

Leaded Gasoline: An overview of environmental and health impacts

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INTRODUCTION

Since the introduction of tetraethyl lead as an antiknock agent in gasoline, vehicular lead has made an important contribution to lead exposure in the general population, which, together with other sources of lead emissions, has been associated with adverse health effects in humans. Lead is considered as a pollutant that is persistent in all media of the environment. Once absorbed in the human body it can remain in tissues and bones for long periods. Regulators in many countries are now aware of the insidious consequences of cumulative low-level exposure to lead, most notably decrements in neurobehavioural development of children. In many countries, lead has been or is being phased out as a consequence of this appreciation. However, in countries that have not yet started phasing out lead as an antiknock agent, most of the lead in their environment originates still from vehicular emissions. In those countries, the addition of tetraalkyllead in motor fuels accounts for an estimated 80-90% of lead in ambient air. The degree of pollution from this source differs from country to country, depending on motor vehicle density and efficiency of efforts to reduce the lead content of petrol.

The present overview on the environmental and human health effects is based on reviews prepared by the World Health Organization (WHO 1989; 1995; 2000) and a recent report from Wadgaonkar (1999) and Romieu (1999).

LEVELS OF TRAFFIC-RELATED LEAD

Sources of human exposure

Atmospheric lead concentrations of 50 pg/m³ have been found in remote areas. In developed countries, where most gasoline sold is unleaded, typical annual average concentrations are between 0.1 and 0.2 µg/m³ in urban areas and between 0.01 and 0.05 µg/m³ in rural parts. Higher annual average concentrations are found in the vicinity of industrial sites, ranging between 0.2 and 1 µg/m³. Over the past 25 years, levels of airborne lead have fallen considerably as a result of progressive reductions in the maximum permitted concentration of lead in gasoline and increased use of unleaded petrol. A typical example of decrease of lead concentrations in urban sites is presented in Figure 1.

Insert here Figure 3 of WHO 1995.

Figure 1 Comparison of national trend (USA) in the composite average of the maximum quarterly lead concentrations at urban and point source oriented sites.

Concentrations in excess of $1 \mu\text{g}/\text{m}^3$ are recorded in cities in developing countries, which still heavily rely on leaded gasoline.

Background levels of lead in soil range between 10 and 70 mg/kg and a mean level near roadways of 138 mg/kg has been reported. Present levels of lead in water rarely exceed a few micrograms/litre; the natural concentration of lead in surface water has been estimated to be $0.02 \mu\text{g}/\text{litre}$.

Environmental transport, distribution and transformation

Airborne lead can be deposited on soil and water, thus reaching humans through the food chain and in drinking-water. Atmospheric lead is also a major source of lead in household dust.

The transport and distribution of lead from fixed, mobile and natural sources are primarily via air. Most lead emissions are deposited near the source, although some particulate matter ($< 2 \mu\text{m}$ in diameter) is transported over long distances and results in the contamination of remote sites such as arctic glaciers. Airborne lead can contribute to human exposures by the contamination of food, water and dust, as well as through direct inhalation. The removal of airborne lead is influenced by atmospheric conditions and particulate size. Large amounts of lead may be discharged to soil and water. However, such material tends to remain localized because of the poor solubility of lead compounds in water.

Lead deposited in water, whether from air or through run-off from soils, partitions rapidly between sediment and aqueous phase, depending upon pH, salt content, and the presence of organic chelating agents. Above pH 5.4, hard water may contain about $30 \mu\text{g}$ lead/litre and soft water about $500 \mu\text{g}$ lead/litre. Very little lead deposited on soil is transported to surface or ground water except through erosion or geochemical weathering; it is normally quite tightly bound (chelated) to organic matter.

Environmental levels and human exposure

In the general non-smoking adult population, the major exposure pathway is from food, dust and water as shown in Figure 2.

Insert here Figure 11 of WHO 1995.

Figure 2 Percentage of lead intake from food and other sources in two-year old infants and woman of child-bearing age in the USA.

