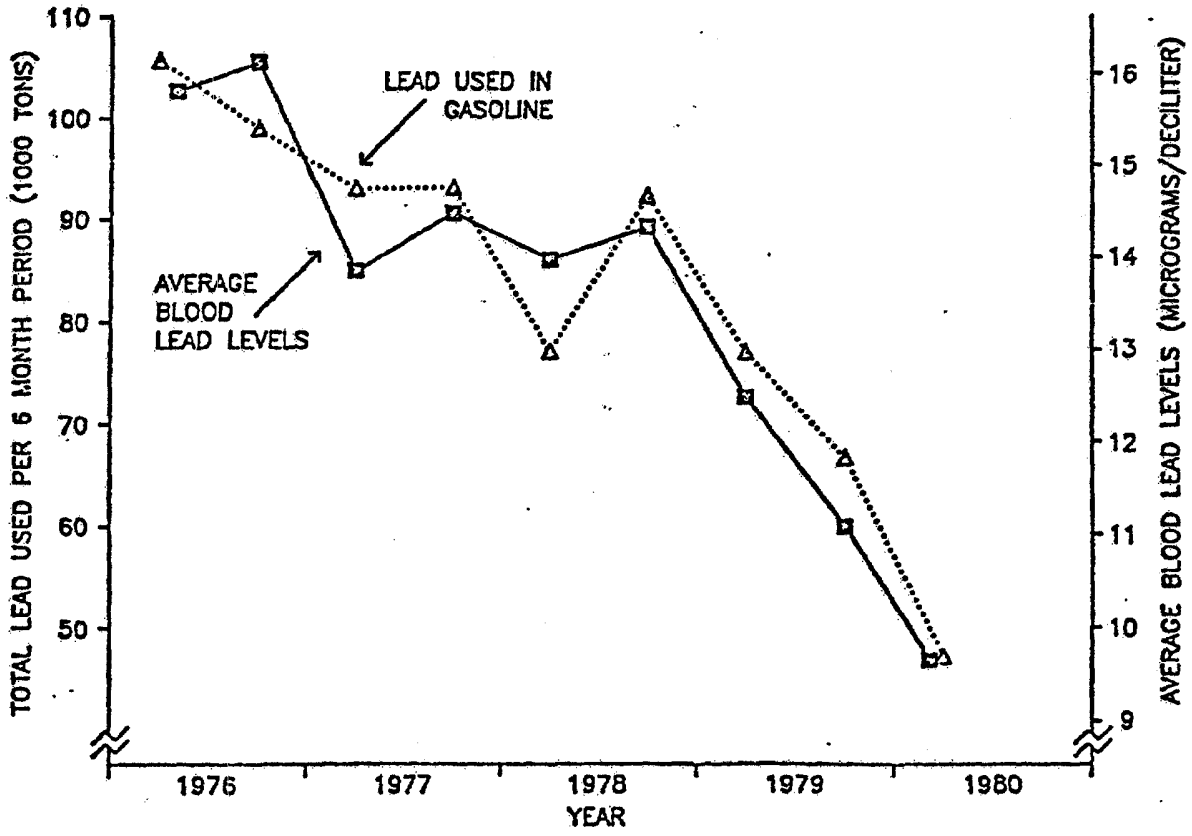
All age groups included both sexes, whites and blacks, urban and rural groups, and several income levels. During the 4-year period, the overall mean blood-lead levels decreased about 37%, from 15.8 μg/dl to 10.0 μg/dl, with approximately the same percentage decrease for the different groups. However, it should be emphasized that there was no repetitive sampling of areas or subjects. Thus, the survey was not designed to establish blood-lead trends during the 4-year period.

A brief partial summary of the blood-lead data was first issued in March 1982[3] and included a chart showing the similarity of the decrease in average blood-lead levels and gasoline lead usage with time. This summary stated that, "The decrease in mean blood-lead levels reflects the decrease in lead used in gasoline production." In April 1982, Dr. Houk presented a written statement at two hearings[4] in Washington, D.C., which included a graph (Figure 1). In referring to the reduction in mean blood lead, Dr. Houk stated, "It was not due to chance, laboratory error, nor sampling of age, sex, race, urban vs rural areas, income levels, or geographic regions." On April 24, 1982, Dr. Houk appeared on national TV[5] and showed the Figure 1 graph. In referring to the lead-usage curve and the blood-lead curve, he stated, "One never sees, or seldom sees such a nice, tight correlation as cause and effect as we see on this chart." The apparent "nice, tight correlation" is based on the fact that both blood leads and gasoline lead usage decreased during this time period. However, this does not necessarily mean that there is a relationship between blood lead and gasoline lead usage. In addition, Dr. Houk adjusted the scales of the graph to make the slopes of the two curves as nearly identical as possible. It will subsequently be shown that these statements by Dr. Houk at the hearings and on TV are not completely true and that the cause-effect relationship simply does not exist.

