Effects of Reducing Lead in Gasoline: An Analysis of the International Experience

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To assess the relations between use of lead in gasoline (GPb) and population blood lead levels (BPb), we examined 19 studies from six continents. In 17 of the studies, there are data on changes in BPb before and after changes in use of lead in gasoline. In 11 of the studies, there are data on air lead levels (APb). For a given location, there is a strong linear correlation between BPb and GPb, with a median correlation coefficient of 0.94. Across locations for a given GPb, BPb is positively correlated with city population. As GPb is reduced to zero, blood lead levels across locations converge to a median of 3 μg/dL. This convergence of BPb levels occurs at different times for different locations and corresponds to the timing of gasoline lead reductions. For those locations with available air lead data, air lead concentrations converge to $\leq$0.2 μg/m^3 as GPb is reduced to zero. Together, these features indicate that reductions in gasoline lead levels have been a major causal factor in the observed reductions in population blood lead levels at these locations and show that BPb levels of about 3 μg/dL are widely achievable.

Introduction

It is well-established that leaded gasoline is a major source of population lead exposure. Isotopic experiments have demonstrated human uptake of lead from gasoline additives (*1*). Data from the United States have been widely cited in support of the strong correlation between blood lead and gasoline lead levels (*2*).

Although gasoline lead is initially emitted to the atmosphere, the pathways of human exposure to atmospheric lead include ingestion of lead deposited on soil, dust, and food crops as well as direct inhalation. The U.S. EPA has developed models that relate environmental levels of lead in air, soil, dust, and food to blood lead concentrations (*3*, *4*). However, because the relationship of lead in air to lead in soil and dust and the resulting human exposure is complex and highly variable, the models require knowledge of air lead, soil lead, dust lead, and dietary lead levels in order to predict human lead exposure. They do not directly link the use of lead in gasoline or even atmospheric lead levels to population blood lead levels. Thus, there has been little ability to predict how blood lead levels will change, for a given population, as leaded gasoline is phased out.

Now that many countries have reduced the use of lead in gasoline, there is a substantial database on how population blood lead levels have changed with reduced use of lead in gasoline. In this study, we use these data to explore the patterns of changes in population blood lead levels associated with changes in the use of lead in gasoline.

Data

We have found 17 studies, from five continents, in which population blood lead levels were measured before and after changes in the use of lead in gasoline. All studies that met this criterion were included in our analysis. As a benchmark of populations with low exposures to gasoline lead, we have also examined studies of population blood lead levels from the Himalayas and from rural Japan. Table 1 summarizes the data on gasoline lead, blood lead, and air lead concentrations from all 19 locations. A fuller description of the original studies, including reported quality control measures, is given elsewhere (*5*).

The variable used here to characterize the use of lead in gasoline is the average concentration of lead in gasoline. This variable is defined as the consumption-weighted average lead concentration of all gasoline grades sold, including unleaded gasoline. Clearly, exposure to lead in gasoline will depend not only on the concentration of lead in gasoline but also on the total amount of gasoline used as well as many other factors, such as local meteorology and sources of food. However, because some of the studies measured blood lead levels in a single city, while others measured blood lead levels in entire countries, the average concentration of lead in gasoline was the only consistent, well-defined, and available measure of the use of lead in gasoline. The highest concentration of lead in gasoline was 0.84 g/L in both Cape Town, South Africa, through 1984 and Christchurch, New Zealand, through 1985. The highest concentration of lead used in gasoline anywhere in the world is basically 0.84 g/L; at higher concentrations, lead additives provide little additional benefit to the octane rating of the gasoline.

The studies are not completely parallel. Some studies report blood lead and air lead values in major cities, others report national averages, and others refer to nonurban areas. Some studies measured adult blood lead levels, other studies measured children's blood lead levels, and still others measured blood lead levels of a representative sample of the entire population. Nevertheless, as will be seen below, there is consistency across locations in the changes in population blood lead levels associated with changes in leaded gasoline concentrations.

