LEAD IN PETROL:
THE MISTAKE OF THE XXTH CENTURY

Carl M. Shy

This article aims to provide some perspective of the public health consequence of using lead as an additive in petrol. The emphasis will be on leaded-petrol use in the United States of America, although the conclusions are applicable to all countries where leaded motor-vehicle fuel is still in use.

Through its use in paint and petrol, lead became one of the most widely dispersed toxins of the XXth century. The subtle effects of lead accumulation in human tissue, by multiple routes of exposure, have become known only in recent years (1). Although lead has long been recognized as a neurotoxin and haematological poison at high doses, little was known of its adverse effects at concentrations now considered to be "normal" in human blood. This lack of anticipation of the low-dose effects of lead resulted in a major mistake by the United States Public Health Service in 1925, when a Blue Ribbon Committee appointed by the Surgeon General concluded that there were "no good grounds for prohibiting use of ethyl [that is, tetraethyl lead] gasoline" (2). The arguments marshalled by proponents and opponents of leaded petrol are familiar because they apply to many other economically useful but potentially harmful substances such as pesticides, acid air pollutants and synthetic organic chemicals.

Proponents stated that leaded petrol was essential to the industrial progress of America; that it would lower the cost of motor-vehicle travel and allow for the manufacture of more powerful engines; that people would be exposed to low and insignificant doses; that illness among exposed workers was due to their own carelessness; that such exposures would not occur in the community; and finally that any innovation entails some risk. Opponents argued that lead was a poison, accumulated in the body and should not be dispersed into the environment, that workers were not responsible for lead-induced illness and death, and that the burden of proof on the safety of motor-vehicle combustion of leaded petrol should be on industry. In a paper on tetraethyl lead published in 1925 in the Journal of the American Medical Association, Alice Hamilton, one of the foremost industrial physicians of this century, and her colleagues from the Workers' Health Bureau (3) said:

"The evidence so far available seems to show a real danger of chronic lead poisoning connected with garage work when ethyl gasoline is used and a possible danger to the public from lead dust in the streets of large cities... The discharge of... [lead] particles... which fall to the ground... on crowded streets of cities, might constitute a far from negligible danger".

More than 50 years passed before Alice Hamilton's fears about population exposure to lead in combusted petrol were widely appreciated (4, 5).

Population exposure to lead from leaded petrol

Combustion of leaded petrol since 1925 accounts for about 90% of total atmospheric lead (5). The history of global lead emissions can be constructed from chronological records of lead deposition in polar snow strata, marine and freshwater sediments and annual rings of trees. Murozumi et al. (6) provide a revealing time-profile in their plot of lead concentrations in snow strata of northern Greenland. Lead in snow and ice increased linearly with time from the beginning of the industrial revolution, from 1750 until 1950, a 200-year interval during which lead concentrations increased threefold. Between 1950 and 1965, however, lead concentrations in snow and ice strata increased more than threefold again, owing to the delayed transport to northern Greenland of lead emissions from combusted leaded petrol, which was introduced in the United States in 1923 (Fig. 1). In the peak year 1972, 250 000 metric tons of lead were utilized in the United States for leaded petrol, an average of 2.4 pounds of lead per person per year (5). Between 1972 and 1984, consumption of lead for petrol additives correlated by year with a decline in ambient lead concentrations. The effect of these temporal reductions in the consumption of leaded petrol, and in air-lead levels, on blood-lead levels of the United States population was remarkable, as shown in Fig. 2 (7). Blood-lead levels of a probability sample of the United States population, as observed in the Second National Health and Nutrition Examination Survey (NHANES II) (8), declined by 37% between 1976 and 1980. During the same period, the estimated lead intake in the diet of teenage males showed no change (4). The correlation between changes in blood lead, adjusted for demographic variables, and leaded petrol use, was particularly strong for white children aged 6 months-5 years, with a correlation coefficient of 0.95. Thus, while inhalation of lead in air contributes only 1-2% of the total lead intake of humans, indirect exposure to atmospheric lead via ingestion and inhalation of lead in dust, soil, food and water can contribute up to 50% of the total lead intake. In support of the NHANES II results are temporal declines in blood-lead levels of black children aged 2-3 years examined in lead-screening programmes in New York City and Chicago between 1970 and 1980 (9, 10), among 5-6 year-old schoolchildren studied in Newark, N.J., from 11 847 births between 1979 and 1981 in Boston (12).