The Environmental Protection Agency readily accepted the implied cause-effect relationship between decreasing blood-lead levels and gasoline lead usage with time despite the criticisms of their interpretation by several groups, including Ethyl, at the Lead Hearings in Washington, D.C., on April 15 and 16, 1982. Subsequently, Ethyl obtained the tapes of the in-

Figure 1

LEAD USED IN GASOLINE PRODUCTION AND AVERAGE NHANES II BLOOD LEAD LEVELS (FEB. 1976 – FEB. 1980)



dividual blood-lead data with related information and proceeded to make an in-depth analysis. First of all, there was a sampling bias to fewer children and fewer non-rural residents during the latter period of the survey, as shown in Figures 2 and 3. Both of these effects could affect the trend toward lower blood-lead levels. Linear regressions of these parameters with time showed that the correlation coefficients with time of three of these parameters--blood lead (0.92), gasoline lead usage (0.91), and percent children less than 6 years old (0.90)--were essentially equal, with the correlation coefficient for percent non-rural residents being 0.70. Thus, Houk could have ignored gasoline lead usage and equally well concluded that the decrease in blood lead with time was due to the decrease in percent children in the sample less than 6 years old. Furthermore, the 33% reduction in blood-lead levels from 1976 to 1980 in the rural population was almost twice the 19% reduction in the urbanized population of greater than 1,000,000. The reverse should be true if the decline in auto-lead emissions was a major factor in the blood-lead reductions.

In-Depth Statistical Analysis of NHANES II

Thus, the preliminary analysis showed that sex, race, age, family income, and general location (urban vs non-urban) were significant independent variables affecting blood-lead concentration. Since there was a need to reduce or eliminate the confounding of these variables, Ethyl designed a regression model to separate the contributions to blood lead into those factors relating to personal attributes and those factors relating to the environment in which the person lived.

The model assumes that the blood-lead content of an individual can be described as a sum of two contributing parts. These two contributing parts are distinctively different in character. The first contribution to blood lead is descriptive of the individual (e.g., age, sex, race, and family income). The second contribution is descriptive of the environment in which the individual lives. A basic premise is that a specific individual's blood-lead content will respond to outside influence in the same manner wherever