Analysis

Population Blood Lead versus Gasoline Lead Concentrations. Figure 1 shows the blood lead concentrations from the 17 locations versus the average concentration of lead in gasoline. Because there is considerable variation when population blood lead levels are measured at intervals of 1 year or less, the data for the studies with more frequent measurements (U.S., U.K., and Mexico City) have been averaged at 2-year intervals. Information on confidence intervals, standard deviations, and ranges of the data are given in Table 1.

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TABLE 1. Summary of Studies Containing Data on Lead in Gasoline, Air, and Blood

location	year	blood lead (μg/dL)	lead in gasoline (g/L)	air lead (μg/m^3)	population age range	refs
Athens, Greece	1979		0.80	3.2	adults	*17*, *18*
	1982	16.0	0.40	1.76		
	1984	11.8	0.22	0.91		
	1988	8	0.15	0.7		
	1993	5.5	0.14	0.43		
Barcelona, Spain	1984	18.6 ($\pm$6.6)[a]	0.60	na[b]	20−60	*19*
	1994	8.8 ($\pm$6.6)	0.13	0.24 ($\pm$0.06)[a]		
Belgium	1979	17.0	0.45	1.05	20+	*20*
	1983	14.7	0.40	0.66		
	1987	9.0	0.15	0.49		
Cape Town, South Africa	1984	9.7 ($\pm$4.1)[a]	0.84	na	adults	*21*−*23*
	1990	7.2 ($\pm$3.6)	0.40	na		
Caracas, Venezuela	1986	17.4	0.62	1.9	15+	
	1989	15.2	0.45	1.3		
	1991	15.6	0.39	1.3		
Christchurch, New Zealand	1978−1981	15.2	0.84	na	adults and children	*7*−*9*, *24*
	1982−1983	11.8	0.84	na		
	1984−1985	8.1	0.84	na		
	1989	7.3	0.45	na		
	1994	4.9	0.2	na		
Helsinki, Finland	1983	4.8 ($\pm$2.3)[a]	0.35	0.33	children	*25*, *26*
	1988	3.0 ($\pm$2.3)	0.14	0.095		
	1996	2.6 (1.7−3.7)[c]	0.00	0.007		
Himalayas, Nepal	1980	3.4	0.00	0.004	adults and children	*10*, *27*
Landskrona, Sweden	1978	6.0 (1.8−25.0)[c]	0.40	na	3−19	
	1982	4.8 (1.5−10.0)	0.15	na		
	1994	2.5 (1.2−12.3)	0.00	na		
Mexico City, Mexico	1988	12.2	0.2	na	0.5−3	*14*, *28*−*30*
	1989	14.6	0.2	na		
	1990	9.8	0.18	na		
	1991	8.6	0.08	na		
	1992	9	0.07			
	1993	7	0.06			
Ontario, Canada	1984	11.9 (11.3−12.6)[d]	0.30	na	3−6	*31*, *32*
	1988	5.1 (4.8−5.4)	0.09	na		
	1990	3.6 (3.3−3.9)	0.04	na		
	1992	3.5 (3.1−3.8)	0.00	na		
Rural Japan	1977−1980	3.8 ($\pm$ 0.2)	0.00	na	20+	*11*
Stockholm, Sweden	1980	7.7 ($\pm$ 3.3)[a]	0.40	1.20	adults	*33*
	1983	5.4 ($\pm$ 3.3)	0.15	0.50		
Switzerland	1984−1985	10.3 (8.0−17.2)[e]	0.15	na	25−74	*34*
	1988−1989	7.3 (5.6−12.7)	0.10	na		
	1992−1993	5.9 (4.4−10.2)	0.05	na		
Tarragona, Spain	1990	12.0 ($\pm$ 1.8)[a]	0.40	2.0 (0.97−3.26)[c]	6−65	*35*
	1995	6.3 ($\pm$ 1.8)	0.13	0.23		
Trelleborg, Sweden	1979	5.6 (2.7−10.4)[c]	0.40	na	3−19	*27*, *36*
	1983	4.2 (1.9−8.1)	0.15	na		
	1993	2.3 (1.0−6.7)	0.00	na		
Turin, Italy	1980	21	0.6	3	18+	*1*, *37*
	1985	15.1 ($\pm$ 3.9)[a]	0.4	2.3		
	1989		0.3	1		
	1993	6.4 ($\pm$1.7)	0.11	0.53		
United Kingdom	1979	12.9	0.42	na	adults and children	*38*−*40*
	1981	11.4	0.38	na		
	1985	9.5	0.38	0.48		
	1986	8.4	0.14	0.24		
	1995	3.1	0.055	na		
United States	1976	15.9	0.465	0.97	1−74	*2*, *6*, *41*, *42*
	1977	14.0	0.394			
	1978	14.6	0.349			
	1979	12.1	0.306	0.71		
	1980	9.5	0.30	0.49		
	1988−1991	2.8 (2.7−3.0)[d]	0.00	0.07 (0.05−0.12)[d]		