Airborne lead may contribute significantly to exposure, depending upon such factors as use of tobacco, occupation, proximity to motorways, lead smelters, etc., and leisure activities (e.g., arts and crafts, firearm target practice). Food, air, water and dust/soil are the major potential exposure pathways for infants and young children. For infants up to 4 or 5 months of age, air, milk, formulae and water are the significant sources of lead exposure.

Levels of lead found in air, food, water and soil/dust vary widely throughout the world and depend upon the degree of industrial development, urbanization and lifestyle factors. Ambient air levels over $10 \mu\text{g}/\text{m}^3$ have been reported in urban areas near a smelter, whereas lead levels below $0.2 \mu\text{g}/\text{m}^3$ have been found in cities where leaded petrol is no longer used. Lead intake from air can, therefore, vary from less than $4 \mu\text{g}/\text{day}$ to more than $200 \mu\text{g}/\text{day}$.

Levels of lead in drinking-water sampled at the source are usually below $5 \mu\text{g}/\text{litre}$. However, water taken from taps (faucets) in homes where lead is present in the plumbing can contain levels in excess of $100 \mu\text{g}/\text{litre}$, particularly after the water has been standing in the pipes for some hours.

The level of dietary exposure to lead depends upon many lifestyle factors, including foodstuffs consumed, processing technology, use of lead solder, lead levels in water, and use of lead-glazed ceramics.

HEALTH EFFECTS OF LEAD

In humans, lead can result in a wide range of biological effects depending upon the level and duration of exposure. Effects at the sub cellular level, as well as effects on the overall functioning of the body, have been noted and range from inhibition of enzymes to the production of marked morphological changes and death. Such changes occur over a broad range of doses, the developing human generally being more sensitive than the adult.

Lead has been shown to have effects on many biochemical processes; in particular, effects on haem synthesis have been studied extensively. Increased levels of serum erythrocyte protoporphyrin and increased urinary excretion of coproporphyrin and δ -aminolaevulinic acid are observed when PbB concentrations are elevated. Inhibition of the enzymes δ -aminolaevulinic acid dehydratase and dihydrobiopterin reductase is observed at lower levels.

The effects of lead on the haemopoietic system result in decreased haemoglobin synthesis, and anaemia has been observed in children at PbB concentrations above $1.92 \mu\text{mol}/\text{litre}$ ($40 \mu\text{g}/\text{dl}$).

For neurological, metabolic and behavioural reasons, children are more vulnerable to the effects of lead than adults. Both prospective and cross-sectional epidemiological studies have been conducted to assess the extent to which environmental lead exposure affects CNS-based psychological functions. Lead has been shown to be associated with impaired neurobehavioural functioning in children. These findings are illustrated in Figure 3 and Figure 4.

Insert here Figure 15 of WHO 1995.

Figure 3 Estimated mean change in IQ for an increase in blood level from 0.48 to 096 $\mu\text{mol/litre}$ (10 to 20 $\mu\text{g/dl}$) in prospective studies.

Insert here Figure 17 of WHO 1995.

Figure 4 Estimated mean change in IQ for an increase in blood level from 0.48 to 096 $\mu\text{mol/litre}$ (10 to 20 $\mu\text{g/dl}$) in cross-sectional studies.

Impairment of psychological and neurobehavioural functions has been found after long-term lead exposure of workers. Electrophysiological parameters have been shown to be useful indicators of subclinical lead effects in the CNS.

Peripheral neuropathy has long been known to be caused by long-term high-level lead exposure at the workplace. Slowing of nerve conduction velocity has been found at lower levels. These effects have often been found to be reversible after cessation of exposure, depending on the age and duration of exposure.

The effect of lead on the heart is indirect and occurs via the autonomic nervous system; it has no direct effect on the myocardium. The collective evidence from population studies in adults indicates very weak associations between PbB concentration and systolic or diastolic blood pressure. Given the difficulties of allowing for relevant confounding factors, a causal relationship cannot be established from these studies. There is no evidence to suggest that any association of PbB concentration with blood pressure is of major health importance.