Figure 2

NHANES II Data
% Children <6 Years Old and Blood-Lead Levels

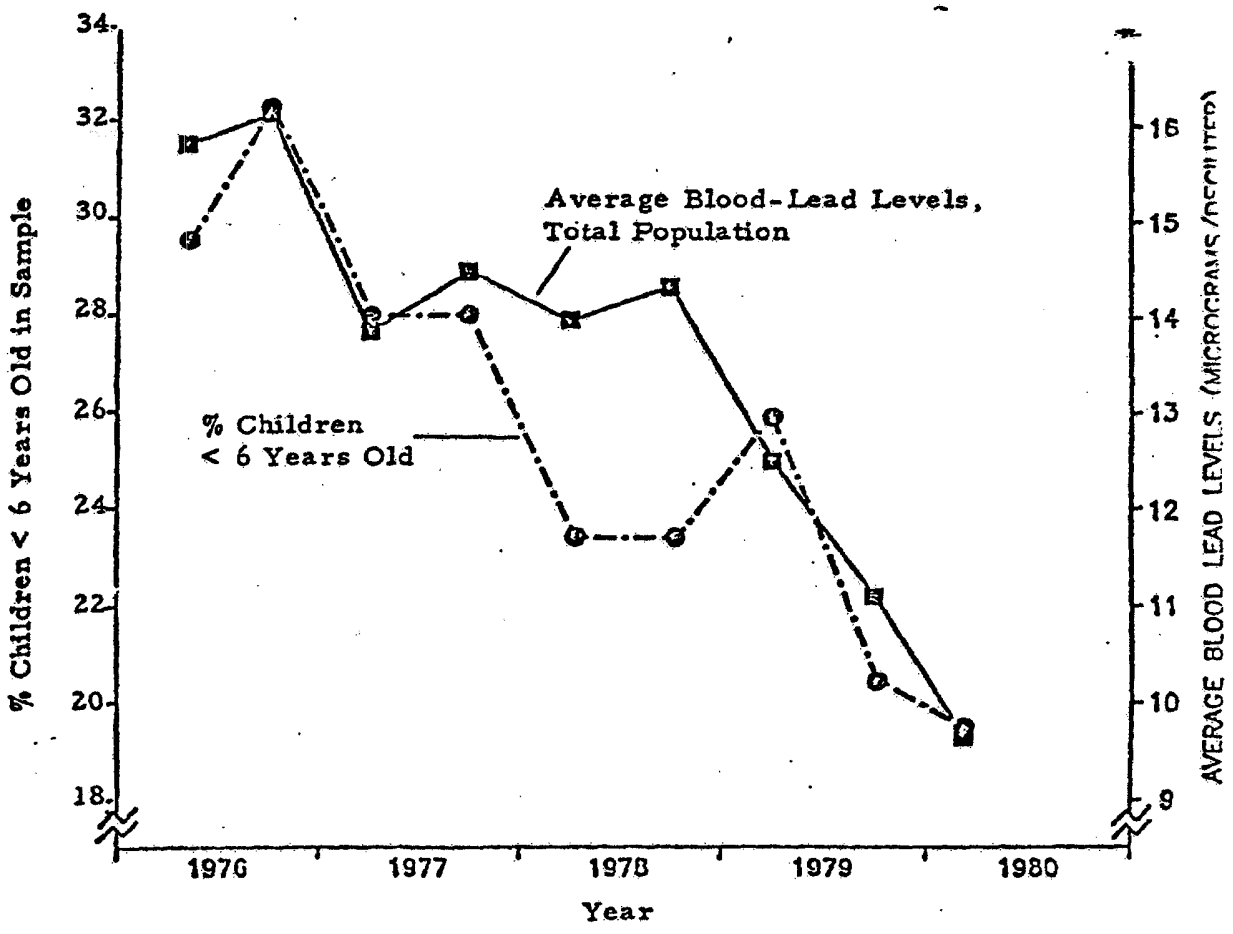
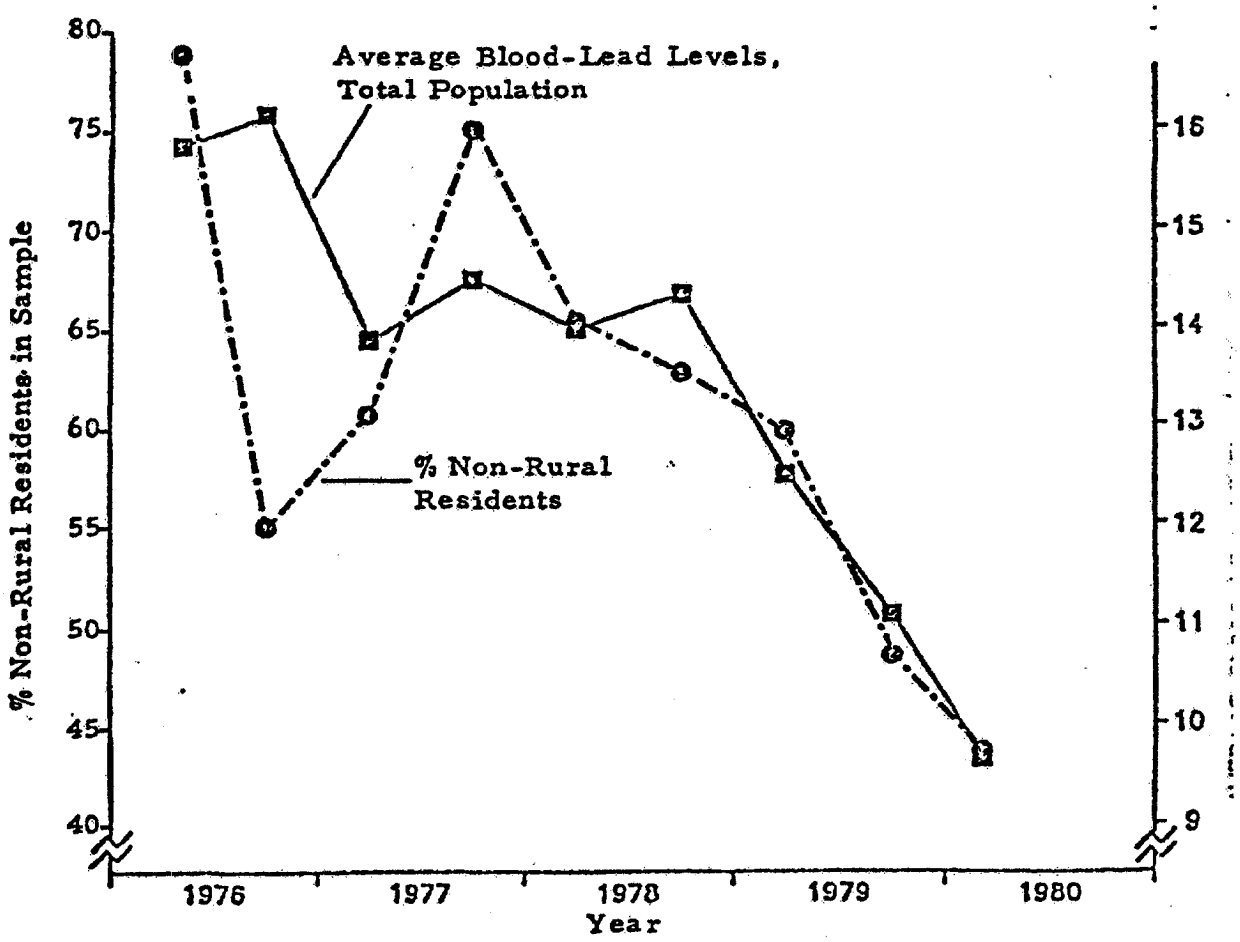


