

# LEAD EXPOSURE TO HUMANS AND OTHER ORGANISMS

Lead can reach the biosphere, including humans, by a number of routes. The main exposure routes to humans are:

- **Food** – This is a major source of lead intake for the adult population. Produce can be contaminated from airborne deposition and lead-rich soil, though this can be reduced with careful washing. Previously, leaded solders in cans and wine bottle capsules contributed to lead intake in the general population, but these have been phased out. In the home, the use of lead crystal or ceramic tableware glazed with leaded glazes can cause a small contribution, but such articles manufactured in the Western World are tested to ensure that leaching is acceptably low.
- **Water** – Old lead pipes, which are still in place in many dwellings, can slowly dissolve in some soft and acidic waters. Improved water treatment to reduce plumbosolvency, reduces the lead content of water to acceptable levels in the majority of dwellings. However, the problem is not eliminated without replacement of lead pipes within the home, which incurs expense to the homeowner. A WHO guideline of 10µg/l for drinking water is complied with in most cases.
- **Air** – Direct absorption by inhalation is a minor exposure route for most people, though it can be significant to individuals occupationally exposed. Airborne lead-containing particles fall to earth, most within a short distance of source, and add to lead contents of dusts, soils and food. A weak link between levels of lead in air and exposed populations has been established. The WHO recommended an air quality standard in 1987 of 0.5-1.0µg/m<sup>3</sup>. The EU lead in air standard is currently 0.5µg/m<sup>3</sup> and the UK has set an air quality target of 0.25µg/m<sup>3</sup>. Air lead levels are falling in Western Europe, as leaded petrol is being phased out. Elevated levels can be found in some industrial areas.
- **Soil and dust** – Ingestion of soil and house dust is a major pathway for the exposure of young children to lead, due to “hand-to-mouth” activity. The

## LEAD: THE FACTS

major source of lead in house dust in older properties is leaded paint (now phased out, but still *in situ* in many dwellings). This is the major source of lead exposure to children living in older dwellings in the USA. Other contributions to dust are from airborne emissions, particularly in countries where leaded petrol is still used. Lead contents in house dusts in urban areas of the UK are declining. Lead contents of soils cannot be expected to decline for many years, as lead has a low mobility in soil.

Lead exposure levels in the general population have markedly declined over the past 30 or so years and by the 1990s average national levels for blood lead in the EU were mostly well below 10µg/dl.

### TRENDS IN LEVELS OF LEAD EXPOSURE IN HUMANS

Levels of lead exposure in the general population have fallen in the USA and Western Europe, with average national values for blood lead in the EU in the 1990s mostly well below 10µg/dl. Decreased exposure from several sources has brought this about, though it is believed that the reduction of leaded petrol is a major contributory factor. Other measures have included: improved water treatments reducing plumbosolvency; phasing out of leaded solders in food cans; and phasing out of the use of leaded paints. Improved industrial practices have resulted in lower emissions. A small proportion of individuals continue to receive doses of lead which are deemed unhealthy, particularly in the developing World and in Eastern Europe. The most highly exposed individuals tend to be the more deprived members of society. Occupational exposure has also decreased dramatically in the western world with improved technology, hygiene and management practices.

### EFFECTS OF LEAD EXPOSURE ON HUMAN HEALTH

A small number of adults occupationally exposed to lead have in the past shown increased risk of kidney damage, nerve damage, infertility and, possibly, a small increase in blood pressure and the risk of contracting certain cancers at high levels of exposure. However, today such effects are rarely observed. The greatest concern for the general population is that lower levels of lead exposure, which some of the general population may receive, appear to cause a small decrease in the intellectual development of young children. Children are more vulnerable because their nervous system is developing; they absorb more lead than adults because of behavioural and physiological differences. There is no accepted threshold level, but the body of evidence to date does not find any effect below 10µg/dl blood lead. Individuals whose diet is lacking in iron or calcium absorb more lead than those who are well nourished.

## ECOTOXICITY

Lead can have adverse effects on living organisms. High doses can interfere with some biochemical processes required for normal functioning. Most lead compounds have low solubilities in water and are not readily absorbed by most living organisms. Soluble compounds of lead can readily be taken in, and have been studied most widely (particularly for aquatic organisms). However, some organisms (such as molluscs) can absorb solid lead compounds from sediments, and there is little data available on the toxicity of this. Tetraethyl lead is much more toxic than inorganic lead compounds, but it breaks down quickly in the environment. Some aquatic species bioaccumulate lead, but there is no evidence for biomagnification at higher levels in food chains. Lead in soil generally does not have great effects on plants or earthworms except at highly elevated concentrations; its potency varies with soil type. In general, the bioavailability and toxicity of lead compounds are greater in acidic conditions, and less in alkaline or saline conditions.

The major impact of lead on wildlife, particularly waterfowl, results from the ingestion of lead shot (from ammunition or fishing weights). This can cause acute lead poisoning, sometimes fatal. For this reason, the use of lead shot for small fishing weights, and in some cases, in ammunition, is restricted in an increasing number of countries.

## POLICY APPROACHES

A strategy based on risk assessment is generally used to predict the potential harm to target populations and to assist the regulatory process. This requires knowledge of the nature of the hazard (i.e. the lead compound), the exposure pathways to the target population, and the dose-response relationship. Sources of uncertainty in these predictions should be clearly stated. The precautionary principle can be used when there is lack of data on risk, and the potential for harm exists.

## **7.1 PATHWAYS TO THE BIOSPHERE AND HUMANS**

### **7.1.1 LEAD IN AIR**

Natural and anthropogenic sources of lead in the atmosphere and their dispersion have been discussed in Chapter 6. Direct inhalation of lead-bearing particles in air is one route of exposure for both humans and other animals. For most people, this is a minor exposure route, the majority of lead being taken in by ingestion (described later). However, leaded air particles fall to earth and contribute to lead levels in dusts, which can contaminate foods, hands, etc and thus be ingested. People exposed to lead fume at work can absorb significant amounts by inhalation if protective equipment is not worn and good working practices not followed.

The World Health Organisation set an air quality guideline for lead of between 0.5 and 1.0 $\mu\text{g}/\text{m}^3$  as an annual average (WHO, 1987), though a WHO working group recently recommended revising this guideline to 0.5 $\mu\text{g}/\text{m}^3$  (WHO, in press, cited in DETR, 1998). The EU lead in air standard is currently 0.5 $\mu\text{g}/\text{m}^3$ . Ambient lead levels in air peaked in the early to mid 1980s in Britain. Annual average concentrations of airborne lead at the kerbside of a busy London road were around 1.4 $\mu\text{g}/\text{m}^3$ , and in 1985 were still higher in Manchester (2.0 $\mu\text{g}/\text{m}^3$ ); general urban levels of lead were in the range of 0.2-1.0 $\mu\text{g}/\text{m}^3$ . Since then, lead levels in urban areas have significantly declined, as the use of leaded petrol has been phased out. In the UK in 1995-6, the urban and kerbside lead levels measured were not above 0.2 $\mu\text{g}/\text{m}^3$ , well within the WHO recommended levels. It is not expected that urban areas will now have lead levels above the lower WHO limit of 0.5 $\mu\text{g}/\text{m}^3$ , though two industrial sites still exceeded this level. The upper WHO limit of 1.0 $\mu\text{g}/\text{m}^3$  was not exceeded at any of the 24 lead monitoring sites (chosen to represent urban, urban kerbside, rural and industrial locations) (UK DETR, 1998).

The relationship between lead levels in the air, and levels of lead absorbed by the body (blood lead levels) is complex, but evidence suggests that a standard for the general population should not be above 1 $\mu\text{g}/\text{m}^3$  (UK DETR, 1998). To allow for some individuals being more vulnerable, the UK Expert Panel on Air Quality Standards recommended a safety factor of 50% and a further safety factor of 50% to allow for uncertainties, resulting in a recommended value of 0.25 $\mu\text{g}/\text{m}^3$ . It is believed at this level any effects on health, even to vulnerable individuals, will be too small to be detectable. This recommendation has, however, been criticised by industry as not being based on sound science.