Lead is known to cause proximal renal tubular damage, characterized by generalized aminoaciduria, hypophosphataemia with relative hyperphosphaturia and glycosuria accompanied by nuclear inclusion bodies, mitochondrial changes and cytomegaly of the proximal tubular epithelial cells. Tubular effects are noted after relatively short-term exposures and are generally reversible, whereas sclerotic changes and interstitial fibrosis, resulting in decreased kidney function and possible renal failure, require chronic exposure to high lead levels. Renal effects have recently been seen among the general population when more sensitive indicators of function were measured.

The reproductive effects of lead in the male are limited to sperm morphology and count. In the female, some adverse pregnancy outcomes have been attributed to lead.

Lead does not appear to have deleterious effects on skin, muscle or the immune system. Except in the case of the rat, lead does not appear to be related to the development of tumours.

Evaluation of human health risks

Lead adversely affects several organs and organ systems, with subcellular changes and neurodevelopmental effects appearing to be the most sensitive. An association between PbB level and hypertension (blood pressure) has been reported. Lead produces a cascade of effects on the haem body pool and affects haem synthesis. However, some of these effects are not considered adverse. Calcium homeostasis is affected, thus interfering with other cellular processes.

- a) The most substantial evidence from cross-sectional and prospective studies of populations with PbB levels generally below 1.2 $\mu\text{mol/litre}$ (25 $\mu\text{g/dl}$) relates to decrements in intelligence quotient (IQ). It is important to note that such observational studies cannot provide definitive evidence of a causal relationship with lead exposure. However, the size of the apparent IQ effect, as assessed at 4 years and above, is a deficit between 0 and 5 points (on a scale with a standard deviation of 15) for each 0.48 $\mu\text{mol/litre}$ (10 $\mu\text{g/dl}$) increment in PbB level, with a likely apparent effect size of between 1 and 3 points. At PbB levels above 1.2 $\mu\text{mol/litre}$ (25 $\mu\text{g/dl}$), the relationship between PbB and IQ may differ. Estimates of effect size are group averages and only apply to the individual child in a probabilistic manner.

Existing epidemiological studies do not provide definitive evidence of a threshold. Below the PbB range of 0.48-0.72 $\mu\text{mol/litre}$ (10-15 $\mu\text{g/dl}$), the effects of confounding variables and limits in the precision in analytical and psychometric measurements increase the uncertainty attached to any estimate of effect. However, there is some evidence of an association below this range.

- b) Animal studies provide support for a causal relationship between lead and nervous system effects, reporting deficits in cognitive functions at PbB levels as low as 0.53-0.72 $\mu\text{mol/litre}$ (11-15 $\mu\text{g/dl}$) which can persist well beyond the termination of lead exposure.
- c) Reduction in human peripheral nerve conduction velocity may occur with PbB levels as low as 1.44 $\mu\text{mol/litre}$ (30 $\mu\text{g/dl}$). In addition, sensory motor function may be impaired with PbB levels as low as about 1.92 $\mu\text{mol/litre}$ (40 $\mu\text{g/dl}$), and autonomic nervous system function (electrocardiographic R-R interval variability) may be affected at an average PbB level of approximately 1.68 $\mu\text{mol/litre}$ (35 $\mu\text{g/dl}$). The risk of lead nephropathy is increased in workers with PbB levels above 2.88 $\mu\text{mol/litre}$ (60 $\mu\text{g/dl}$). However, recent studies using more sensitive indicators of renal function suggest renal effects at lower levels of lead exposure.
- d) Lead exposure is associated with a small increase in blood pressure. The likely order of magnitude is that for any two-fold increase in PbB level (e.g., from 0.8 to 1.6 $\mu\text{mol/litre}$, i.e. 16.6 to 33.3 $\mu\text{g/dl}$), there is a mean 1 mmHg increase in systolic blood pressure. The association with diastolic pressure is of a similar but smaller magnitude. However, there is doubt regarding whether these statistical associations are really due to an effect of lead exposure or are an artefact due to confounding factors.