Figure 3

NHANES II Data
% Non-Rural Residents and Blood-Lead Levels



he lives. The extension of this premise is that, to explain the variations of blood-lead content, one must describe the environment in which the individual exists. Heretofore, the confounding effects between the personal descriptors and the environment descriptors have clouded any attempts to understand the cause-effect relationships for the individual. By discrete-type regression techniques, these two contribution types have been separated for the NHANES II data so that the personal descriptors and environment descriptors are essentially independent of each other. The personal descriptors are not subject to further analysis, but the environment descriptors can be analyzed and/or correlated with any desired quantities related to outside factors that might affect the blood-lead content of an individual living therein.

The analysis was constructed by grouping the individuals into 3 age groups (less than 6, 6-18, and 19 and over), 2 racial groups (white and black), sex (male and female), and 3 annual family income levels (less than $6,000, $6,000-$15,000, and over $15,000). These groupings and all combinations thereof were assigned numbers from 1 to 36, called personal identifiers. The 64 sampling locations were assigned numbers from 1 to 64. The complete NHANES II data were then regressed using 99 variables (the 36 personal identifiers plus the 64 locations less one combination representing a base). From this, we obtained an average blood-lead contribution for each location with variations of age, sex, race, and income removed. The regression partitioned the lead response into two parts. The first partition represents an averaged mean blood-lead contribution for each personal identifier that is independent of the location effects. The second partition represents an averaged mean blood-lead contribution for each location that is independent of the personal identifier effects. This separation of effects permits the study of the averaged means for each location in terms of the unique environmental factors of each location that might influence the blood-lead content of any person residing in that locale.

Dr. Houk's chart implies a relationship between blood lead and national gasoline lead usage. This is not a meaningful variable for exposure of an individual because the blood-lead concentration of an individual responds to the local lead exposure conditions rather than to a national average value. For example, exposure conditions are very different between the central city and rural areas. The Ethyl analysis correlates the averaged means for each location with selected indepependent variables to attempt to represent the environmental lead exposure factors. These independent variables are:

Population Density - Blood leads are known to respond to the air-lead concentration by respiration of airborne lead particles, some of which are retained in the lungs with lead absorption into the blood. Air-lead concentration data at the time of blood-lead sampling are not available for most NHANES II locations. Since gasoline lead usage would be expected to be highly correlated with the population, population density can be used as an estimate of the relative gasoline usage per unit area and thus the relative air-lead exposure. The population of each location was estimated for the average date of sampling by linear interpolation between 1970 and 1980 census populations. This population was then divided by the location area in square miles to obtain a population density.