### **7.1.2 LEAD IN WATER**

Lead in drinking water is of intermediate significance as a source of lead intake, but is highly significant for both children and the foetuses of pregnant

women. Lead levels in drinking water, sampled at source, are usually below  $5\mu\text{g}/\text{l}$ . However, many old houses still have drinking water distribution pipes made of lead. This can result in tap water concentrations above the maximum safe level recommended by the WHO of  $10\mu\text{g}/\text{l}$ , and even above the EU statutory limit of  $50\mu\text{g}/\text{l}$ ; occasionally, levels of over  $100\mu\text{g}/\text{l}$  have been found. When the pipes need replacing, alternative materials are used, but it is expensive to replace all existing lead pipes within the home. The greatest problems are in areas where the water has a higher concentration of dissolved organic compounds, which increase plumbosolvency (Alloway and Ayres, 1997).

*Case study of a city with relatively high lead contents in drinking water - Glasgow, Scotland, UK*

In the UK, higher levels of lead in water are found in many areas of Scotland, where the water supply is from upland areas (and consequently, usually acidic, soft, and containing organic acids, which all aid dissolution of lead) and lead piping was commonly installed up until the 1960s. The city of Glasgow receives its water from such a source. It was reported that in 1974, the majority of water samples taken (84%) contained over  $50\mu\text{g}/\text{l}$  of lead. However, following the implementation of a programme to treat the water with lime, making it alkaline and less corrosive to lead, the proportion of water samples exceeding this level had dropped to 7% by late 1989. However, a 1981 EC study of mothers living in households in this supply area, found that over 50% of households received water supplies exceeding the WHO recommended level of  $10\mu\text{g}/\text{l}$ , and 13% exceeding the UK statutory limit of  $50\mu\text{g}/\text{l}$ . (Some samples contained considerably higher lead concentrations.) An improved treatment programme, which took effect from 1989-1992, consisted of treating the water with orthophosphate, which further reduces the tendency of lead to dissolve. A study in 1993 found that over 90% of the households studied had less than  $10\mu\text{g}/\text{l}$  of lead in their water, and less than 1% had over  $50\mu\text{g}/\text{l}$  lead in the water, though again some received water with well over  $50\mu\text{g}/\text{l}$  lead.

This shows that treatment of the water, with lime or preferably orthophosphate, can reduce plumbosolvency and thus lead concentrations in water to low levels, for the majority of households. Also, lead pipes for mains water have been replaced by the water company, up to the property. However, replacement of lead piping in the home is the only way to remove the possibility of lead being dissolved in water, and this is only being done in a relatively small number of buildings, because of the cost and disruption involved.

The study also found a direct relation between lead concentrations in water and in blood of the subjects (mothers of young children) studied. However, blood lead levels had declined over these 16 years for a number of reasons, and the blood lead levels associated with particular concentrations of lead in tap water

had also declined (Glasgow 93 Lead Study, 1996). This shows that lead intake through water is a significant source of exposure in some areas, though it is one of many other sources.

### 7.1.3 LEAD IN SOIL AND DUST

As detailed in Chapter 6, soils can contain lead from many sources and this can remain for centuries. Housedusts contain lead from road dusts brought indoors on shoes, as well as sources within the home, primarily leaded paint, and a small potential contribution from some window blinds which contain lead, e.g. some vinyl types.

Plants can take up that fraction of lead which is present in the soil in a soluble form and this will add to their lead content. However, there is general agreement that only a small proportion of the total soil lead content is available for uptake by plants, that much of this is then accumulated in root cell walls, and that little is translocated to the plant shoot (Davies and Thornton, 1989). Animals which eat the plants will ingest their lead content. This can be increased by particles of soil attached to the plants and lead dusts which fall on the plants from the air. Some animals, particularly grazing animals, ingest large quantities of soil - up to 20% of the dry matter intake in cattle, and up to 30% in sheep which graze closer to the ground, although statutory limits for lead in edible animal tissues are rarely exceeded (MAFF, 1989).

Ingestion of housedust and soil, which contain small amounts of lead, is one source of lead intake in humans. Dust can contaminate hands, food, etc., but this is not considered an important exposure route for adults, except possibly for occupationally exposed workers, if hygiene practices are poor. However, this route is significant for children, who spend considerable time playing on the floor, in gardens, etc. In infants aged 1-3 years, "hand-to-mouth activity" has been shown to account for 50% of their total lead intake (Davies et al 1990). Furthermore, some individuals, particularly very young children, have a pica (a habit of eating non-food substances) for leaded paint or soil, and exposure in these individuals will be higher.

A number of countries have set standards, defining maximum acceptable levels of lead in soil. These are aimed at the protection of human health. In the USA there is also a standard for lead levels in housedust. Different countries have taken varying approaches to the setting of permitted levels.

A number of studies have attempted to find a link between lead levels in soils, and exposure to humans. In some cases, the population living adjacent to industrial areas show elevated levels of lead exposure, believed to be caused by the higher level of lead in the soil (following a study of an old lead mining and smelting town, Leadville, in Colorado, USA (Cook et al, 1993, cited in INSERM, 1999), and Broken Hill, a major lead mining area in Australia (Gulson et al,

1994). However, this is not always the case: a survey in the UK identified a village with very high lead concentrations in soils (ranging from several hundred mg/kg to 3% lead in local gardens) arising from historical mining and smelting activities (Colbourne and Thornton, 1978), but where the local population did not appear to be significantly exposed. The differences are believed to be largely due to different chemical and mineral forms of the lead: analysis of the soil dusts in the UK site found much of the lead appeared to be in a relatively insoluble type of lead phosphate (pyromorphite) which is not readily absorbed by living organisms (Cotter-Howells and Thornton, 1991). Other differences could include climate, lifestyle, and nutritional status. In Northern Europe, children spend less time outdoors, and thus exposed to soil, than in other areas such as Australia.

The Society for Environmental Geochemistry and Health has proposed a method of calculating maximum permissible soil lead content needed to protect populations from exceeding a given blood lead concentration. Typical values given are presented below (from Wixson and Davies, 1994, cited in INSERM, 1999).

Guidelines for acceptable concentrations of lead in soils in the UK, Canada and the Netherlands are given below.

**TABLE 7.1** Acceptable Lead Concentration in Soils (ppm, or  $\mu\text{g/g}$ )

Blood lead concentration not to be exceeded ( $\mu\text{g/dl}$ )	Percentage of the population to protect		
	95%	98%	99%
10	880	500	300
15	2300	1860	1400
20	3750	3000	2600

*Note:* these concentrations are based on a fairly low lead bioavailability, and other sources of lead exposure in the population giving a blood lead concentration of  $4\mu\text{g/dl}$ . Where these assumptions are not valid, the calculation must be modified, and the permitted soil lead levels will be different.

## UK

UK Department of the Environment ICRCCL threshold trigger concentrations (DoE, 1987), below which there is no cause for concern, and above which professional judgement may require more detailed investigation and/or remediation. These are “more pragmatic, and based mainly on effects to human health” (Alloway and Ayres, 1997).

Parks, playing fields, open space	2000 $\mu\text{g/g}$
Gardens, allotments	500 $\mu\text{g/g}$

## Canada

Selected values from the Canadian interim environmental quality criteria for soil (Canadian Council of Ministers of the Environment, 1992).

“Normal” background	25µg/g
Agricultural	375µg/g
Residential	500µg/g
Industrial	1000µg/g

## Netherlands

The standards used in the Netherlands are based largely on the concept of multi-functionality, including ecological function. These standards comprise target values for soils which represent the final environmental quality goals for the Netherlands. Guide values and quality standards used in the Netherlands for assessing soil and water contamination by heavy metals (Netherlands Ministry of Housing, Physical Planning and the Environment, 1991; Moen et al, 1986).

