

Relation of Pediatric Blood Lead Levels to Lead in Gasoline

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Analysis of a large data set of pediatric blood lead levels collected in New York City (1970-1976) shows a highly significant association between geometric mean blood lead levels and the amount of lead present in gasoline sold during the same period. This association was observed for all age and ethnic groups studied, and it suggests that possible exposure pathways other than ambient air should be considered. Even without detailed knowledge of the exact exposure pathways, sufficient information now exists for policy analysis and decisions relevant to controls and standards related to lead in gasoline and its effect on subsets of the population.

Blood lead levels provide a measure of human lead exposure resulting from all environmental sources. The quantification of relations between environmental exposure and blood lead levels, especially in children, is of considerable importance for assessing the relative contributions of various sources of environmental lead and for judging the adequacy of control measures (1-3).

The present report is part of a research program exploring several large data sets of pediatric blood lead levels collected by local lead poisoning screening programs. We have previously reported (4) on the analysis of venous blood lead levels of 178,533 New York City children screened for the first time during 1970-1976. This investigation revealed a significant relationship between geometric mean (GM) blood lead levels and ambient air lead measurements obtained from a single sampling station in Manhattan. We now expand upon and refine these findings by considering another possible indicator of lead exposure: lead in gasoline.

The protocol for analysis of the New York City data base was to study relatively homogeneous subpopulations defined according to ethnic group, age, and quarterly sampling date. The blood lead levels

within each subpopulation, which closely approximated a lognormal distribution (5), were characterized by their geometric mean (GM). A typical plot of GM blood lead level against quarterly sampling date is shown for black and Hispanic children, aged 25-36 months, in Figure 1. The GM blood lead levels follow a consistent seasonal pattern superimposed on an overall decreasing trend from 1970 to 1976. Also graphed in Figure 1 is the (quarterly averaged) ambient air lead level over the same time period, measured at a single monitoring station in Manhattan by the U.S. Department of Energy, Health, and Safety Laboratory (6). Additional details concerning the underlying data base and the statistical analyses performed are described elsewhere (4).

An indication of the amount of lead present in gasoline was derived from industry data on monthly retail gasoline sales by grade and state (7), and from the Bureau of Mines semiannual survey of motor gasoline (8). The latter source reports the gasoline lead concentration in grams/gallon by grade on a regional basis. Data for District 2 (Mid-Atlantic Coast) have been used in the present study; gasoline lead concentrations were obtained on a quarterly basis by interpolating the reported semi-annual values.

Figure 2 graphs the resulting estimates of total amount of lead present in gasoline sold in New York, New Jersey, and Connecticut over the time period 1970-1976, as well as the same time series of GM blood lead levels given in Figure 1. The similarity in seasonal pattern and overall decline between the GM

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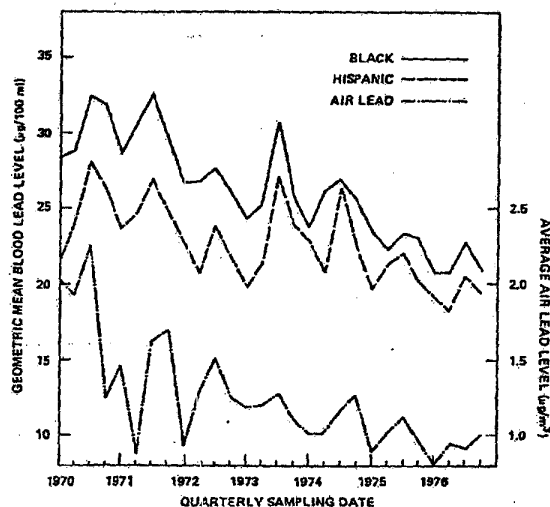


FIGURE 1. Geometric mean blood lead levels of New York City children (aged 25-36 months) by ethnic group, and ambient air lead concentration versus quarterly sampling period, 1970-1976.

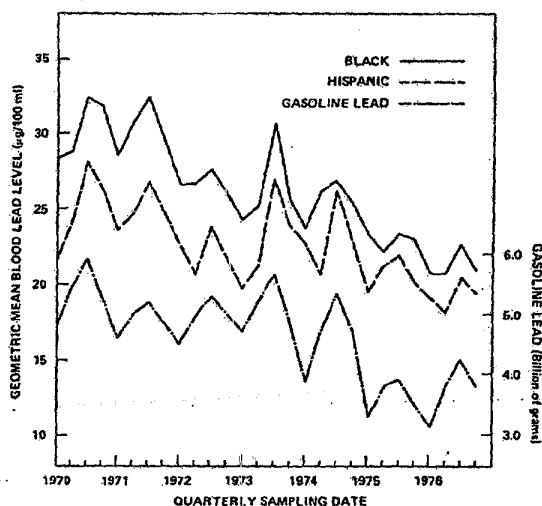


FIGURE 2. Geometric mean blood lead levels of New York City children (aged 25-36 months) by ethnic group, and estimated amount of lead present in gasoline sold in New York, New Jersey, and Connecticut versus quarterly sampling period, 1970-1976.

blood lead levels and the gasoline lead is quite striking, and is also observed for all the other age and ethnic groups. This similarity still persists whether

the total gasoline lead for New York, New York plus New Jersey, or New York plus Connecticut is used.