- e) Some but not all epidemiological studies show a dose-dependent association of pre-term delivery and some indices of foetal growth and maturation at PbB levels of 0.72 $\mu\text{mol/litre}$ (15 $\mu\text{g/dl}$) or more.
- f) The evidence for carcinogenicity of lead and several inorganic lead compounds in humans is inadequate.
- g) Effects of lead on a number of enzyme systems and biochemical parameters have been demonstrated. The PbB levels, above which effects are demonstrable with current techniques for the parameters that may have clinical significance, are all greater than 0.96 $\mu\text{mol/litre}$ (20 $\mu\text{g/dl}$). Some effects on enzymes are demonstrable at lower PbB levels, but the clinical significance is uncertain.
- h) Population studies of children and adults who are not occupationally exposed to lead show relationships between 1 and 3 $\mu\text{g PbB/dl}$ per 1 $\mu\text{g/m}^3$ of lead in air. Estimates of the relationship between airborne lead and blood lead which include the total contribution from air via indirect consumption of dust and soil as well as direct inhalation suggest a value between 3 and 5 $\mu\text{g PbB/dl}$ per $\mu\text{g/m}^3$. Representative relationships of blood lead median level to intake of lead through various pathways for the general population are shown in Table 1.

Table 1. Representative relationships of blood lead median level to intake of lead for the general population^a

Medium	Population	
	Children	Adults
Air ^b	0.09 $\mu\text{mol Pb/litre}$ per $\mu\text{g Pb/m}^3$ (1.92 $\mu\text{g Pb/dl}$)	0.079 $\mu\text{mol Pb/litre}$ per $\mu\text{g Pb/m}^3$ ^c (1.64 $\mu\text{g Pb/dl}$)
Water		0.003 $\mu\text{mol Pb/litre}$ per $\mu\text{g Pb/litre}$ (0.06 $\mu\text{g Pb/dl}$)
Food	0.01 $\mu\text{mol Pb/litre}$ per $\mu\text{g Pb/day}$ (0.16 $\mu\text{g Pb/dl}$)	0.002-0.003 $\mu\text{mol Pb/litre}$ per $\mu\text{g Pb/day}$ (0.04-0.06 $\mu\text{g Pb/dl}$)
Dust ^b	0.09 $\mu\text{mol Pb/litre}$ per 1000 $\mu\text{g Pb/g}$ (1.8 $\mu\text{g Pb/dl}$)	
Soil ^b	0.11 $\mu\text{mol Pb/litre}$ per 1000 $\mu\text{g Pb/g}$ (2.2 $\mu\text{g Pb/dl}$)	

^a These data are provided for illustrative purposes only recognizing that the relationships are curvilinear in nature and are broad guidelines which will not apply at lower or higher levels of exposure.

^b A value between 0.144 to 0.24 $\mu\text{mol Pb/litre}$ or 3-5 $\mu\text{g Pb/dl}$ per $\mu\text{g/m}^3$ is obtained when one considers indirect contribution through deposition on soil/dust.

^c The air to blood lead relationship in occupational settings is best described by a curvilinear relationship having slopes between 0.02 and 0.08 $\mu\text{g/m}^3$ air. The slope is variable but lower than that found for humans in the general environment, which is between 1.6 and 1.9 $\mu\text{g/m}^3$.

ENVIRONMENTAL EFFECTS OF LEAD

General

In aquatic and aquatic/terrestrial model ecosystems, uptake by primary producers and consumers seems to be determined by the bioavailability of the lead. Bioavailability is generally much lower whenever organic material, sediment or mineral particles (e.g. clay) are present. In many organisms, it is unclear whether lead is absorbed onto the organism or actually taken up. Consumers take up lead from their contaminated food, often to high concentrations but without biomagnification.

In general, inorganic lead compounds are of lower toxicity to microorganisms than are trialkyl- or tetraalkyllead compounds. Tetraalkyllead becomes toxic by decomposition into the ionic trialkyllead. One of the most important factors, which influence the aquatic toxicity of lead, is the free ionic concentration, which affects the availability of lead for organisms. The toxicity of inorganic lead salts is strongly dependent on environmental conditions such as water hardness, pH, and salinity. There is evidence that tolerant strains of microorganisms exist and that tolerance may develop in others.