Soil target value	85µg/g
<i>Surface waters</i>	
total content - target value	4µg/l
total content - limit value	25µg/l
dissolved content - target value	0.2µg/l
dissolved content - limit value	1.3µg/l

It should be noted that the Netherlands also uses a series of intervention values at which the authorities are required to investigate the level of contamination and decide on what action, if any, is required. The intervention value for soil in the Netherlands is 530µg/g compared to the target value of 85µg/g.

### *Groundwater*

dissolved content - target value	15µg/l
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(from 1991 Environment Quality Standards for soils and waters)

## USA

### *Lead in soils*

Soil-lead hazard > 2000µg/g in bare residential soil. Remedial action necessary (such as turfing, removing top 15cm and replacing with uncontaminated material, covering with gravel). Where there are young children or pregnant women resident, placing of washable doormats at house entrance, and more frequent washing of hands and toys are recommended.

Soil-lead level of concern > 400µg/g. This is a level for guidance only, suggesting that there is at least a small risk to young children living at the property, and some less costly action should be taken. No action is required by law.

*Lead in dusts*

Dust lead hazard in residential dwelling equals or exceeds  $50\mu\text{g}/\text{ft}^2$  on uncarpeted floor, or  $250\mu\text{g}/\text{ft}^2$  on interior window sills.

*Lead-based paint*

Lead-based paint hazard: requires action when property has lead-based paint in poor condition (over  $10\text{ ft}^2$  of deteriorated paint on exterior components, over  $2\text{ ft}^2$  of deteriorated paint on interior components with large surface areas, or over 10% of the total surface area of interior or exterior components with small surface areas) (U.S. Environmental Protection Agency, 1998).

**7.1.4 LEAD IN FOOD**

Ingestion of traces of lead with food is the main source of lead intake to the general adult population. Lead is ubiquitous in the environment, and all foodstuffs contain small amounts of the metal. Fresh food may be contaminated by small amounts of lead (as well as many other undesirable substances) from airborne fallout, particularly noted for foods such as lettuce, parsley and mint, which have a very large surface area compared to their mass (Sherlock, 1987); vegetables grown in soils with high lead contents can also contain traces of soil. In both these cases, careful washing will reduce the amount of lead consumed in vegetables by as much as 70 per cent (Thornton and Jones, 1984).

Lead-containing solders used in food cans could cause lead contamination, particularly of foods containing acidic fruits, tomatoes or similar, until their use was prohibited in the early 1980s. A study of lead contents of foods in the UK in 1984 found canned foods, including fruit and food in tomato sauce, often contained elevated lead contents. However, this use of leaded solder in food cans was starting to decline at that time, as manufacturers were switching to different can designs, and no longer presents a problem for food canned in the West. Offal, such as animal liver and kidney also sometimes contain elevated levels of lead, because lead taken in by livestock tends to be stored in the liver and kidneys (as well as the bones), rather than the muscle (Sherlock, 1987). Cooking food in lead-contaminated water has also been shown to result in increased levels of lead in the food (Sherlock, 1987).

Beverages consumed from or stored in lead crystal containers, or ceramic vessels glazed with leaded glazes, are another potential source of lead intake. However, products made by manufacturers in most countries, including Europe and North America, must comply with standards which ensure that lead leaching rates are acceptably low. The tests in the UK involve adding a solution of acetic acid (4% strength, which is approaching that of vinegar), which is very potent at dissolving lead, at room temperature ( $22^\circ\text{C}$ ) for 24 hours. The acid solution is then analysed for its lead content. However, the USFDA (1997) recommend that pregnant women should avoid drinking hot tea, coffee, or other hot acidic

beverages from glazed cups and pottery as high temperature can increase dissolution of lead. Storage of acidic fruits or beverages in such vessels over a longer period of time could lead to increased lead content.

### 7.1.5 ESTIMATES OF TOTAL LEAD ABSORBED BY HUMANS (ADULTS AND CHILDREN) FROM AIR, DUST, FOOD AND WATER

The table presented below is adapted from the WHO (1987) report.

**TABLE 7.2** Estimated Daily Intake of Lead

Mean Air Concentration (µg/m <sup>3</sup> )	Dust Intake (mg/day)	Lead Uptake				
		Air	Dust	Food	Water	Total Absorbed (µg/day)
<b>ADULTS</b>						
0.3	non-smoker	2.4	-	10	2	14.4
0.5	non-smoker	4.0	-	10	2	16.0
1.0	non-smoker	8.0	-	10	2	20.0
2.0	non-smoker	16.0	-	10	2	28.0
<b>CHILDREN (1 - 5 years)</b>						
0.3	-	0.6	-	25	5	30.6
0.5	-	1.0	-	25	5	31.0
1.0	-	2.0	-	25	5	32.0
2.0	-	4.0	-	25	5	34.0
1.0	25	2.0	12.5	25	5	44.3
1.0	50	2.0	25.0	25	5	57.0
1.0	100	2.0	50.0	25	5	82.0
1.0	200	2.0	1.0	2	5	132.0
		0.0		5		

*Assumptions on which tabulated data is based:*

**Air:** Respiratory volume in adults is 20m<sup>3</sup>/day, and in children 5m<sup>3</sup>/day, and the respiratory absorption is 40%.

**Dust:** Dust concentration of lead is 1000µg/g, and absorption 50%.

**Food:** Intake of lead by adults is 100µg/day with 10% absorption, and 50µg/day for children.

**Water:** A lead concentration of 20µg/l, with adult consumption of 1 litre per day, 10% absorption; for children 0.5 litre consumption per day, 50% absorption.

## 7.2 ENVIRONMENTAL EFFECTS AND ECOTOXICITY

Lead is not an essential trace element for either plant or animal nutrition. However, lead and its compounds, if present in sufficient amounts, are responsible for toxic effects to populations of both terrestrial and aquatic ecosystems. The form which the metal is in is a crucial factor in determining its mobility, bioavailability and toxicity (Chimie et Ecologie, 1998).

Measured effects of lead on living organisms include those related to survival, growth, learning, reproduction, development, behaviour and metabolism. Lead has no known essential biological functions and all lead compounds are potentially harmful or toxic, especially tetraethyl lead. Lead functions as a cumulative poison (Environmental Contaminants Encyclopaedia, Lead Entry, 1997). However, many effects are reversible once exposure levels fall.

Populations can be exposed to lead in the following media:

- water soluble compounds of lead are readily absorbed into living cells. Compounds which are insoluble, in suspension or in sediments, can be ingested or filtered by benthic organisms (those which live on the floor of the watercourse);
- soil soluble compounds are the most bioavailable and can be taken up by organisms. However, most compounds of lead have very low solubility, except in very acid or very alkaline conditions;
- air plants, insects, birds and other organisms can be exposed to vapours, aerosols or particles of lead-rich material (Chimie et Ecologie, 1998).

Exposure can be a single dose, such as in the rare case of an accident or production incident, or continuing over a long period, as is the result of routine industrial and natural emissions. The potential toxic effects depend largely upon the form of the lead, the “target” population, and the conditions of exposure. The presence of other pollutants or naturally occurring constituents of the environment can modify the effects of the lead (Chimie et Ecologie, 1998).

### Results of field studies

There are not many studies on actual ecosystems, and the results of these are difficult to interpret, because

- lead and its compounds are only one of a number of substances present in the environment, and it is difficult to establish which effects are due to which pollutant;
- though the total lead content can be determined, the concentrations of different chemical forms of lead, and thus its actual available dose, are often not known (Chimie et Ecologie, 1998).

Under normal conditions, it does not appear that lead has a marked effect on ecosystems, because of its generally low bioavailability. In waters, lethal poisoning of fish has not been observed, even when concentrations of lead exceed 3,000mg/kg in aquatic bryophytes (moss-like plants which readily absorb pollutants from their surroundings and are used as a measure of long-term pollution) (Chimie et Ecologie, 1998). In soils, lead generally has a low bioavailability and total concentrations reaching or exceeding 1,000mg/kg do not necessarily cause significant effects on vegetation. The damage to some forests near roads with heavy traffic is generally considered the result of atmospheric pollutants, (such as SO<sub>2</sub>, NO<sub>x</sub> and ozone) rather than lead (Académie des Sciences, 1998).