[a] Standard deviation. [b] na, not available. [c] Range. [d] 95% confidence interval. [e] 90% confidence interval.

As can be seen from Figure 1, for a given location there is a strong linear correlation between gasoline lead concentrations and population blood lead levels. For those 13 locations where population blood lead levels were measured at three or more time periods, the relation of blood lead to gasoline lead is largely linear, with a median correlation coefficient, R^2, of 0.94. Results of linear regression analysis for these 13 locations and results of linear fits for all seven-

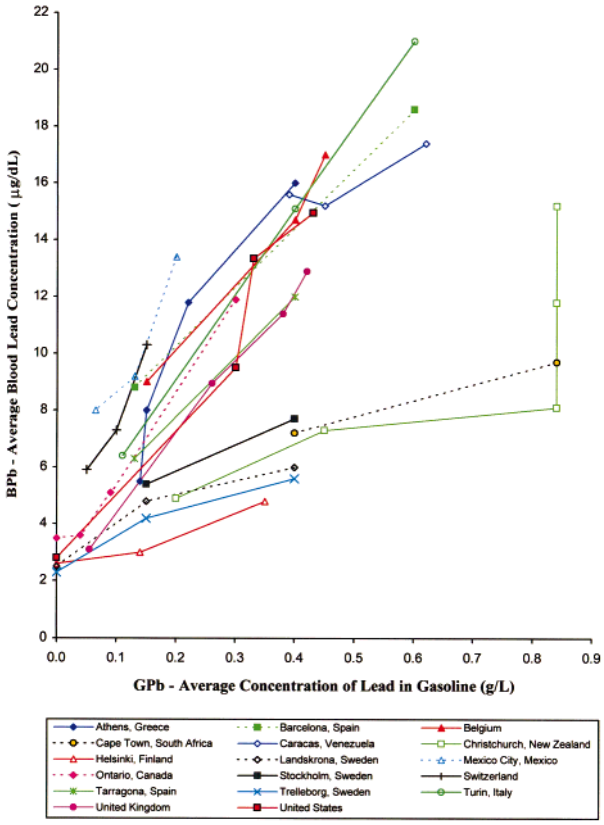

FIGURE 1. Seventeen studies of changes in population blood lead concentrations with changes in the concentration of lead in gasoline. Data are from Table 1. As shown in Table 2, the median of the linear correlation coefficients for each location is 0.94, and the median intercept of linear extrapolations to zero gasoline lead is 3.1 ± 2.3 μg/dL.

teen locations are shown in Table 2.

Some of the variation in blood lead level between locations can be explained by city population. Figure 2 includes the 11 locations that are cities or towns, as opposed to larger regions, and shows that population blood lead levels for a given gasoline concentration (in this case, 0.2 g/L) are correlated with city population. For these 11 locations, gasoline lead concentration explains 42% of the variation in population blood lead levels, city size explains 23%, and both variables together explain 60% of the variation in the population blood lead data ($R^2 = 0.6$). Thus, while city population and gasoline lead concentration can explain a substantial part of the variation in blood lead levels across locations, these two variables alone are nevertheless insufficient to accurately predict population blood lead levels.