Based on NHANES II results, it is evident that blood-lead levels vary by age and race (Fig. 3); the highest levels occur in the first few years of life, and blacks have higher lead levels than whites at all ages. Superimposed on these demographic factors is an effect of degree of urbanization; high blood-lead levels are found among black children of central cities of the largest metropolitan areas, and the lowest values among white rural children (Table 1). Depending upon the boundary for defining excess blood lead, from 200 000 to 2 380 000 children aged

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FIG. 1
LEAD CONCENTRATION PROFILE IN SNOW STRATA OF NORTHERN GREENLAND
PROFIL DE LA CONCENTRATION DE PLOMB DANS LES COUCHES DE NEIGE DU GROENLAND SEPTENTRIONAL

Source: Reference (6) – Référence (6).

FIG. 2
PARALLEL DECREASE IN BLOOD-LEAD VALUES OBSERVED IN THE SECOND NATIONAL HEALTH AND NUTRITION EXAMINATION SURVEY AND AMOUNTS OF LEAD USED IN PETROL DURING 1976-1980, UNITED STATES OF AMERICA

Source: Reference (7) – Référence (7).
BLOOD-LEAD LEVELS BY RACE AND AGE IN THE UNITED STATES OF AMERICA ACCORDING TO THE SECOND NATIONAL HEALTH AND NUTRITION EXAMINATION SURVEY, 1976-1980


FIG. 3

6 months-5 years living in metropolitan areas of the United States in 1984 were estimated to have blood-lead levels associated with adverse health effects (Table 2).

There are several major environmental sources of these excess blood-lead levels. As shown in Table 3, lead in paint, petrol dusts and soil is responsible for the largest number of more highly exposed children. Unfortunately, there is considerable overlap of children exposed to these environmental sources, in that children living in central cities are often simultaneously exposed to the highest air and soil/dust lead levels and to the highest density of older homes painted with lead-based paint. An important source of lead exposure to the fetus is lead in the blood of pregnant women. Not only does lead transfer across the placenta to the fetus, but women appear to mobilize lead as well as calcium from their own body stores during pregnancy and transfer these substances to the fetus (13). The Agency for Toxic Substances and Disease Registry (14) estimated that there were 400 000 pregnant women living in metropolitan areas in 1984 whose blood lead exceeded 10 mg/100 ml, a level associated with potentially adverse development of fetuses and newborn infants.

That atmospheric lead, and therefore leaded petrol, contributes substantially to the total lead intake of human populations has been demonstrated in two studies. Firstly, the NHANES II survey showed a 37%
TABLE 2. ESTIMATED NUMBER OF CHILDREN AGED 6 MONTHS-5 YEARS WITH EXCESS BLOOD LEAD IN METROPOLITAN AREAS, UNITED STATES OF AMERICA, 1984

<table>
<thead>
<tr>
<th>Metropolitan area — Zone métropolitaine</th>
<th>Bloodlead — Plombémie (μg/100cc)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&gt;15</td>
</tr>
<tr>
<td></td>
<td>&gt;20</td>
</tr>
<tr>
<td>Large — Grande (&gt;1 million)</td>
<td>1493 400</td>
</tr>
<tr>
<td>(Percentage children — Pourcentage d’enfants)</td>
<td>(21%)</td>
</tr>
<tr>
<td>Small — Petite (&lt;1 million)</td>
<td>483 000</td>
</tr>
<tr>
<td>(Percentage children — Pourcentage d’enfants)</td>
<td>(13%)</td>
</tr>
<tr>
<td>Total metropolitan areas — Total pour les zones métropolitaines</td>
<td>2380 600</td>
</tr>
<tr>
<td>(Percentage children — Pourcentage d’enfants)</td>
<td>(17%)</td>
</tr>
</tbody>
</table>

Source: Reference (14) — Référence (14).