The impact of lead is more clearly established in the case of bird mortality due to exposure to lead shot from fishing or shooting (Académie des Sciences, 1998). This has been widely reported, particularly in the USA (for example, US Department of Interior, Fish and Wildlife Service, 1986, cited in Chimie et Ecologie, 1998) and Nordic countries. Birds which ingest lead show anorexia and ataxia, weakness and loss of condition. Egg production, fertility and hatchability decrease and mortality may be high; high doses cause coma and death (Booth et al, 1982 and Clarke et al, 1981, cited in Irwin et al, 1997). At least 2% of waterfowl in North America are estimated to die as a result of ingesting lead shot (Canadian Environmental Protection Branch, from web page, 1999). Black ducks in urban areas in Nova Scotia (Canada) have been found to have high concentrations of lead in their blood, though many had not ingested lead shot. The source of exposure was believed to be from run-off from road dusts, contaminated with leaded petrol derivatives, being deposited and accumulated in the sediments of ponds. As lead is relatively immobile in sediments, this will continue to pose a hazard to the ducks unless the contaminated sediments are removed (Canadian Environmental Protection Branch, from webpage, 1999). Similarly, in Europe, the deaths of seagulls which have been noted, could be caused by lead alkyl compounds from petrol (Académie des Sciences, 1998).

### **Laboratory studies**

There are many results from experimental studies, though most concentrate on soluble inorganic compounds of lead. The insoluble forms which can be ingested or filtered are rarely studied. Most studies are on aquatic ecosystems, rather than terrestrial ones. There is much more data on lethal or severe effects following high exposure; long term low exposure is more rarely studied, even though this is more likely to occur in practice (Chimie et Ecologie, 1998; Académie des Sciences, 1998).

### **Bioaccumulation, biomagnification, biomethylation**

Little bioaccumulation of inorganic bioavailable forms of lead has been found in plants and earthworms. In contrast, there is evidence of significant

bioaccumulation in phytoplankton, filter-molluscs and bryophytes (moss-like plants) and, to a smaller degree, in fish and micro-crustaceans. Lead-alkyl compounds appear to be the most easily bioaccumulated, especially in algae, molluscs and fish. There is no evidence of biomagnification (increasing concentrations found in organisms at higher levels in the food chains) of lead. The possibility of conversion of inorganic lead compounds to organic, methylated compounds (which could potentially be much more toxic) by biota, has been demonstrated in sediments, but the reaction is extremely slow and only occurs to a small degree (Académie des Sciences, 1998).

Inorganic lead compounds have greater solubility and thus potential toxicity in more acidic conditions, and are less toxic in more alkaline or more saline environments (Chimie et Ecologie, 1998). Lead-alkyl compounds have important ecotoxicological effects, though these effects diminish rapidly as the compounds break down (Académie des Sciences, 1998).

A selection of results from toxicological tests are presented below.

**TABLE 7.3** Examples of Lead Toxicity Tests

Species	Effect	Inorganic dissolved lead compound	Organic lead compound (tetraethyl lead)	Concentration of lead (mg/l)
bacteria	inhibition of bioluminescence	inorganic		0.4 - 30
marine micro-organisms	inhibition of respiration		tetraethyl	0.08
daphnia (water flea)	inhibition of reproduction	inorganic		0.012
Artemia salina (a salt-water shrimp)	LD50 (96hrs)	lead nitrate		>100
Artemia salina	LD50 (96hrs)	lead nitrate	tetraethyl	0.003
molluscs	LD50 (96hrs)	inorganic compounds		10 - 2000
molluscs	LD50 (96hrs)		tetraethyl	0.1 - 1
fish	LD50 (96hrs)	ionic compounds		0.6 - 242
fish	LD50 (96hrs)		tetraethyl	0.02 - 0.03
fish	effects on juveniles and on reproduction			

*Note:* LD50 (96hrs) is the lethal dose for 50% of the specimens within the time interval of 96 hours.

(Source: Académie des Sciences, 1998)

### **Effects on plants**

Lead in soil - effect on test plant (radish): inhibition of germination or growth is observed at concentrations of 125 $\mu\text{g/g}$  in sandy soils, but no effects are observed in chalk soils up to concentrations of 200 $\mu\text{g/g}$ .

Solution of lead nitrate - disturbances in the development of the root systems are found at concentrations of 13.1mg lead/l.

Excessive concentrations of lead in plants have been found to cause a number of biochemical effects, including upon the processes of respiration, photosynthesis, transpiration and the proper functioning of dark green leaves (Kataba-Pendias and Pendias, 1984). Normal concentrations of lead generally found in plant leaves (in the range 5-10 $\mu\text{g/g}$ ) are not far below concentrations which cause toxic effects (30-300 $\mu\text{g/g}$ ). However, it must be stated that this is also true for many other metals, including copper, chromium, zinc, arsenic and cadmium (Alloway, 1995).

Elevated concentrations of lead in water are particularly toxic to many species of algae. Synergistic effects of lead and cadmium, and additive effects of lead, mercury, copper, zinc and cadmium, have been documented for aquatic biota (Irwin et al, 1997).

### **Effects on animals**

Some lead compounds are classified as ecotoxic. Like arsenic, lead exposure can lead to excess mucous formation which can coat the gills and impair respiration. In vertebrates, sub-lethal lead poisoning is characterised by neurological problems, kidney dysfunction, enzyme inhibition and anaemia. Animal studies indicate that high levels of lead exposure interfere with resistance to infectious disease (Gainer et al, 1974, cited in Irwin et al, 1997). Both water hardness and acidity are important factors affecting lead toxicity.

It is doubtful whether the term "toxic dose" has any real meaning when applied to lead, as it is affected by many factors. These include: environment, nutrition, disease and, most importantly, age, with young animals being considerably more sensitive than adults. Lead is cumulative and ubiquitous, and all living creatures are continually absorbing it - the toxic dose is only the amount necessary to bridge the gap between normal intake and a potentially dangerous level. Three or four lead shot will kill a duck, 10 a goose (Clarke et al, 1981). Lead shot poisoning of waterfowl has been widely publicised, but can also occur in bald eagles and other species of fish and wildlife (Irwin et al, 1997).

The acute lethal dose for lead (LD50), tested by injection into rats, was found to be 70mg/kg bodyweight. Lead administered in this way is less toxic than many other elements, including silver, gold, platinum and tin. When taken in by ingestion, much of the lead is not absorbed but passes out in the faeces (Alloway and Ayres, 1997).

## **7.3 HEALTH EFFECTS ON HUMANS**

### **7.3.1 HISTORICAL ACCOUNTS OF PLUMBISM**

It has been known for many centuries that excessive intake of lead causes a number of adverse health effects, and very high doses are fatal. Even the Romans were familiar with some of the toxic effects of lead. Several writers of the time (including Pliny and Vitruvius) refer to ill effects that craftsmen could suffer as a result of working with lead, though it must be stated that many of the miners and artisans would have been exposed to very high doses of lead. Even among the general population, it is believed that many, particularly in the wealthier classes, were exposed to lead from its use in piping, and much more seriously, in kitchenware in the preparation and storage of acidic dishes. There are accounts of epidemics of colic, occasionally paralysis, and general poor constitution, which are consistent with plumbism, though of course could have many other causes. It has even been suggested that chronic lead poisoning in the nobility, was a contributory factor (though it must be said, one of many) in the downfall of the Roman Empire, as severe lead poisoning would have affected the judgement of the rulers, and the failure to produce heirs capable of government could have contributed to political instability (Nriagu, 1983, citing his own work, and that of Kobert, 1909, and Gilfillan, 1965).

Another example was the “Devonshire colic” in England, diagnosed as lead poisoning by Baker, in 1767 resulting from the addition of lead acetate to locally produced cider.