The correlation with city population can be at least partially explained by the increase in vehicle miles driven as population increases. For a given concentration of lead in gasoline (GPb), one would expect higher blood lead levels in regions that use more gasoline and hence emit more lead. The finding of higher blood lead levels in more populated areas is consistent with the results of the U.S.-based NHANES III study, which found consistently higher blood lead levels for central city residents than for the rest of the population (*6*).

Changes in other sources of lead can also affect population blood lead levels. This is made particularly clear by the Christchurch, New Zealand, study that reports a nearly 50% drop in blood lead concentrations while gasoline lead concentrations remained unchanged (*7*−*9*). Between 1978 and 1985 in Christchurch, the average concentration of lead in gasoline remained constant at 0.84 g/L, but the geometric mean blood lead level dropped from 15.2 to 8.1 μg/dL. Between 1974 and 1983, lead was gradually removed from another source—lead-soldered food cans. Between 1974 and 1982, studies showed a 24% drop in dietary lead content and a reduction of canned food consumption from 14% to 8% of the diet (*7*). In some of the other 16 locations, lead may have been removed from food cans and other sources of exposure while gasoline lead levels were being reduced, and these measures may explain some of the reductions in blood lead levels. For example, the U.S. EPA estimates that 10−15% of the U.S. population lead exposure in 1978 was due to lead from soldered food cans (*3*).

A key feature of Figure 1 is the convergence of population blood lead levels as gasoline lead concentrations go to zero. Extrapolating linearly to zero gasoline lead, the median intercept, across all locations, is a blood lead level of 3.1 μg/dL (with a standard deviation of 2.3 μg/dL). With the exception of Caracas, Venezuela, the linear intercepts for all locations fall between 2.1 and 6.1 μg/dL. This blood lead level distribution at zero gasoline lead is consistent with levels measured in remote populations (3.4 μg/dL in the Himalayas in Nepal (*10*) and 3.4 μg/dL in rural Japan (*11*)). Some of the observed differences in blood lead intercepts at zero gasoline lead could be due to other sources of lead exposure, such as industrial emissions, lead-based paint, and lead-soldered food cans.

The linearity of the relation between gasoline lead and blood lead and the convergence to 3 μg/dL at zero gasoline lead suggest a fixed-intercept, one-parameter equation of the following form for each location *i*:

$$\mathrm{BPb}_i = a_i \mathrm{GPb}_i + 3 \qquad (1)$$

where the slope, a_i, can be determined from the gasoline lead and blood lead concentration at any point in time. For example, using the initial gasoline lead concentration, GPb_{i0}, and the initial blood lead level, BPb_{i0}:

$$a_i = \frac{\mathrm{BPb}_{i0} - 3}{\mathrm{GPb}_{i0}} \qquad (2)$$

Table 2 shows, for each location, both the slope a_i predicted by eq 2 and the best-fit slope. The correlation coefficient, R^2, for the two slopes across the 17 sites is 0.87. The median difference between the predicted and best-fit slope is ±20%.

This model predicts changes in BPb for a given change in GPb. By evaluating the change in BPb rather than its absolute value, the model isolates the effects due to GPb from all of the other factors responsible for the differences in BPb between locations.

Changes in Population Blood Lead Levels over Time. Figure 3 shows the changes in blood lead levels as a function of time. There has been a widespread decrease in population blood lead levels from the late 1970s to the early 1990s. The average decrease in population blood lead levels is 0.8 ± 0.5 μg/dL per year. Moreover, the decreases are largely linear; for the 11 locations where population blood lead levels were measured at three or more time periods, the median correlation coefficient, R^2, for a linear fit is 0.94 (Table 3). Of course, as time passes, the linearity of these data sets is certain to be broken, because blood lead levels cannot fall below zero.