Gasoline Lead Usage - Lead fallout from the atmosphere has been speculated to contribute to blood-lead concentration through the food chain. One way of estimating a lead fallout contribution is to consider gasoline lead usage for a location divided by the square miles of area of the location. Annual gasoline lead data are available for each state. Thus, the annual gasoline lead usage for a state in the year of blood-lead sampling divided by the area of the state provides a variable to represent lead fallout entering the food chain.

Time - Blood-lead concentrations have generally decreased with time because of reduction of lead in food packaging, paint, and other potential diet sources. Thus, time is a potential independent variable to represent lead from all sources, rather than gasoline lead alone, as contributors to blood lead. The time used in this analysis is the weighted average of all of the sampling dates for a given location, with this date translated to the number of days after January 1, 1976.

A linear regression of the external contribution to blood-lead concentration (dependent variable) for each location was made with the independent variables of time, natural logarithm of the population density, and lead usage for each location. The natural logarithm of the population density was used because these values covered over a three order-of-magnitude range. Linear representation of population density would have placed excessive weighting on the high numbers.

A conclusion from the regression analysis is that both time and population density are significant variables and gasoline lead usage is an insignificant variable. The time variable is the major contributor to changes in blood lead while population density is a minor contributor. Thus, this analysis shows that, when all factors are considered, the fallout aspect of gasoline lead usage does not significantly affect blood-lead concentration and that population density representing air-lead is a significant but small contributor to blood-lead concentration. Houk incorrectly attributes blood lead to gasoline lead usage because other contributing factors are not properly considered (e.g., personal factors and other lead sources).

Using this model, blood-lead concentrations were calculated for three blood-lead concentration groups--maximum, median, and low--in areas of low population density (10 persons/sq. mile) and high population density (3000 persons/sq. mile). These data (Table 1) can be used to estimate two responses:

1. The difference in blood-lead concentration between the high and low population densities.
2. The difference in blood-lead concentration from the beginning to the end of the survey period.

TABLE 1

Regression Model Predictions of Blood-Lead Concentration

Year	Level of Personal Contribution to Blood Lead*	Blood-Lead Conc. (y'), µg/dl @ Population Density (P), persons/sq. mile		Δ
		10	3000	
1976	Max.	23.9	26.7	2.8
	Median	18.2	20.4	2.2
	Min.	10.8	12.1	1.3
	Average	18.7		2.1
1980	Max.	17.2	19.3	2.1
	Median	13.2	14.8	1.6
	Min.	7.8	8.7	0.9
	Average	13.5		1.5

*Personal Descriptors

	Age Group	Race	Sex	Family Income
Max.	Young	Black	Male	Low
Median	Old	White	Male	Low
Min.	Middle	White	Female	High

For 1976, the differences in blood leads between the high and low population densities range from 1.3 to 2.8 µg/dl, with an average of 2.1. For 1980, the differences in blood leads between the high and low population densities range from 0.9 to 2.1 µg/dl, with an average of 1.5. These differences

include the effect of factors that are essentially proportional to population, such as gasoline lead, industrial lead emissions, and lead in paint.

The average blood leads of the two population density groups are 18.7 μg/dl in 1976 and 13.5 μg/dl in 1980. This decrease of 5.2 μg/dl represents the decrease in contribution to blood lead from all sources, including gasoline, paint, solder in food containers, industrial emissions, etc. The sampling bias due to personal factors has been excluded from this figure of 5.2 μg/dl. In 1976, the high-population density group had a 2.1 μg/dl higher average blood lead than the low-density group. This difference between these two density groups had decreased to 1.5 μg/dl in 1980. If we assume that gasoline lead has the same percent contribution to blood lead relative to other sources in 1980 as in 1976, the 0.6 μg/dl decrease from 2.1 to 1.5 is due to population-related factors alone. Therefore, it must be the maximum possible reduction in blood lead due to the air-lead aspect of gasoline lead usage. We conclude that the 5.2 μg/dl decrease in blood lead from 1976 to 1980 largely results from factors other than the reduction in gasoline lead usage.