### **7.3.2 ABSORPTION OF LEAD**

Adults absorb only about 5-15% of the lead they ingest, and generally retain less than 5% of what is absorbed (American Council of Science and Health, 1997). However, absorption of inhaled lead is much higher, about 50%. Children absorb much more of what they ingest (approximately 30-40%, can be up to 50%), because of physiological and metabolic differences.

Absorption by individuals is also influenced by a number of factors. Besides the chemical form which the lead is in (which has already been discussed in Chapter 6), the nutritional status of the individual is important, as people whose diet is low in iron or calcium absorb more lead. Lead absorption is also higher if ingested in water or other beverages than in food. Ingestion after fasting is also increased compared with ingestion after or with food.

### **7.3.3 FATE OF LEAD IN THE HUMAN BODY**

Once lead has been absorbed into the bloodstream, it is distributed between the bones and teeth, the soft tissues (kidneys, brain, liver) and the blood, and in part

excreted in the urine and in bile. Lead can cross the placenta and be transmitted in the breast milk.

The body does not distinguish between lead and calcium, and so the majority of lead which is absorbed is stored in the bones and teeth. In children, about 70% of lead is distributed in this way; in adults up to 95%. However, physiological stress, such as pregnancy, chronic disease or demineralisation of the skeleton after the menopause, can cause small amounts of this stored lead to be mobilised and blood lead levels to increase. However, the clinical significance of this mobilisation is still uncertain.

### **7.3.4 MEASURES OF LEAD EXPOSURE**

Blood lead levels are the usual measure of lead exposure and indicate recent exposure to lead. Blood plasma may reflect the “active” fraction of lead in blood, which can determine the accumulation of lead in the organs. However, blood lead concentrations do not give an indication of previous exposure to lead, or the amount of lead stored in the body. (After a single exposure, blood lead levels fall to about half of their initial value after about 20 days depending on length and intensity of the exposure, age of the exposed subject, etc.)

Children’s teeth give an indication of cumulative lead exposure, and have been used in some studies. However, the type and part of the tooth and presence of any plaque, dental caries and fillings, all influence the measured lead concentrations. Milk teeth are usually assessed, although these are not available until the child is at least 6 years old, which is several years past the age of maximum exposure and maximum effect on the nervous system.

A more recent approach is to measure bone lead by non-invasive X-ray fluorescence (Todd and Chettle, 1994). This also gives measurements of cumulative exposure, but improved standardisation is still needed.

Workers who are occupationally exposed to lead, should be routinely monitored for blood lead concentration and possibly other measures such as haemoglobin level, d-amino laevulinic acid in urine (ALA-U), and zinc-protoporphyrin (ZPP) in the blood, to indicate previous exposure and general health (CEDAC-Plomb, 1996). Lead in urine measurements are mainly useful on a group basis, but are limited by contamination and differences in urinary output. Some such measures are required by law in many countries (ILZSG, 1996).

Hair has not been found to be a good way to measure exposure to lead, mainly because of problems of surface contamination.

### **7.3.5 HEALTH EFFECTS OF LEAD EXPOSURE**

#### **7.3.5.1 Effects of lead exposure on mental development and IQ**

It has been shown that lead can impair the mental development of young children, and even relatively low levels of exposure can cause a measurable reduction in IQ

(Pocock et al, 1994; Smith, 1998). This effect of lead is of particular concern, because evidence from prospective longitudinal studies (which study the same children over a period of several years) has shown that neurobehavioural effects, such as impaired academic performance, may persist even after blood lead levels have returned to normal. Although no threshold level for these effects has been established, the available evidence suggests that lead toxicity may occur at blood lead levels of 10-15µg/dl, or possibly less (ATSDR 1988).

Young children are much more susceptible to damage than adults, because their nervous system is developing. Children can be exposed both before and after birth. The foetus can receive lead from the bloodstream of the mother, as lead can pass through the placenta after the 14th week of pregnancy; the blood lead level of the foetus is often similar to that of the mother (INSERM, 1999). Continued exposure of the infant can occur during lactation, as human milk can also contain lead (which follows the same route as calcium in the body). As previously stated, lead stored in the skeleton of the mother can be mobilised during pregnancy, so the exposure of the foetus reflects the mother's exposure to lead both during and before the pregnancy. Animal experiments have shown effects on the developing brains of foetuses, but it has been difficult to demonstrate similar effects in humans.

Children are likely to have higher blood lead levels than adults, because of increased absorption of lead, and behaviour which increases exposure, as outlined above. Those aged 2-3 may be most at risk from lead-contaminated soil, and Mahaffey et al (1982) reported that children in this age group had the highest blood lead concentrations. This is also the age at which pica tendencies are most prevalent.

The symptoms of chronic low level lead exposure are non-specific and include: poor concentration, lower vocabulary and grammatical-reasoning scores, poorer hand-to-eye co-ordination, insomnia, hyperactivity, increased absenteeism, and lower class standing in high school. They are rarely diagnosed and epidemiological studies, comparing similar populations with differing lead exposure levels, are necessary to determine any effects.

The results from many studies show an association between blood lead levels in early childhood and performance of young children at school. A reduction of 1-3 IQ points appears to result from an increase in blood lead concentrations from 10µg/dl to 20µg/dl. This is only a small reduction in IQ compared with the variation in IQ in the general population, but if the population is widely exposed to lead, this could have important consequences for public health (INSERM, 1999). The International Programme on Chemical Safety (WHO, 1995) reported a possible IQ deficit of 1-3 points for each 10µg/dl increment in blood lead concentration, based on populations with blood lead levels generally below 25µg/dl. At higher concentrations, effects may be greater. In the USA, 10µg/dl blood lead is the level above which further surveillance or action is deemed necessary.

### **Confounding factors**

There are many factors which can influence a child's development, including: its home environment, the amount of stimulation which it receives, the intelligence of its parents, and its nutrition. Many of these factors are influenced by socio-economic status. Exposure to lead is also influenced by socio-economic status, with children from poorer backgrounds being more likely to be exposed to lead. This is certainly the case in the USA and Europe, where the major cause of lead exposure in young children is from old leaded paint - most common in older, dilapidated houses, which are generally occupied by poorer sections of the community. There are methods of allowing for these potential sources of error, which have been included in the more recent studies; however, older studies were not fully adjusted to take account of these factors. A further factor of confusion is that hyperactive children could be more highly exposed to lead because of their behaviour (ACSH, 1997; INSERM, 1999).

### **7.3.5.2 Other Effects of Lead Exposure**

Lead exposure is associated with a range of effects on many different parts of the body. These effects vary from subtle biochemical changes to severe clinical symptoms such as lead poisoning (plumbism).

#### **Biochemical effects**

Lead has been shown to have effects on many biochemical processes. The most widely studied is the effect on the synthesis of haem (a vital constituent of red blood cells) in children and adults. Lead inhibition of haemoglobin synthesis has caused anaemia in children at blood lead levels above 25 - 40µg/dl (Fergusson, 1990, INSERM, 1999), and in adults above 50µg/dl.

Low blood lead levels are known to cause altered levels of some blood enzymes (which are essential to normal functioning of the body). In the haem synthetic pathway, the maximum blood lead levels at which a change cannot be detected are: for zinc protoporphyrin (ZPP) - 20µg/dl; coproporphyrin - 40µg/dl; urinary blood aminolaevulinic acid (ALA) - 30µg/dl; and aminolaevulinic acid dehydratase (ALAD) - 10µg/dl. These changes show that lead is having an effect on normal body functioning and the body is responding. The significance of these subtle changes is arguable, as there is little clear evidence that these are harmful in themselves. The practical significance of some of these effects is their use as measures of lead exposure.

#### **Nervous system**

Adverse effects of lead on the central nervous system have been found in individuals occupationally exposed to lead. At blood lead levels of approximately 30µg/dl, slowing of nerve conduction in peripheral nerves has been observed. Distinct symptoms of peripheral neuropathy, such as wrist drop

(weakness of the wrist and fingers) appear at higher blood lead levels of around 60µg/dl or greater. These effects are largely reversible if exposure ceases, though recovery is slow (INSERM, 1999). Encephalopathy (a severe effect on the functioning of the brain) giving rise to epileptic seizures, and coma may occur when acute lead exposure results in blood lead concentrations exceeding 80-100µg/dl (WHO, 1995).