Figure 3 shows that blood lead levels decreased at different times in different locations. For example, in the United States, blood lead levels fell from about 16 μg/dL to about 10 μg/dL between 1976 and 1980 (as gasoline lead concentrations fell from 0.47 to 0.3 g/L); in Athens, Greece, blood lead levels made a similar drop, from 16 to 8 μg/dL, between 1982 and 1988 (when gasoline lead concentrations were reduced from 0.4 to 0.15 g/L). The fact that blood lead levels fell in

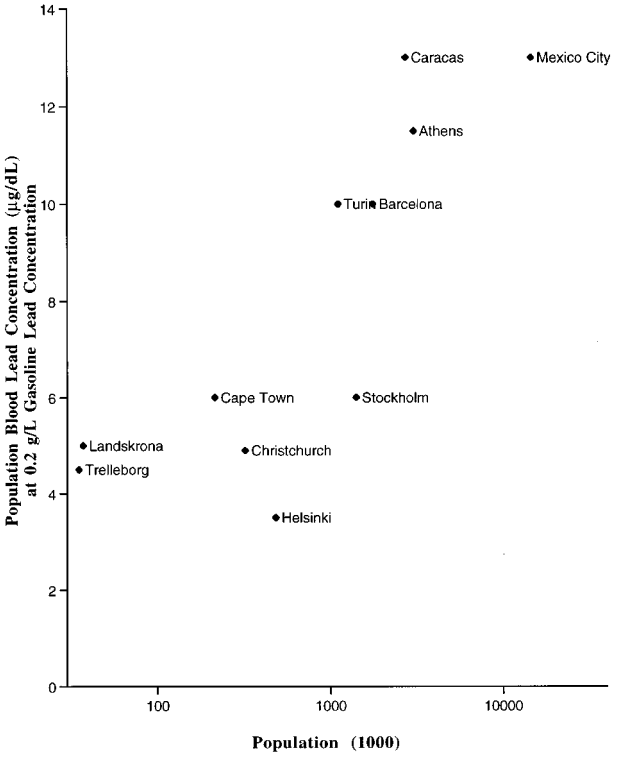

FIGURE 2. Eleven studies showing city population versus average population blood lead concentration, for an average gasoline lead concentration of 0.2 g/L.

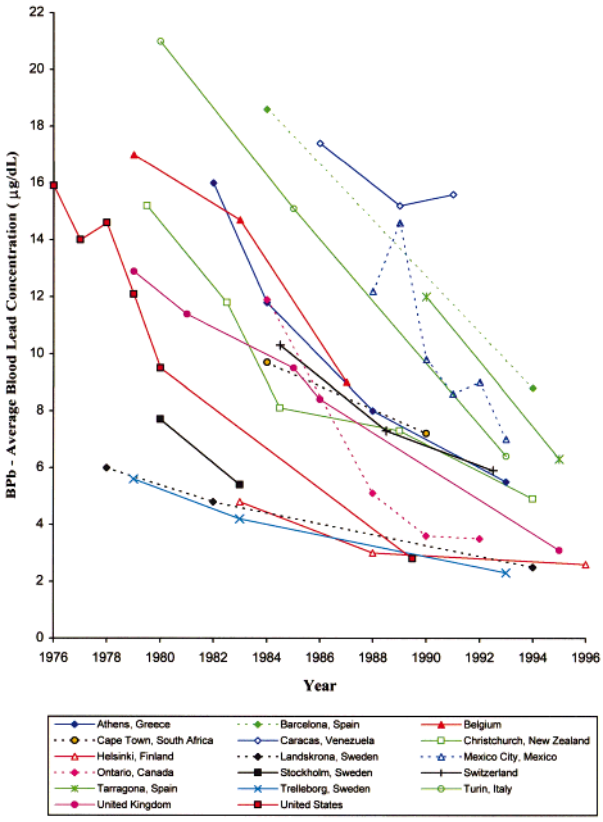

FIGURE 3. Seventeen studies of changes in population blood lead concentrations as a function of time. Data are from Table 1. As shown in Table 3, the median of the linear correlation coefficients for each location is 0.94.

conjunction with gasoline lead levels, as shown in Figure 1, and that these reductions happened at different times in different locations, as shown in Figure 3, points to gasoline lead as a key contributor to the reductions in blood lead levels.