TABLE 3. SOURCES OF CHILDHOOD LEAD EXPOSURE, UNITED STATES OF AMERICA, 1984

<table>
<thead>
<tr>
<th>Source of lead — Source de plomb</th>
<th>Estimated number of children highly exposed (millions)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Maternal blood — Sang maternal</td>
<td>0.4 a</td>
</tr>
<tr>
<td>2. Paint — Peinture</td>
<td>5.9 b</td>
</tr>
<tr>
<td>3. Petroleum — Essence</td>
<td>5.6 c</td>
</tr>
<tr>
<td>4. Stationary sources — Sources fixes</td>
<td>0.013 d</td>
</tr>
<tr>
<td>5. Dusts and soils — Poussières et sol</td>
<td>5.9-11.7 e</td>
</tr>
<tr>
<td>6. Drinking-water — Eau potable</td>
<td>3.8 f</td>
</tr>
<tr>
<td>7. Food — Aliments</td>
<td>&lt;1.0 g</td>
</tr>
</tbody>
</table>

a Number of pregnant women in 1984 with blood lead >10 μg/100 cc in standard metropolitan statistical areas — Nombre de femmes enceintes en 1984 avec une plombémie >10 μg/100 cc dans les zones statistiques standard des grandes agglomérations.

b Number of children <7 living in oldest, highest-point residential units — Nombre d’enfants <7 ans vivant dans les unités résidentielles les plus anciennes et les plus élevées.

Number of children potentially exposed to lead from combusted petrol and residing in the 100 largest cities — Nombre d’enfants potentiellement exposés au plomb provenant de l’essence en combustion et résidant dans les 100 plus grandes villes.

Number of children exposed to primary and secondary lead smelter, and who have blood-lead levels >20 μg/100 cc — Nombre d’enfants exposés à une fonderie primaire ou secondaire de plomb, avec une plombémie >20 μg/100 cc.

Number of children exposed to lead paint, leaded petrol combustion, or stationary sources of lead — Nombre d’enfants exposés à la peinture au plomb, à la combustion d’essence contenant du plomb ou à des sources de plomb fixes.

Number of children exposed to residential drinking-water containing the Environmental Protection Agency proposed level of 20 μg/l — Nombre d’enfants exposés chez eux à de l’eau de boisson contenant le niveau de plomb proposé par l’Environmental Protection Agency de 20 μg/l.

d Number of children exposed to residual petrol — Nombre d’enfants exposés à des aliments de provenance dépassant les concentrations en plomb recommandées.

Source: Reference (14) — Référence (14).

Health effects of ambient air lead

Table 4 summarizes the most current estimates of blood-lead levels at which adverse effects may occur in children (5, 14). Prior to 1975, a blood lead of 40 mg/100 ml was considered to be the lowest observable effect level. However, in the past 15 years, a number of well-conducted longitudinal studies of children conclude that adverse developmental effects occur in early life in association with blood lead in the range of 15-25 mg/100 ml (16). The developmental effects of chronic low-level lead exposure in early life include:

- reduced birthweight;
- impaired mental development in the first two years of life;
- IQ deficits in school-age children; and
- disturbances in sensory pathways within the central nervous system persisting for five or more years.

The cognitive and neurosensory effects of slightly increased blood-lead levels are particularly difficult to study for several reasons: (i) there are many different tests of cognition and neurosensory function; (ii) these tests are not equally standardized; (iii) social class, home environment, birth order, inheritance and other factors are important deter-
minants of cognitive abilities, and some of these factors are difficult to measure; finally, (iv) an operational model for understanding the effect of lead on growth in cognitive and neurosensory function is lacking. Thus it is not surprising that there are inconsistencies in the existing body of studies relating relatively low blood lead to intellectual and sensory development. However there is an impressive convergence of evidence from experimental animal studies and from human epidemiological observations on the effect of lead on learning and neurological function. Even those epidemiological studies that fail to find a statistically significant effect of slightly high blood-lead levels consistently detect effects in the postulated direction of adverse outcomes.