### **Kidneys**

High blood lead levels (in excess of 60µg/dl) have been shown to cause renal dysfunction, one of the effects of acute lead poisoning in occupationally exposed individuals. However, chronic kidney effects require relatively high and prolonged exposure to lead. It is not known how reversible these effects are (INSERM, 1999). Lower blood lead levels can cause subtle effects to kidney biomarkers, and some studies suggest there could be effects on kidney function at blood lead concentrations as low as 10µg/dl (INSERM, 1999).

### **Bone function and vitamin D metabolism**

Numerous studies have found associations between blood lead levels (ranging from 12-120µg/dl) and decreased metabolism of vitamin D (which is required for bone formation). It has been suggested that low levels of lead exposure could have adverse effects on bone growth in children. There is no conclusive evidence of a threshold (a "safe" level of blood lead) for this effect. However, some other studies have reported that low to moderate lead exposure does not cause any effect on vitamin D metabolism or bone mineral content in children who are adequately nourished (American Council on Science and Health, 1997).

### **Reproductive health**

#### *Men*

High levels of lead exposure can result in decreased sperm count and mobility, and an increased number of morphologically abnormal sperm. However, these effects are unlikely to affect the general population, or occupationally exposed individuals with low or moderate blood lead levels. A range of studies on workers exposed to lead for between 6 and 10 years suggest that blood lead levels above 40µg/dl cause a reduction in the production of sperm. Exposure to levels below 40µg/dl appears to cause little or no effect (INSERM, 1999). Current data suggest that effects on fertility are most likely when blood lead levels are 50-60µg/dl or higher for prolonged periods.

#### *Women*

High levels of lead exposure resulting from occupational exposure have been known to cause serious adverse effects. Severe lead intoxication is associated with sterility, miscarriage, stillbirth and effects upon the foetus.

Evidence for the effects of low-level exposure is less clear. Data are mixed with respect to the risk of spontaneous abortion and reduced birthweight associated with maternal blood lead levels below 30µg/dl (WHO, 1995). Recent epidemiological studies have shown exposure-related perturbations in length of gestation, significantly greater risks being associated with blood lead levels of 15µg/dl or more (Murphy et al, 1990; WHO, 1995). Bellinger et al, 1991, cited in INSERM, 1999, investigated birthweights at blood levels below 15µg/dl but did not find any effect related to blood lead.

### **Cancer**

A number of studies have been performed on workers occupationally exposed to lead, often at high levels. Exposure to lead (and its inorganic compounds) gives a possible increased risk of contracting certain cancers, namely of the lungs, the stomach and perhaps also the kidneys. However, the results of these studies are not conclusive because the individuals were also exposed to other substances, such as chromium, arsenic, or certain hydrocarbons at work, and of course cigarette smoke, which are all known to cause cancers (INSERM, 1999). There are no studies on lead exposure in the general population and the occurrence of these cancers (INSERM, 1999). Animal experiments have found that long term exposure to high doses of lead acetate have caused kidney cancer in rats and mice (Kazantzis, 1990), brain cancer in rats, and lung cancer in mice. It is not known how relevant these results are to humans (ACSH, 1997), but it is considered that certain lead compounds should be regarded as potential carcinogens to humans (INSERM, 1999). The International Agency for Research on Cancer (IARC, 1987) has classified, lead and lead compounds as possibly carcinogenic to humans, based on evidence from animal experiments.

### **Blood pressure**

Animal experiments show that exposure to lead can result in increased blood pressure, but epidemiological evidence of effects on humans is not conclusive. Several studies have found a weak association between lead exposure and elevated blood pressure, though these are generally for fairly high exposures (over 45µg/dl, ACSH, 1997) which are relevant only to individuals occupationally exposed to lead. An analysis of 15 studies suggested that reduction of blood lead concentrations in men from 10µg/dl to 5µg/dl (levels commonly found in the general population) could be associated with a small reduction in blood pressure (Schwartz, 1991 and 1995, cited in INSERM, 1999). However, other studies have found only a weak association, or an association which was not statistically significant. As with studies on cancer and other potential effects of exposure to lead, confounding factors such as environment, lifestyle, and socio-economic status, obscure the results (INSERM, 1999). A dose-response relationship for lead exposure and blood pressure has not been established (ACSH, 1997).

### Thyroid gland

Lead exposure in animal experiments and cases of severe lead poisoning in humans have been reported to reduce the functioning of the thyroid gland (Sandstead et al, 1969, Sandstead, 1967, cited in INSERM, 1999). Two studies on occupationally exposed workers (some very highly exposed to lead) found an association between lead exposure and reduced function of the thyroid gland, and previous exposure appeared to be an important factor. Two other studies on exposed workers and exposed children in the USA failed to find an association. Tuppurainen et al (1988) found no correlation between blood lead levels (mean 56µg/dl) in workers in secondary lead smelters and total thyroxin, total triiodothyronine or thyroid stimulating hormone. They reported a weak negative correlation between durational exposure and total thyroxin and free thyroxin. However, Gennart et al (1992) examining a group of battery workers found no lead-related effects on the same endocrine parameters. No effects of lead on thyroid function have been found in children (ATSDR, 1998). It has been concluded that there is only an effect at high levels of lead exposure (above 60-70µg/dl) and children do not appear to be any more vulnerable than adults (INSERM, 1999). It has also been concluded by an EU scientific committee that there is no scientific evidence that lead is an endocrine disruptor.

The effects of lead on health are summarised in table 7.4 below. **This table is to be taken only as a guide.** Also, while there is general agreement about the effects, the exposure levels at which they generally occur is open to some debate, as different studies obtain different findings. Symptoms do not start abruptly at set levels, but affect different individuals over a range of exposures. Some studies may use different definitions of an ailment.

### 7.3.6 LEAD POISONING

Elevated lead exposure may affect many organs of the body, particularly the nervous system, reproductive system, the blood and the kidneys. However, confirmed cases of actual non-occupational lead poisoning in the EU are now rare. For example, only one death, of a two year old child, was ascribed to lead poisoning in England over the period 1981-1996 (Elliott et al 1999). The initial symptoms of inorganic lead poisoning are non-specific, the most important being fatigue and lassitude, anorexia, headache, joint pains, indigestion, constipation or intermittent diarrhea, and a metallic taste in the mouth. During the later stages there may be abdominal discomfort, colic, vomiting, and weakness of the muscle groups most often used. Encephalopathy may develop, with headache, confusion and epileptic seizures.

Poisoning by lead alkyls (petrol additives) causes a rapidly developing and life-threatening encephalopathy. Initial irritability, restlessness and confusion may be followed by ataxia, tremor, disorientation and coma.

**TABLE 7.4** Health Effects of Lead

	Concentration of lead in blood (in µg/dl) above which an effect may be observed
Likelihood of small impairment to mental development and probable reduction in IQ	10
elevated levels of biomarkers, indicating an effect on the production of haemoglobin	
ALAD	10
ZPP	20
ALA in urine	30
coproporphyrin	40
anaemia in children	25 - 40
anaemia in adults	50
renal dysfunction - biomarker	10
renal dysfunction - kidney damage	60
slowing of nerve conduction velocity	30
peripheral neuropathy (e.g. wrist drop)	60
male - reduced sperm count	approximately 40 - 50
female - small reduction in birth weight	may be around 20
colic	above about 80
encephalopathy	80 - 100

Prolonged high exposure to lead may cause reduced fertility, and any offspring born may suffer some damage to the nervous system.

In the human diet, a high dose of lead is needed to cause death, the lethal dose being around 10g per day, higher than silver, cadmium, chromium or zinc (lethal doses of these elements ranging from 1.3 to 9g per day). Of elements for which data are available (but excluding the extremely toxic uranium, plutonium and beryllium) only mercury has toxic effects at lower doses. Slightly higher amounts of even arsenic and cadmium are needed to give toxic effects (Bowen, 1979, cited in Alloway and Ayres, 1997).