Air Lead versus Gasoline Lead. The case for a causal relation between gasoline lead and blood lead concentrations would be strengthened by evidence that gasoline lead is a primary source of lead exposure at these locations. While detailed analysis of sources is rarely available, 9 of the 17 studies do include data on changes in air lead concentrations, and 6 of these include air lead data for more than two time points. Figure 4 shows the data on air lead concentrations versus the average concentration of lead in gasoline. At a given concentration of lead in gasoline, there is a wide variation in measured air lead concentrations. At about 0.40 g/L, for example, reported air lead concentrations range from about 0.4 $\mu g/m^3$ (Helsinki in 1983) to 2.3 $\mu g/m^3$ (Turin in 1985). Using all of the points in Figure 4, the correlation between gasoline lead concentrations and air lead concentrations has an R^2 of 0.52. This correlation shows that, while air lead is strongly influenced by gasoline lead concentrations, GPb alone is insufficient for direct prediction of air lead concentrations. A higher correlation should not have been

TABLE 2. Linear Regression Analysis of Population Blood Lead Levels (BPb) versus Gasoline Lead Concentrations (GPb)

location	no. of time periods studied	linear correlation coeff R^2	linear intercept at zero GPb (μg/dL)	best-fit slope,[a] (μg/dL)/(g/L)	slope from eq 2,[a] (μg/dL)/(g/L)
Athens, Greece	4	0.90	2.1	36	33
Barcelona, Spain	2		6.1	21	26
Belgium	3	0.98	5.1	25	31
Cape Town, South Africa	2		4.9	5.7	8
Caracas, Venezuela	3	0.83	11.7	8.9	23
Christchurch, New Zealand	5	0.61	2.6	11	15
Helsinki, Finland	3	0.98	2.5	6.5	5.1
Landskrona, Sweden	3	0.90	2.9	8.3	7.5
Mexico City, Mexico	6	0.76	5.7	33	46
Ontario, Canada	4	0.99	2.8	30	30
Stockholm, Sweden	2		4.0	9.2	11.8
Switzerland	3	0.96	3.4	44	49
Tarragona Province, Spain	2		2.8	17	22.5
Trelleborg, Sweden	3	0.95	2.6	8.0	6.5
Turin, Italy	3	1.00	3.1	30	30
United Kingdom	5	0.82	3.4	21	24
United States	5	0.94	2.6	29	28
median		0.94	3.1[b]		

[a] Correlation of slopes: $R^2 = 0.87$. Median difference = 20%. [b] SD = 2.3.

TABLE 3. Linear Regression Analysis of Population Blood Lead Levels (BPb) and Gasoline Lead Concentrations (GPb) versus Year and Air Lead Concentrations (APb) versus GPb

		linear correlation coefficients			air lead vs gasoline lead[a]	
location	no. of time periods studied	blood lead vs year R^2	gasoline lead vs year R^2	air lead vs gasoline lead R^2	best-fit slope, (μg/m^3)/(g/L)	fixed-intercept slope, eq 4, (μg/m^3)/(g/L)
Athens, Greece	4	0.92	0.68	0.98	3.4	3.6
Barcelona, Spain	2					
Belgium	3	0.94	0.87	0.69	1.5	2.3
Cape Town, South Africa	2					
Caracas, Venezuela	3	0.70	0.98	0.94	2.8	3.1
Christchurch, New Zealand	5	0.87	0.91			
Helsinki, Finland	3	0.78	0.89	0.98	0.93	0.93
Landskrona, Sweden	3	0.99	0.83			
Mexico City, Mexico	6	0.73	0.88			
Ontario, Canada	4	0.89	0.95			
Stockholm, Sweden	2				2.8	3.0
Switzerland	3	0.96	1.00			
Tarragona Province, Spain	2				7.0	5.3
Trelleborg, Sweden	3	0.98	0.86			
Turin, Italy	3	1.00	0.99	0.93	5.3	5.0
United Kingdom	5	0.96	0.54		1.0	1.3
United States	5	0.99	0.98	0.95	2.2	2.4
median		0.94	0.89	0.95		

[a] Correlation of slopes: $R^2 = 0.87$. Median difference = 9%.