Moreover, the sharp peak in GM blood lead level observed for all age and ethnic groups during the summer of 1973 is paralleled by a similar peak in the level of gasoline lead at that time. Significantly, the graph of air lead levels in Figure 1 does not reflect this sharp peak.

Regression analysis has been performed on the data using two similar models to explore the correlation GM blood lead levels with age, race, season, ambient air lead and gasoline lead. The first model chosen for study was the simplified additive model used earlier (4):

$$Y = \alpha_0 + \sum_{j=1}^9 \alpha_j X_j + e \quad (1)$$

where Y is the quarterly geometric mean for the subpopulation defined by the age dummy variables $X_1, \dots, X_6$, and ethnic group dummy variables X_7, X_8 . The final variable, X_9 represents the environmental lead variable and analysis was performed using either, quarterly ambient air lead levels (in $\mu\text{g}/\text{m}^3$), or quarterly gasoline lead consumption (in billions of grams). The term e represents the statistical disturbance.

The second model selected was

$$Y = \alpha_0 + \sum_{j=1}^9 \alpha_j X_j + \sum_{j=10}^{12} \alpha_j X_j Z + e \quad (2)$$

where $X_1, \dots, X_6$ are the age dummy variables, $X_7, \dots, X_9$ season dummy variables, $X_{10}, \dots, X_{12}$ the race dummy variables. The variable Z represents either ambient air level or gasoline lead consumption.

Results from several analyses using these two models are given in Table 1. Models A, C, and E used an equation similar to Eq. (1), while B, D, and F used an equation similar to Eq. (2).

There is no a priori reason for selecting one of the models over the other. Model F appears to provide the highest statistical correlation of the observed data. Estimated values for the parameters of Model F are shown in Table 2. This model explains some 75% of the total variation in GM blood lead level as a function of age, race, season, and gasoline lead, with a residual standard deviation of $1.74 \mu\text{g}/100 \text{ ml}$. It is seen that GM blood lead level first increases monotonically and then decreases monotonically with age group, reaching a peak for 25-36 month-old children. Also, the estimated slope (with respect to gasoline lead) for blacks is significantly greater than that for Hispanics, which is significantly greater than that for whites. This table also provides a means of estimating the GM blood lead level for various subpopulations.

Table 1. Linear models relating GM blood lead level to age, race, season, air lead, and gasoline lead.

Model	Explanatory variables			R ²
	Affecting intercept	Affecting slope	Environmental	
A	Age, race		Air lead	0.599
B	Age	Race	Air lead	0.610
C	Age, race		Gas lead	0.706
D	Age	Race	Gas lead	0.718
E	Age, race		Air lead, gas lead	0.708
F	Age, season	Race	Gas lead	0.745

Table 2. Regression of GM blood lead level on age, season, race, and gasoline lead, 1970-1976.

Variable	Coefficient value	Standard deviation ^a	t ratio	Significance level
Intercept				
Constant	7.01	0.519	13.5	<0.0001
Age dummies				
13-24 months	2.37	0.274	8.6	<0.0001
25-36 months	3.11	0.274	11.4	<0.0001
37-48 months	2.73	0.274	10.0	<0.0001
49-60 months	2.32	0.274	8.5	<0.0001
61-72 months	1.73	0.274	6.3	<0.0001
> 72 months	1.06	0.274	3.9	<0.0001
Season				
Quarter 2	-1.37	0.226	-6.1	<0.0001
Quarter 3	-0.101	0.244	-0.41	0.68
Quarter 4	-0.175	0.221	-0.79	0.43
Slope (with respect to gasoline lead)				
White	2.66	0.117	22.7	<0.0001
Black	3.48	0.117	29.7	<0.0001
Hispanic	2.84	0.117	24.3	<0.0001

^aResidual Standard Deviation = 1.74; R² = 0.745.

Discussion

By far the greatest source of lead released into the environment is from the combustion of lead-containing gasoline additives. In 1968 over 90% of the estimated lead emissions in the United States were from this source (9). While the amount of lead from mobile sources has been decreasing (as illustrated in Fig. 2), automotive lead still represents the single most important dynamic source of lead into the environment. Research, monitoring and control of lead in the environment, with regard to effects on human lead levels, have for the most part concentrated on individual pathways of exposure, e.g. combustion→inhalation or combustion→air→soil deposition→ingestion (1, 9), rather than on demonstrating relationships between environmental sources and biological response. Exceptions to this approach have been those studies which relate blood lead levels and proximity to roadways or traffic volume (1).