Cases of such very high exposure are now extremely rare (in the Western World), and would result from the person being at exceptional risk, in most cases from occupational exposure.

## 7.4 EXTENT OF LEAD EXPOSURE IN THE GENERAL POPULATION

### 7.4.1 THE GENERAL POPULATION (non-occupational exposure)

#### USA

Measures to reduce exposure to lead began in the 1950s and 1960s, as the number of cases of severe lead poisoning in the cities was high. Prevention efforts included screening of children (even those not displaying symptoms), efforts to reduce exposure to leaded paint in the home, and research into the effects of low level exposure to lead. The National Health and Nutrition Examination Survey (NHANES) has performed surveys of the general population. There was a dramatic decline in the blood lead levels in pre-school children from the late 1970s to the early 1990s (Pirkle et al, 1994). The earlier survey found geometric mean blood lead levels of the order of  $15\mu\text{g}/\text{dl}$ , but the more recent survey found a geometric mean level of only  $2.7\mu\text{g}/\text{dl}$ . This reduction has benefited all groups in society, though exposure to lead varies substantially with race, income, urban status and age of dwelling in which the child lives. Low income children living in housing built before 1946 are over 30 times more likely to have elevated blood lead levels (taken as over  $10\mu\text{g}/\text{dl}$  in the USA) than middle income children living in housing built after 1973.

There are several reasons for this change:

- The use of leaded petrol began to decline in the late 1970s.
- High levels of lead in most paint used in homes was found until the 1950s. Around 1950, the US paint industry began to voluntarily phase out the use of leaded pigments, though smaller amounts of other lead additives continued to be used. The addition of lead to house-paint was banned in 1978. The number of older houses still containing leaded paints is falling as older buildings are demolished. Measures for assessing the risk of lead exposure in the home have been put in place, along with measures aimed at reducing exposure.
- The phasing out of leaded solder on food and beverage cans (resulting from different manufacturing processes for food cans), which was previously a major contributor to lead exposure in the general population.
- Lead levels have fallen in drinking water because leaded solder is now banned in household plumbing (since 1988) and public water systems are required to test for lead contamination at taps, and treat water to reduce its tendency to dissolve lead if high lead concentrations are found (since 1990).
- Ongoing screening programmes and public education are believed to have reduced exposure in the more highly exposed sections of the population.

## France

Blood lead levels have significantly declined over the period from around 1980 to 1995. A study of the adult populations of Paris, Lyon and Marseille, found a decline in blood lead levels of approximately 50% (INSERM 1999). The reduction was greater for men than for women, of the order of about 6µg/dl blood lead, though men had higher lead exposure than women. Other studies of blood lead levels of expectant mothers show a similar drop in blood lead, for example from an average of 6.1µg/dl in 1984 to a current level of 3.3µg/dl, in mothers in Brittany. Similar results have been found in Lorraine (INSERM, 1999).

The major cause of this reduction is believed to be the phasing out of leaded petrol. However, petrol is not the only source of lead exposure. The age of dwellings is another important factor, because of the presence of leaded paint in older buildings, which contributes to levels of lead in house dusts. A significant association has been found between the age of dwellings, (before or after 1945) and blood lead levels of inhabitants. Also, residents of some geographical regions have higher exposure to lead and receive above-average levels of lead in drinking water. Smoking and drinking habits were also observed to have a small effect on blood lead levels, by a number of authors. Levels of blood leads in populations living in some industrial regions are also elevated (INSERM, 1999).

## England

A survey of blood lead levels in the population in 1995 (Primatesta et al, 1998) found the following results:

“the population in general had low blood lead levels, most in the range 1-4µg/dl, for individuals exposed only to environmental lead. There were small differences in blood lead levels related to smoking, alcohol consumption, manual work/lower social class, urban residence as compared with rural. Males had higher lead exposure than females. Exposure increased with age. Whites had higher lead exposure than non-whites (all treated as one category). Age of dwelling (before/after 1945) gave a possible small increase in blood lead but, in contrast to surveys in France and the USA, this difference was not statistically significant”.

Blood lead values in the general population have fallen from around 10-16µg/dl in 1980, to around 1-4µg/dl in 1995 (Delves et al, 1996). Blood lead levels above 10µg/dl are now unusual and indicate some additional exposure source. However, some 4,400 blood lead analyses undertaken by the Medical Toxicology Unit at St Thomas' Hospital London over the period 1991-1997 found that among 547 children aged 0-4 years, 45 (8.2%) had blood lead concentrations in excess of 25µg/dl, the action level in the UK for investigation/removal of environmental sources of lead (Elliott et al 1999). It should be noted however that these tests were undertaken in cases of suspected high lead exposure and/or lead poisoning.

**Scotland**

There are no available data from similar large scale surveys. However, a study in Glasgow in 1993 of young mothers resident in areas supplied with soft water from a particular source which had caused enhanced plumbosolvency, found that the geometric mean blood lead level was 3.7µg/dl. This compared with a mean level of 11.9µg/dl in 1981. A small proportion of the mothers had blood lead levels above 10µg/dl, but none above 25µg/dl. Lead in tap water was believed to be the cause of elevated blood lead levels (over 10µg/dl) in approximately 70% of cases. However, blood lead levels of mothers exposed to tap water containing above 50µg/dl was on average 7.2µg/dl in 1993, compared with 21.6µg/dl in 1981. This clearly shows that exposure from other sources had greatly declined. Similar trends have been noted in other industrialised countries.

**Belgium**

A dramatic decline in the levels of lead in blood has been found in the urban Belgian population over the period from 1970 (14-21µg/dl) to 1988 (7-11µg/dl) (Ducoffre et al, 1990). This study covered both smoking and non-smoking males and females. The fall in blood lead corresponded with a parallel reduction in the lead content of petrol.

The WHO recommend that action be taken for individuals with blood lead levels above 25µg/dl, and the current EU standard is that no more than 2% of the population should have blood lead levels above 35µg/dl. In the US, the aim is to reduce exposure in the population to blood lead levels below 10µg/dl.

#### **7.4.2 EXPOSURE OF INDIVIDUALS OCCUPATIONALLY EXPOSED TO LEAD**

Individuals working in many diverse industries can be exposed to lead. Besides mining and metal smelting, industrial workers can be exposed during activities including lead-acid battery manufacture, scrap metal work, painting, soldering, ship repair and demolition, plumbing, manufacture of pottery etc. (UK DETR, 1998). Other activities which could cause exposure include waste disposal, particularly at incinerators, manufacture of leaded PVC or the lead-containing additives, work in steelworks, and petrol stations. Certain leisure pursuits can also result in increased lead exposure, particularly pottery making and enamelling (using leaded glazes and enamels), stained glass working, and target practice in enclosed firing ranges.

In the Western World, there are regulations in place to protect workers from excessive exposure. Many countries have laws stating maximum permitted levels of lead in air, and good hygiene and regular medical surveillance are either obligatory or recommended. Many countries also have regulations stating the

maximum concentration of lead in blood or other measures of exposure. In the EU, the maximum allowable level is currently 70µg/dl (with 80µg/dl permitted if standard measures of urine or haemoglobin, indicating lead exposure, are acceptably low), (Directive 82/605 on Risk at Work due to Lead (1982), cited in ILZSG, 1996). Some Member States have stricter regulations (50µg/dl in Denmark, Sweden, Finland and Norway). In the UK, the Control of Lead at Work Regulations, 1998, require suspension from further exposure to lead at work at a blood lead level of 60µg/dl (for young persons aged 16-17, of 50µg/dl, and for women of reproductive capacity, of 30µg/dl) (HSE, 1998). Furthermore, an action level, to prevent the employee's blood lead concentration from reaching a suspension level, has been set at 50µg/dl (for young persons at 40µg/dl and for women of reproductive capacity at 25µg/dl). For comparison, the maximum permitted blood lead concentrations in the USA is 50µg/dl; in Canada 50-80µg/dl (in different Provinces) and in Australia 50-75µg/dl (in different States) with a recommended level below 50µg/dl. Some countries specify much lower levels for women of reproductive age (20-40µg/dl) to protect any future children (Source: ILZSG, Environmental and Health Controls on Lead, November 1996). Any individuals showing excessive lead exposure (in blood or urine) are required to be moved away from areas of high lead levels in the workplace until their blood or urine lead concentrations fall to an acceptable level. These permitted levels of exposure are set by governmental Departments of Health to provide adequate worker protection; many companies aim for much lower levels of lead exposure for their workers.