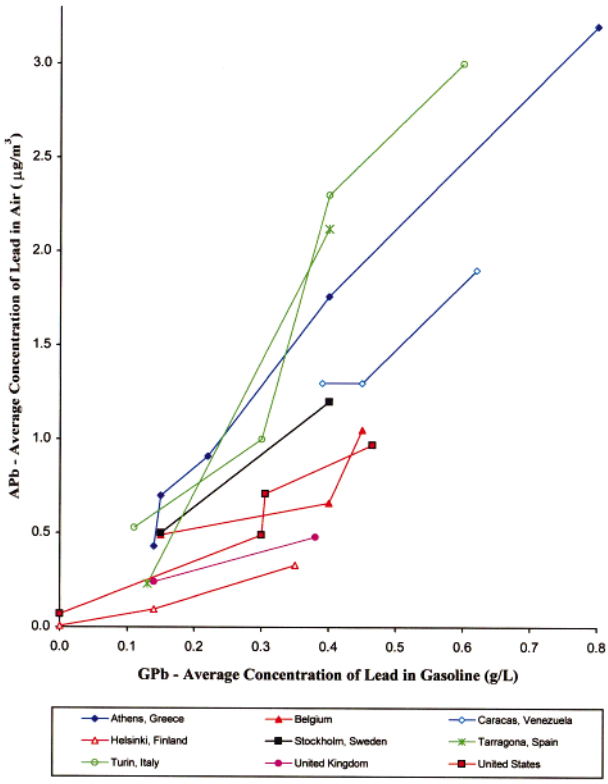

FIGURE 4. Nine studies of changes in air lead concentrations as a function of changes in gasoline lead concentrations. Data are from Table 1. As shown in Table 3, the median of the linear correlation coefficients is 0.95, and the average intercept of linear extrapolations to zero gasoline lead is ≤0.2 μg/m^3.

expected, not only because of differences in meteorology but also because air lead measurements depend on the position of the monitoring equipment and the details of the measurement and reporting procedures, which vary considerably between studies. The air lead concentration will also be affected by the vehicle miles driven in the airshed.

However, for a given location, there is a strong linear correlation between air lead concentrations and gasoline lead concentrations. For the six locations with data for more than two time points, the relations between gasoline lead and air lead concentrations are well-described by linear equations (the median correlation coefficient, R^2, of the linear fit for these six locations is about 0.95). Extrapolating linearly to zero gasoline lead, all air lead concentrations fall to 0.2 μg/m^3 or less. The variation of air lead concentration with gasoline lead concentration at a given location, for this database, is well-characterized by a zero-intercept, one-parameter equation:

$$\mathrm{APb}_i = b_i \mathrm{GPb}_i \tag{3}$$

where the slope b_i can be determined from the air lead and gasoline lead concentration at any point in time. For example, using the initial air lead concentration, APb_{i0}, and initial gasoline lead concentration, GPb_{i0}:

$$b_i = \frac{\mathrm{APb}_{i0}}{\mathrm{GPb}_{i0}} \tag{4}$$

Table 3 shows the best-fit slopes relating air lead and gasoline lead levels and the slopes as given by eq 4. The correlation coefficient, R^2, for the best-fit slope versus the slope given by eq 4 is 0.87 for all nine locations. By modeling the change in APb for a given change in GPb, the model isolates the effects due to GPb from all of the other factors responsible for the differences in APb between locations. This analysis shows that the air-lead response to a change in gasoline lead concentrations across our entire database was predictable and largely linear. While industrial emissions may have contributed to air lead levels at some of these locations, gasoline lead appears to have been the major source of lead in air. Although air lead is only one pathway to human lead exposure, these air lead data strengthen the case for a causal link between gasoline lead and blood lead levels at these locations.