Air-lead concentration data are available from the National Air Sampling Network for 24 of the NHANES II locations for the quarter in which the blood-lead sampling occurred. Air-lead concentrations ranged from 0.14 to 1.84 $\mu g/m^3$ and cover the four years of the NHANES II study. Correlation of the averaged mean blood-lead contribution for each location with time, air-lead concentration, and population density shows that the air-lead concentration and population density are redundant. The correlation of blood lead with the combination of time and air-lead concentration shows a slightly higher correlation coefficient than the correlations with (1) time and population density and (2) time, air-lead concentration, and population density. This confirms our selection of the population density as an estimate of the response of the air-lead concentration. The correlation of blood lead with time and air-lead concentration shows an increase in blood-lead concentration of 1.35 μg/dl for one $\mu g/m^3$ increase in air-lead concentration. This value is consistent with many observations of this response relationship and is

consistent with the conclusion that air lead was a minor contributor to the decrease in blood-lead concentration during the NHANES II study.

In summary, our analysis of the NHANES II data has shown that time is the major contributor to differences in blood lead between 1976 and 1980. That portion of gasoline lead usage representing potential fallout into the food chain was found to be insignificant, and that portion representing air lead had a maximum possible effect of reducing blood lead by 0.6 μg/dl during the 4-year period. The major contribution of time to the decrease in blood lead indicates that other factors that vary with time are the major causes of the 1976 to 1980 decrease in blood lead and not gasoline lead usage. For example, the use of lead solder in cans and shipping containers for food and beverages has dropped from 16,401 short tons in 1976 to 9,541 short tons in 1981. (A detailed statistical analysis will be supplied on request.)

Acceptance of the incorrect alleged cause-effect relationship between blood-lead levels and gasoline lead usage ignores the beneficial impact of the nationwide blood-lead screening programs, the lead-paint control and educational programs, and the lead-content control of processed food and beverages in reducing blood-lead levels in recent years.

Confirmation of Ethyl's Statistical Analysis

Dr. Ralph Bradley (an eminent statistician, past president of the American Statistical Association, and now with the University of Georgia) has reviewed Dr. Houk's chart and Ethyl's in-depth statistical analysis of the NHANES II data. He confirms the error in the assumption of a cause-effect relationship between blood-lead concentrations and gasoline lead usage. He also confirms that Ethyl's analysis represents correct statistical applications[6].

Dr. Norman R. Draper, Professor of Statistics, University of Wisconsin, also has reviewed Ethyl's analysis and finds that it provides a reasonable approximate method of examining the effect of the remaining prime variables (sample time, population density, and gasoline lead usage) on the individual blood leads[7].

OTHER STUDIES

Other studies show that level of gasoline lead usage does not materially affect the public's blood-lead concentration. These studies include:

- The Frankfurt (West Germany) study[8] showed only a 10% (1-2 g/dl) decrease in average blood lead when gasoline lead was reduced by 63%, from 0.40 g/L to 0.15 g/L. The study was specifically designed to show the impact of reduced lead-in-gasoline usage on blood-lead concentrations. Subjects surveyed included residents of central Frankfurt, office workers, street cleaners, policemen, and taxi drivers.