Two epidemiological studies illustrate the nature and complexity of the findings. Bellinger et al. (17, 18) studied the relationship between umbilical cord blood lead and early cognitive development between 6 and 24 months of age. Cord-blood samples were obtained for 9,489 births in Boston at the Brigham and Women’s Hospitals, and those in the lowest 10th, middle 10th and highest 10th percentiles of lead (n=1,207) were eligible for study; the final sample consisted of 249 of these infants. The enrolled infants were healthy at birth and their parents were in the top two social strata according to Hollingshead’s index; 87% of the families were white. Cord-blood lead levels in the highest 10th percentile groups ranged only from 10-25 mg/100 ml, a level considered to be within normal variation. Infants were tested at 6, 12, 18 and 24 months, using the Bayley Scales of Infant Development. Fig. 4 shows results for the three cord-blood lead groups in terms of the mental development index score at six-month intervals. The mental development index is a composite age-corrected scale that assesses sensory-perceptual acuities, memory, learning and problem-solving ability, verbal communication, and early ability to form generalizations, as well as other cognitive functions; 15-20% of infants did not complete all four test runs, and losses were greater among infants born to nonwhite or unmarried mothers and to mothers of lower age, education level and socioeconomic status. Mental Development Index scores were regressed against cord-blood lead and 12 factors associated with infant development, including mother’s age, race, IQ, education, smoking and alcohol habits, social class, care-giving environment and infant’s sex, birthweight and birth order, and gestational age. At all ages, infants in the high prenatal exposure group scored lower than infants in the other two groups, and the difference between the high and each of the other two exposure groups was statistically significant. Test scores were not associated with concurrent postnatal blood-lead levels or with cumulative postnatal lead exposure up to the time of assessment. Blood-lead differences between the three groups were much larger for cord-blood than for postnatal blood-lead levels.

The striking feature of this study is that the mean blood-lead level of the study sample as a whole was about one-half the mean level of United States preschool-age children, based on measurements made in the NHANES II survey. Likewise, study children came from families in the higher socioeconomic strata. These factors reduced the likelihood of finding an effect of prenatal lead exposure during the first two years of life, especially if a favourable home and family environment can offset the potentially small effects of prenatal lead on cognitive development. In their later publication (18), the authors presented evidence that the performance of children in the lower socioeconomic stratum (of this relatively high socioeconomic sample) was adversely affected at lower levels of prenatal exposure, i.e. cord-blood
lead levels of 6-7 mg/100 ml, than children in the higher socioeconomic stratum. However these results are based on small numbers of children in each lead and social class stratum.

The second study, conducted in Port Pirie, South Australia, investigated a cohort of 539 children born between 1979 and 1982 to women living in a community near a lead smelter (19-20). This study provided a considerably larger sample in a community with presumably higher environmental lead exposures and probably a distribution of families of lower socioeconomic strata than in the Boston study. The mean blood-lead concentration in the Port Pirie children at age 15 months was 21 mg/100 cc, and one-third of the children had levels above 25 mg/100 cc on one or more occasions. Maternal intelligence, home environment and the children’s mental development were assessed, the latter with use of the McCarthy Scale of Children’s Abilities, from which a general cognitive index was derived to reflect reasoning, concept formation and memory.

Blood-lead concentrations at each age and the integrated postnatal average blood-lead level were inversely related with the general cognitive index score at age 4 years, as shown in Fig. 5. General cognitive index score is adjusted for 16 factors identified a priori as potential determinants of mental development. The regression line of the figure shows a drop of 7.2 units in the general cognitive index score (where 100 is the expected average score value for a population) associated with an increase in blood lead from 10 to 30 mg/100 ml. There was no evidence that cognitive function at age 4 was more influenced by recent than by earlier blood-lead levels, or that children whose blood-lead levels increased with age were more affected than those with constant levels.