Uptake by and toxicity to aquatic organisms

The uptake and accumulation of lead by aquatic organisms from water and sediment are influenced by various environmental factors such as temperature, salinity, and pH, as well as humic and alginic acid content. In contaminated aquatic systems, almost all of the lead is tightly bound to sediment. Only a minor fraction is dissolved in the water, even in the interstitial water. The lead uptake by fish reaches equilibrium only after a number of weeks of exposure. Lead is accumulated mostly in gill, liver, kidney, and bone. Fish eggs show increasing lead levels with increased exposure concentration, and there are indications that lead is present on the egg surface and rapidly eliminated in the embryo. In contrast to inorganic lead compounds, tetraalkyllead is rapidly taken up by fish and rapidly eliminated after the end of the exposure.

There is little evidence for effects of lead on aquatic plants at concentrations below 1 to 15 mg/litre. Many studies of aquatic plants have been made in sediment-free systems. However, the addition of uncontaminated sediment reduces the toxicity of lead to aquatic plants by reducing its availability.

The results of experiments on the toxicity of lead salts to aquatic invertebrates are difficult to interpret due to the variations in experimental conditions and the lack of a standardized method for determining lead concentrations in water. In most studies, concentrations of lead in water are nominal; the contribution to toxicity of factors such as pH, water hardness, anions, and complexing agents cannot be fully evaluated. In communities, some populations of organisms are more sensitive than others, and community structure may be adversely affected by lead contamination. However, populations from polluted areas can show more tolerance to lead than those from

non-polluted areas. In other organisms, adaptation to hypoxic conditions can be hindered by high lead concentrations.

The toxicity of lead- contaminated water to fish varies considerably, depending on the availability and uptake of the lead ion. Factors affecting this availability are water hardness (prevalence of divalent anions), pH, salinity, and organic matter. Uptake is affected by the presence of other cations and the oxygen content of the water. Organic lead is taken up more readily than inorganic lead. The 96-h LC₅₀ (lethal concentration that kills 50% of the population) for inorganic lead in sensitive species can be as low as 1 mg dissolved lead/litre; nominal concentrations being up to 100 times higher in hard water. The few data available suggest that the toxicity of organic lead may be 10 to 100 times higher than that of inorganic lead. Long-term exposure of adult fish to inorganic lead induces sub lethal effects on morphology, amino levulinic acid dehydratase (delta-ALAD) and other enzyme activities, and avoidance behaviour at available lead concentrations of 10-100 mg/litre. Juvenile stages are generally more sensitive than adults, but eggs are often less sensitive because lead is adsorbed onto the egg surface and excluded from the embryo.

There is evidence that frog and toad eggs sensitive to nominal lead concentrations of less than 1 mg/litre in standing water and 0.04 mg/litre in flow-through systems; arrested development and delayed hatching have been observed. For adult frogs, there are no significant an effect below 5 mg/litre in aqueous solution, but lead in the diet at 10 mg/kg food has some biochemical effects.

Uptake by and toxicity to terrestrial organisms

In bacteria, the majority of lead is associated with the cell wall. A similar phenomenon is also noted in higher plants. Some lead that passes into the plant root cell can be combined with new cell wall material and subsequently removed from the cytoplasm to the cell wall. Of the lead remaining in the root cell, there is evidence of very little translocation to other parts of the plant because the concentration of lead in shoot and leaf tissue is usually much lower than in root. Foliar uptake of lead occurs, but only to a very limited extent. In animals, there is a positive correlation between tissue and dietary lead concentrations, although tissue concentrations are almost always lower. The distribution of lead within animals is closely associated with calcium metabolism. The tetravalent organic form of lead is general more toxic than the divalent, inorganic form, and its distribution in organisms may not specifically follow calcium metabolism.