The distribution of products emitted from automobile usage of tetraethyllead has been estimated (10). Approximately 24% is retained in the car, and the remainder is emitted to the atmosphere where it is deposited or removed by the wind. Of the total lead originally consumed, 50% is deposited while 26% remains in the ambient air.

The quantitative distribution of lead traveling the different pathways is not yet known. At best, the evidence indicates that a positive relationship exists between blood and air lead levels, although the exact functional relationship has not yet been clarified and that blood lead levels also begin to increase at soil lead levels from 500 to 1,000 ppm. Currently, it is estimated that blood lead levels increase by 1 to 2 µg/100 ml for every 1 µg/m³ increase in air lead concentration. Also the mean percent increases in blood lead levels, given a twofold increase in soil lead levels, ranging from 3 to 6 (1).

The data presented here demonstrate that blood lead levels in children are very significantly corre-

lated with a measure of gasoline lead consumption. It can be further argued that the independent variable, gasoline lead consumption in New York State (or New York, New Jersey, and Connecticut), provides a useful surrogate for average exposure of the population studied in New York City. Indeed, the true relation between this surrogate variable and the actual amount of lead in gasoline consumed in New York City is not presently known, but, for the period covered it is not unreasonable to suppose that it is highly correlated.

Our regression models have indicated that ambient air lead levels contribute very little to explaining variations in blood lead levels, after the effect of gasoline lead has been taken into account. This observation can be interpreted either in physical terms and/or as a result of the regression analysis itself.

Several uncertainties about lead measurement exist. Lead in ambient air accounts for only a third of the lead products not retained in the vehicle after combustion; furthermore there are problems with measurement of the ambient air lead. Some of the uncertainty in the latter arises from the most common method of air sampling, the high-volume sampler, which collects particulates only in the 0.1-10 μm range and does not sample vapor phase lead. It has been observed that 40-60% of the mass of collected particulate lead is below 0.4 μm , whereas less than 20% is below this diameter at the exhaust pipe (10). This raises uncertainties as to both the fraction of lead lost due to deposition between the sampling site and the emitter, as well as, the fraction which passes through the filter. The organic phase, while small, is nonetheless significant and may range as high as 13% of the total lead in urban air (11). Furthermore, some of the lead measured may be due to re-entrainment (12), possibly including non-automotive generated lead, and may not be directly correlated with gasoline lead.

The values we used for air-lead measurement are from a single source at a height of 56 m above the street (4). Thus the values used may not be representative of exposure either at street level, at that location, or at other locations in the city or even as an average for the city as a whole.

Our regression analysis between automotive gasoline lead consumption and ambient air lead does not show a high correlation, nor have other studies (13). Moreover, what is more relevant in assessing the relative contributions of air lead and gasoline lead in the models of Table 1 is not the ordinary correlation between these two variables, but rather their partial correlation coefficients.

The observation that there is little improvement in explanatory power (R^2) between the regression of blood lead on gas lead (Model C), and blood lead on

both gas lead, and air lead (Model E) simply reflects the fact that the partial correlation coefficient $r_{\text{B.A.G}}$ between blood lead and air lead, correcting for the effect of gas lead, is rather small. On the other hand, by comparing models A and E of Table 1, one can show that there is a highly significant increase in explanatory power between the regressions of blood lead on air lead, and blood lead on air lead, gas lead; this just confirms that the partial correlation coefficient $r_{\text{B.G.A}}$ between blood lead and gas lead, correcting for the effect of air lead, is statistically significant (14).

While there are many plausible explanations for the relatively small role played by air lead in explaining observed blood lead level, this finding is consistent with the hypothesis that inhalation of lead from ambient air is only a small contribution to the blood lead level (3). During the period of 1974-1976, mean blood lead levels were approximately 20-25 $\mu\text{g}/100\text{ ml}$ and ambient air lead levels never exceeded 1.3 $\mu\text{g}/\text{m}^3$. Even if we double air lead concentration to compensate for height of the sampling station, this could account for only for 2.6-5.2 $\mu\text{g}/100\text{ ml}$ blood lead, on the average, leaving 15-20 $\mu\text{g}/100\text{ ml}$ of blood lead to be explained by other sources, which could very well come from gasoline through other pathways. In reality, the exposure mechanisms are probably considerably more complex (1, 2). The lack of data on the role of secondary sources, such as dust and dirt can only lead to speculation. Further research into these pathways is needed.

The present analysis does not provide insight into the mechanisms or routes of children's exposure, other than suggesting that the total flux of lead into the environment may be more important than the concentration in any single primary or secondary medium of exposure or transport. Whatever the specific explanation turns out to be, however, it is not needed for policy analysis and decisions for control and standards for lead emissions. The present analysis suggests that emissions control rather than ambient standards are more appropriate, at least for the case of controlling blood lead levels in specific populations, such as urban children. Strong arguments can be made for using statistical analysis for such policy decisions rather than waiting for more detailed experimental data on pathways (15).

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