* FIG. 4
MEAN MENTAL DEVELOPMENT INDEX SCORES AT FOUR AGES IN INFANTS ACCORDING TO THE LEAD LEVEL IN UMBILICAL-CORD BLOOD *

INDICES MOYENS DU DÉVELOPPEMENT MENTAL À QUATRE STADES CHEZ LES NOURRISONS SELON LE TAUX DE PLOMBÉMIE CORDALE *
These two studies suggest that there is no clear threshold for the effects of lead on cognitive development in early life. While the failure to find a threshold is not surprising, the existence or lack of a threshold cannot be readily demonstrated in observational studies because enormous sample sizes are needed to compare effects at low levels of exposure. The practical conclusion of these and other studies is fairly clear with respect to lead—there should be as little human exposure as possible, and all evidence points to a greater risk of a variety of adverse effects, particularly effects on cognition and haematological function, at what were formerly considered normal blood-lead levels.

**Lessons learned retrospectively**

Today, 65 years after the United States Surgeon General’s Blue Ribbon Committee concluded that there was no good evidence for prohibiting use of leaded petrol, it can reasonably be claimed that lead is the environmental health disaster of the XXth century. Even discounting the thousands of children who were acutely and severely poisoned by ingestion of lead-based paint residues, there are still millions of children whose cognitive and neurosensory development was probably compromised by excess lead intake attributable to leaded petrol combustion. To this can be added the known lead-induced effects on fetal development, haematological function and blood pressure, and the yet-to-be-discovered effects of mobilization of high body stores of lead among the elderly during periods of bone resorption (21).

How could this environmental health calamity have been avoided? With hindsight, were there major clues missed by the 1925 Blue Ribbon Committee that are applicable to future technological development? Several can be listed.

- **Lead** is a cumulative toxin. Lead combusted in petrol adds to the body burden imposed by other environmental sources. Cumulative toxins from multiple sources magnify the risk of excessive population exposures. Other heavy metals and many chlorinated hydrocarbons also pose these risks.
- **Leaded petrol** was initially associated with energy consumption, which increased dramatically over the next 50 years. Even slight increases in hazardous byproducts of energy consumption have the potential for a widespread adverse public health impact.
- **Combustion** of leaded petrol in millions of automobiles resulted in extraordinarily dispersive contamination of residential environments. Therefore, any other additive to petrol should raise similar concerns, and any hazardous pollutant emitted by motor-vehicle emissions will similarly expose a large segment of the population to potential adverse health effects. Ozone and carbon monoxide are known concerns.
- **Although** the adverse effects of low levels of blood lead were unknown in 1925, there was ample reason for concern. Lead was a known poison to enzyme systems, and as such had the potential to interfere with essential biochemical pathways at low doses, with virtually no threshold. It would have been reasonable to extrapolate from high-dose acute human effects, for which there was abundant evi-

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**FIG. 5**

**LINEAR RELATION, AS ESTIMATED FROM MULTIPLE REGRESSION ANALYSIS, BETWEEN THE INTEGRATED POSTNATAL AVERAGE BLOOD-LEAD CONCENTRATION (SHOWN ON A LOGARITHMIC SCALE) AND THE GENERAL COGNITIVE INDEX (GCI)**

**CORRÉLATION LINÉAIRE ÉVALUÉE PAR REGRÉSSION MULTIPLE ENTRE LA PLOMBÉMIE MOYENNE CUMULÉE EN PERIODE POSTNATALE (ÉCHELLE LOGARITHMIQUE) ET L’INDICE DE MATURATION CÉREBRALE**

![Graph showing the relationship between blood-lead concentration and general cognitive index](image)

Source: Reference (19) — Référence (19).

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toxicity experts, The enquisines nationales have not been able to evaluate these institutions are obsessed with toxins and neurotoxins.

- Government, industry and scientific advisers to these institutions are obsessed with toxins and neurotoxins.

- In spite of warnings from certain experts in materia de plomb, la pratique consistant à mettre du plomb dans l’essence s’est répandue dans le monde.

- The health aspects of the use of lead in petrol were evaluated in the 1920s in the United States of America and, in spite of warnings from certain lead-toxicity experts, lead addition to petrol became standard international practice.