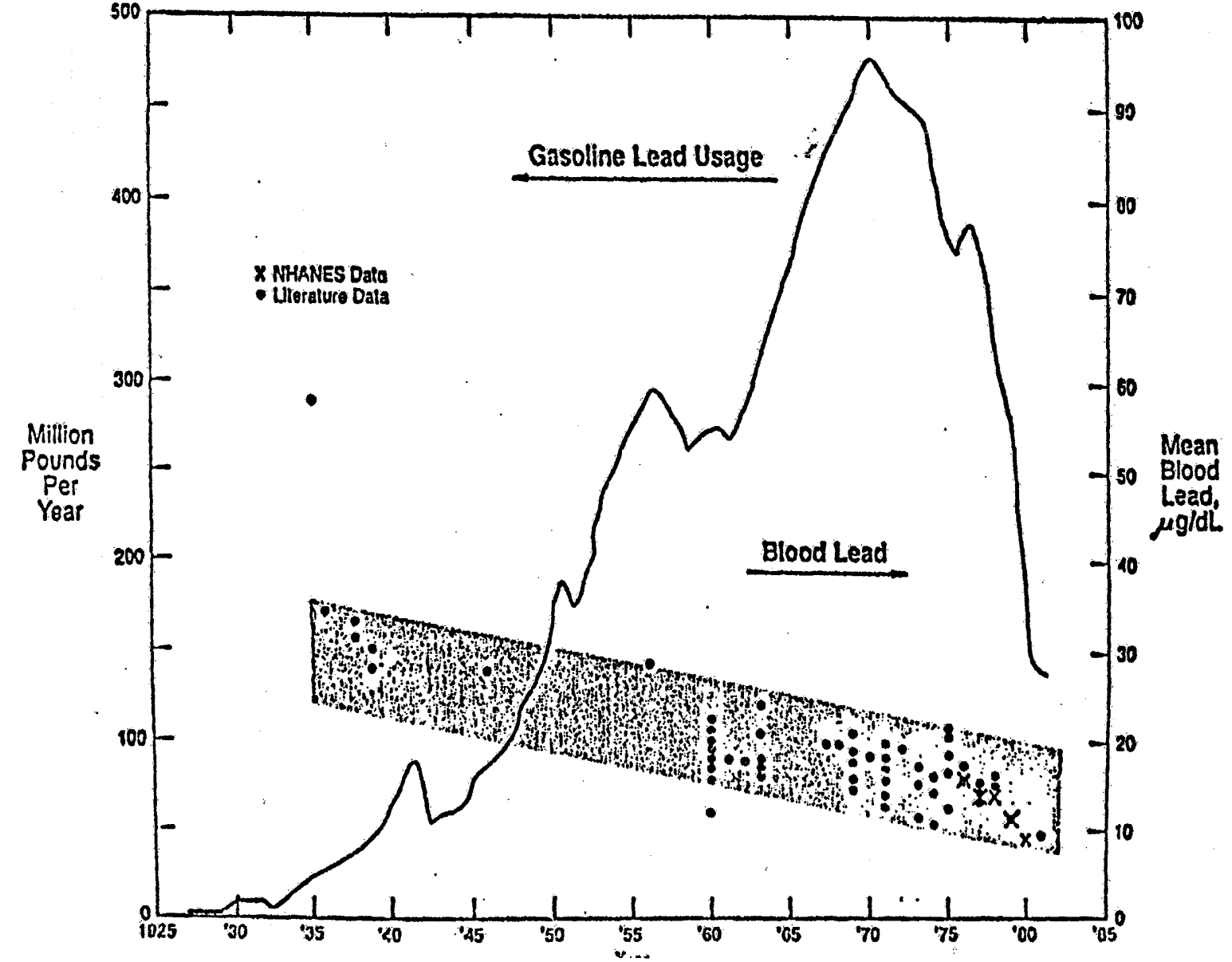
- Oxley's study in Great Britain[9] showed a reduction in blood lead of the general public for the 1978-80 period as compared to the 1967-69 period while gasoline lead usage remained essentially constant.

- Surveys in Wales[10] showed that, "Blood lead levels, estimated in carefully designed and conducted epidemiological surveys in Wales, have shown a fall of over 30% since 1972. This is despite no change in lead in petrol but a steady increase in general traffic flows during the same period, and, apart from a very few small exceptions, no change in water lead. Changes in lead in food seem likely to be largely responsible."

- Ethyl's literature survey of historical blood-lead data[11] showed no relationship between gasoline lead usage and blood-lead values for population groups despite enormous changes in gasoline lead usage over the time period 1935-1981 (Figure 4). Population groups subjected to occupational lead exposure were not included.

Figure 4

U. S. Historical Data



- Data from the U.S. Centers for Disease Control (CDC) blood-lead screening program in Los Angeles County, California[12], showed that the percentage of children with blood-lead levels in excess of 30 μg/dl dropped sharply in early 1977 and, since it remained below 1% after 1979, the program was discontinued in mid-1981. The sharp drop starting in 1977 was correlated with the CDC Lead Poisoning Prevention Program which removed the lead paint hazard from a large number of homes[12]. There was no correlation of percent children with blood leads > 30 μg/dl with gasoline lead usage in California[13], as shown in Figure 5.