The tendency of inorganic lead to form highly insoluble salts and complexes with various anions, together with its tight binding to soils, drastically reduces its availability to terrestrial plants via the roots. Translocation of the ion in the plants is limited and most bound lead stays at root or leaf surfaces. As a result, in most experimental studies on lead toxicity, high lead concentrations in the range of 100 to 1000 mg/kg soil are needed to cause visible toxic effects on photosynthesis, growth, or other parameters. Thus, lead is only likely to effect plants at sites of very high environmental concentrations.

Ingestion of lead-contaminated bacteria and fungi by nematodes leads to impaired reproduction. Woodlice seem unusually tolerant to lead, since prolonged exposure to soil or grass litter containing externally added lead salts had no effect. Caterpillars maintained on a diet containing lead salts show symptoms of toxicity leading to impaired development and reproduction. The information is too meagre to quantify the risks to invertebrates during decomposition of lead-contaminated litter.

Lead salts are only toxic to birds at a high dietary dosage (100mg/kg or more). Almost all of the experimental work is on chicken and other gallinaceous birds. Exposure of quail from hatching and up to reproductive age resulted in effects on egg production at dietary lead levels of 10 mg/kg. Although a variety of effects at high dosage have been reported, most can be explained as a primary effect on food consumption. Diarrhoea and lack of appetite, leading to anorexia and weight loss, are the primary effects of lead salts. Since there is no experimental evidence to assess effects on other bird species, it is necessary to assume a comparable sensitivity. Under this assumption it is highly improbable that environmental exposure would cause adverse effects in birds. There is little information on the effects of organolead compounds. Trialkyllead compounds produced effects on starlings dosed at 0.2 mg/day; 2 mg/day was invariably fatal.

Accumulation and effects in the field

Organisms have been found to incorporate lead from the environment, generally in proportion to the degree of contamination. Lead deposition in a region depends on the air concentrations of the metal, which decrease with distance from the source. In shellfish, lead concentrations are higher in the calcium-rich shell than in the soft tissue; they relate to the concentrations in sediment. Lead concentrations in some marine fish are higher in gills and skin than in other tissues; but this may be largely due to adsorption. Liver levels increase significantly with age. In dolphins, lead is transferred from mothers to offspring during foetal development and lactation. This might be related to the calcium metabolism.

In highways and urban areas, lead concentrations are highest in soils and organisms close to roads where traffic density is high. The lead measured is inorganic and derives most exclusively from alkyllead compounds added to petrol. The lead in the soil and in vegetation decreases exponentially with the distance from the road. Lead is also found in the sediments of streams in the vicinity of highways. Lead contamination increases lead levels in plants and animals in areas close to roads. These levels are positively correlated with traffic volume and proximity of roads. Most lead deposited is found within 500 m of the road and within the upper few centimetres of soil. It can be assumed that lead levels in soil and biota are not influenced by traffic at distances from roads greater than this.

No effect on the reproduction of birds nesting near highways has been observed. Toxic effects have been observed in pigeons in urban areas, the kidneys being most frequently affected. A recurring incident of massive bird kills in estuaries near to industrial plants manufacturing leaded "antiknock" compounds has been reported. The total lead content of the livers was sufficiently high to cause mortalities: lead was mostly present in alkyl form.

CONCLUSIONS

The health effects of lead in the general population have been subject to considerable debate and controversy. Of most concern are a series of population studies of lead measures of childhood intelligence. For adults, there is weak evidence of a possible effect on blood pressure. Although the causal nature of these associations is not without restrictions, the overall consistency and coherence of epidemiological findings with toxicological evidence in laboratory animals suggests a small adverse effect on both IQ in children and blood pressure in adults. Therefore, it has been recommended by WHO to phase-out any remaining uses of lead additives in motor fuels and reduce lead exposure due to other sources of lead (WHO 1995). The WHO air quality guideline for lead is $0.5 \mu\text{g}/\text{m}^3$; because of the other pathways of exposure the air quality guideline for lead should be accompanied by additional preventive measures (WHO 2000). To prevent further increase of lead in soils and sediments and consequent increases in the exposure of future generations, air lead levels should be kept as low as possible.

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