Discussion

What is striking about the data on gasoline lead versus blood lead levels, shown in Figure 1, is the convergence of blood

lead levels across locations as gasoline lead levels go to zero. As shown in Figure 3, the reductions in blood lead levels happened at different times in different locations. And, as shown in Figure 4 and eqs 3 and 4, gasoline lead was by far the major contributor to air lead levels at these locations. All of these results point to gasoline lead reductions as the main cause of the reduced population blood lead levels.

The convergence of blood lead levels across locations also shows that the remaining sources of lead exposure at these locations contribute no more than about 3 μg/dL to the lead exposure of the general population. As can be seen from Figure 3, this is in striking contrast to the data from the 1970s and 1980s when blood lead levels varied by as much as a factor of 4 between these locations, from about 5 μg/dL in Helsinki, Trelleborg, and Landskrona to 18−20 μg/dL in Turin, Barcelona, and Belgium.

For countries still using lead in gasoline and especially for locations with little capacity for accurate, long-term population blood lead monitoring, the analysis developed here should be helpful in evaluating the benefits of reducing gasoline lead levels.

The 17 studies of changes in blood lead level reviewed in our analysis are not representative of the entire world. With the exception of 2 of the 17 studies (Cape Town, South Africa, and Caracas, Venezuela) all of the studies are from countries currently within the OECD. And, except for the Cape Town and Caracas studies, average gasoline lead concentrations had fallen to below 0.2 g/L by the time of the final data point.

In contrast, there are many countries in the world where little or no action has been taken to reduce lead in gasoline. Measured by the total quantity of gasoline lead additives consumed, the world's largest users of lead in gasoline—as of the late 1990s, Nigeria (where the average gasoline lead concentration is 0.66 g/L), Indonesia (0.45 g/L), Saudi Arabia (0.4 g/L), Iraq (0.4 g/L), South Africa (0.4 g/L), and Venezuela (0.37 g/L)—are countries where both gasoline consumption and gasoline lead concentrations are relatively high (*12*). A number of African countries have gasoline lead levels of up to 0.8 g/L. The two data sets included in this analysis from Caracas and Cape Town are notable in providing information about blood lead levels in non-OECD countries. By the time of the final data point, the average gasoline lead level was still 0.4 in Cape Town and 0.39 in Caracas.

The simple linear models, eqs 1−4, must not be interpreted as absolute or universal. In locations with major contamination from sources of lead exposure, such as lead-based paint, industrial lead emissions, or lead-containing household and consumer products, population blood lead levels could easily exceed 3 μg/dL even in the absence of leaded gasoline.

However, in many instances the analysis does provide a basis for predicting the results of reducing lead in gasoline. Consider the case of lead exposure in India. In the early 1980s, a study sponsored by the United Nations Environment Program and the World Health Organization reported that the median adult blood lead concentrations in Bangalore, Ahmedabad, and Calcutta were respectively 17.9, 13.8, and 10.7 μg/dL (*13*). At that time, gasoline lead concentrations in India were approximately 0.25 g/L (*14*). Equations 1 and 2 indicate that, for each of these cities, each reduction of 0.1 g/L of lead in gasoline would result in a decrease of population blood lead of about 6 μg/dL in Bangalore, 4 μg/dL in Ahmedabad, and 3 μg/dL in Calcutta. For comparison, the U.S. Centers for Disease Control has identified 10 μg/dL as the threshold for community intervention to reduce children's blood lead levels (*15*). To bring even the average blood lead concentration in Bangalore down below 10 μg/dL, eqs 1 and 2 indicate that the gasoline lead concentration would need to be reduced to about 0.11 g/L. But as of the mid-1990s, gasoline lead concentrations in India were still in the range of 0.15 g/L (*16*). Analysis such as this could add to the compelling evidence for further reductions of lead in gasoline.

The analysis of the 19 studies considered here provides a benchmark of international experience with the phase-out of leaded gasoline. The analysis shows that average population blood lead levels of about 3 μg/dL are widely achievable and strongly indicates that the phase-out of leaded gasoline has been a key contributor to decreased population blood lead levels.

Acknowledgments

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