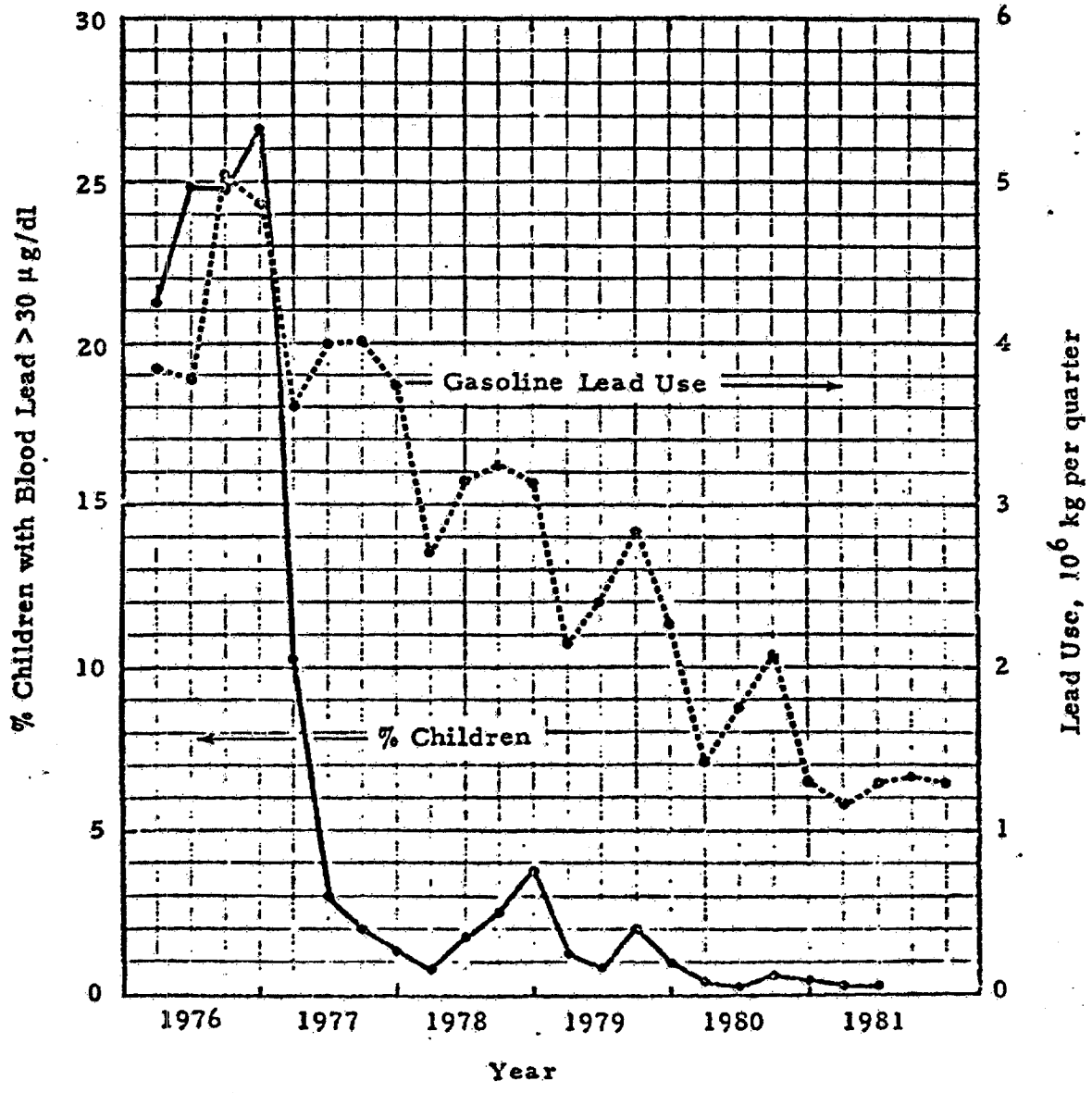
- Dr. Rabinowitz of Harvard Medical School recently presented an analysis of environmental, demographic, and medical factors related to umbilical cord blood-lead levels of 11,387 babies[14]. Univariate analysis of environmental samples revealed that tap water, indoor air lead, amount of dust, and the size of the nearest road were unrelated to cord blood lead. The presence of lead in paint and soil, and refinishing activities were significantly related.

- Studies in California of children living and attending school near and away from heavily traveled freeways[15] showed that neither proximity of school to traffic nor proximity of residence to traffic had an appreciable effect on blood-lead concentration.

- Using the isotopic ratio technique, investigators in California found that the moderately elevated blood leads of the children studied were due to leaded-paint sources rather than vehicle lead emissions[16].

Figure 5

Los Angeles County Children with Blood Lead > 30 μg/dl (CDC Program) and California Gasoline Lead Use



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