- Available data now show that lead in petrol at the scale of use in the 1970s produced significant environmental lead contamination and increased average blood-lead levels in the general population. National sample surveys of blood-lead levels in the United States carried out annually from 1976 show a decreasing trend closely correlated with the use of lead in petrol.

- Recent longitudinal epidemiological studies have concluded that the exposure levels associated with lead in petrol can cause a reduced average mental ability in children. These studies accounted for the potential confounding from socioeconomic and other factors. The practical conclusion from the studies reviewed is that there should be as little human lead exposure as possible, because there may be no threshold for the effects occurring and many thousand children have already been affected in the United States and other countries.

- The environmental health calamity caused by lead in petrol could have been avoided if the initial warnings had been heeded and better preliminary research of the health issues had been carried out. Nevertheless, incontrovertible proof of causality should not be required before regulations are made to protect public health.

- If a new technology having the potential for widespread population exposure to environmental contaminants emerges, one cannot rely on high-dose toxicological studies to assess human risk, particularly for a complex technology. There is ample time to evaluate human exposures, sometimes even by controlled experiments, certainly by obtaining good-quality baseline exposure and health data in pilot communities, and by performing appropriate epidemiological follow-up of the communities. Protection of public health must be an absolute goal, after which the economic benefit of technologies can be considered. Our XXth century experience with environmental lead should drive this lesson firmly home.

In 1786, Benjamin Franklin wrote:

“The Opinion of the mischievous Effect of Lead is at least Sixty Years old; and you will observe with Concern how long a useful Truth may be known and exist, before it is generally received and practiced on” (22).

Following Franklin’s advice, let us not delay another 60 years, as we did before removing lead from petrol, before acting on the evidence of the “mischievous effects” of other pervasive environmental hazards.

**SUMMARY**

The health aspects of the use of lead in petrol were evaluated in the 1920s in the United States of America and, in spite of warnings from certain lead-toxicity experts, lead addition to petrol became standard international practice.

Available data now show that lead in petrol at the scale of use in the 1970s produced significant environmental lead contamination and increased average blood-lead levels in the general population. National sample surveys of blood-lead levels in the United States carried out annually from 1976 show a decreasing trend closely correlated with the use of lead in petrol.

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**RÉSUMÉ**

**Essence contenant du plomb — l’erreur du XXe siècle**

Les incidences pour la santé de la présence de plomb dans l’essence ont été évaluées dans les années 20 aux États-Unis d’Amérique et, malgré les mises en garde de certains experts en matière de toxicité du plomb, la pratique consistant à mettre du plomb dans l’essence s’est répandue dans le monde.

On sait maintenant que le plomb contenu dans l’essence, à l’échelle où il était utilisé dans les années 70, a sérieusement contaminé l’environnement et entraîné une augmentation du taux moyen de plombémie dans l’ensemble de la population. Les enquêtes nationales par sondage sur les taux de plombémie aux États-Unis faites chaque année depuis 1976 ont apparu à une tendance décroissante, étroitement liée à la quantité de plomb utilisée dans l’essence.

Des études épidémiologiques longitudinales récentes ont montré que l’exposition au plomb contenu dans l’essence, selon le niveau, pouvait se traduire par une baisse de la capacité intellectuelle moyenne des enfants. Ces études tiennent compte des dispositions possibles dues aux facteurs socio-économiques et autres. La conclusion pratique qui se dégage de ces études est que l’homme doit éviter
au maximum toute exposition au plomb car il se peut qu’il n’y ait pas d’exposition sans effets et des milliers d’enfants ont déjà été affectés aux États-Unis et dans d’autres pays.

Le fléau qui constitue la présence de plomb dans l’essence pour l’hygiène de l’environnement aurait pu être évité si l’on avait tenu compte des premiers avisements donnés et que les incidences sanitaires avaient d’abord été étudiées plus en profondeur. Il ne devrait cependant pas être nécessaire que la preuve irréfutable soit faite qu’il existe une relation de cause à effet pour protéger la santé publique au moyen d’une réglementation appropriée.

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