There is a legal requirement for medical surveillance of the workforce in most countries and this is generally satisfactory in large companies; however, employers of smaller businesses may not be so well informed about risks and necessary procedures, and medical surveillance may not be routine (CEDAC-Plomb, 1996).

There are possibly many cases of industrial exposure in other countries, particularly in the poorer developing countries, where reasonable conditions of work are not always provided. This is likely to be a particularly severe problem where lead is collected and melted informally by individuals, with no protection or controls on fume. This can also cause other individuals (such as other family members) to be exposed to lead fumes and dusts.

### **7.4.3 TRENDS AND HEALTH IMPLICATIONS**

In the Western World, blood lead levels have decreased significantly in the last two decades. The phasing out of leaded petrol is believed to be a major contributor to this, particularly in urban areas. However, other sources of lead exposure have also decreased. Phasing out of lead piping for the distribution of drinking water, and improved treatment to reduce lead dissolution in water, have

reduced lead contents of the most highly contaminated waters in the UK, though lead in drinking water still continues to be a source of lead intake in some soft water areas. The phasing out of leaded solders in canned food has also removed a general exposure route for the population. Leaded paint, though no longer used except in a few specialist outdoor applications, is still a significant source of lead intake by children living in older houses, where paint residues remain.

Blood lead levels of the vast majority of the population in Western Europe and the USA are well below  $10\mu\text{g}/\text{dl}$ . However, in some countries significant numbers are still exposed to elevated levels. In the USA this is strongly linked with low income and race, with non-whites being particularly exposed. In the USA and France, the age of the dwelling is significant, showing paint from older houses is an important exposure route.

Problems of lead exposure therefore appear to be very few in some countries, though significant minorities continue to be exposed in others.

Individuals occupationally exposed to lead are required to be monitored to limit their exposure. Exposure levels are generally declining with improved practice and stricter legislation, and most employees have blood lead levels well below the legal limits.

The graph below, Figure 7.1, gives a summary of some surveys of population exposure to lead, performed in many countries during the past 3 decades. The absolute levels cannot be directly compared, because the surveys tested different populations. For example, even within one country, the blood lead concentrations are different for men, women and children, and also vary with the age of the individual. Blood lead levels also depend upon many factors including location, occupation, social status, smoking habits. This figure is included to give an overview of trends in population exposure to lead in Europe and a few other countries based on survey results. It is not necessarily representative of the whole populations of countries. Despite these limitations, it is quite clear that there is a marked universal decline in lead exposure, and that this is still continuing.

Outside the Western World, there are instances of elevated exposure to lead in the population, particularly in large urban areas such as Mexico City, where very high levels of blood lead have been found in residents. Similarly, in South Africa in 1984, athletes who trained in urban areas had much higher median blood lead levels ( $55.5\mu\text{g}/\text{dl}$ ) than athletes training in rural areas ( $17.7\mu\text{g}/\text{dl}$ ). Ten years later, the concentration of lead in petrol had halved (from  $0.8\text{g}/\text{l}$  to  $0.4\text{g}/\text{l}$ ) and blood lead levels of urban and rural athletes had declined to  $13.0\mu\text{g}/\text{dl}$  and  $8.5\mu\text{g}/\text{dl}$  respectively (Grobler et al, 1996, cited in IEH, 1998). Lead in petrol is still permitted and continues to expose the population. Occupational exposure is also likely to be higher in some poorer countries, where standards of health and safety at work are less strict than in the West, and control measures may not always be used.

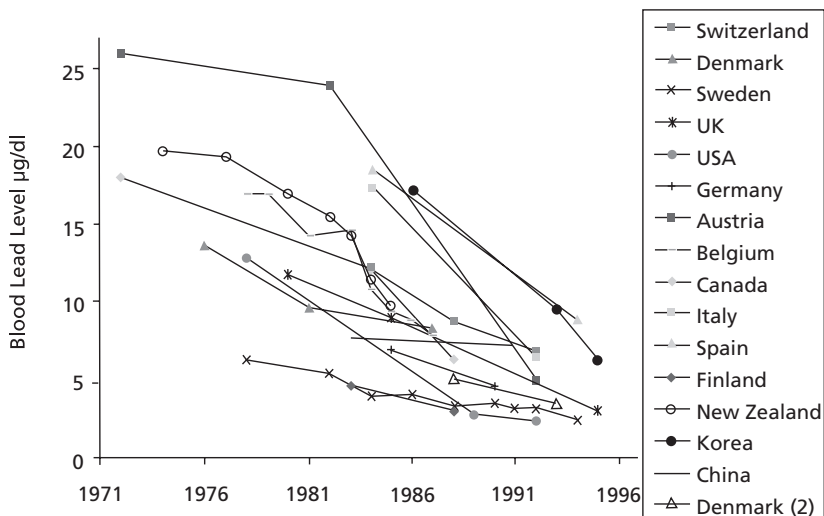


Figure 7.1 Blood lead trends reported in literature

## 7.5 POLICY APPROACHES TO LEAD

### Risk assessment or the precautionary principle?

Two different approaches can be taken to develop policy options for the regulation of a potentially hazardous substance such as lead, where knowledge of the full effects is incomplete. Lead (and other metals) present a special case in that they are sparingly soluble in most products and in most chemical and mineral forms in the environment, but at the same time are persistent in many environmental media.

### Risk assessment

Environmental risk assessment involves a search for a “best route” between social benefit and environmental risk, and providing a “tool” in decision making and in the process of risk management. It is a balancing or trade-off process in which various combinations of risks are compared and evaluated against particular social or economic gains. It does not necessarily imply a no-risk policy, or a minimum one. However, risks should be as fully understood as possible if they are to be effectively managed (SCOPE 15, 1980).

Making a risk assessment involves identifying:

- a hazard (in this case lead),
- a target population which can potentially be affected,
- an exposure pathway by which it reaches the target population, and

- the effect which it has on the population at a given dose, and whether there is a “safe” dose which gives no detrimental effects.

Just because a substance is hazardous, it does not necessarily pose a risk to human health, an ecosystem, or any other “target” population. There must be a route, or pathway, by which a population is exposed to the hazard. For example, a contaminated industrial area which is occasionally visited by adults will pose a much smaller risk than a garden with similar contamination (i.e. a similar hazard), but which is used as a play area for children. Similarly, the risk of using a material which is hazardous, such as lead, depends on there being some way that humans, or other living organisms, are exposed to that material, either during the preparation and manufacturing processes, the use of the article, or its final disposal.

The technique of risk assessment can model both the severity of the hazard to the population, and the probability of the population being exposed to the hazard. The final outcome, i.e. the predicted risk, depends upon the combination of all of these factors. Answers are complex and are given in terms of probabilities, because within a population there are always individual differences in susceptibility and exposure from different sources. However:

- There is no single universal approach used in all situations, and calculations using alternative risk assessment methodologies can yield different end results. A diversity in methodologies should be encouraged, so that all possible risk outcomes can be considered and the potential for error is minimised (Hallenbeck, 1993, *Quantitative Risk Assessment for Environmental and Occupational Health*).
- The calculations assume that all possible exposure routes have been considered, (which may not always be the case, particularly in the long term).
- Even though much study has been done on the behavior of lead in the environment, and its effects, particularly on human health, knowledge is not complete. (This is also true for other substances; in fact, lead has been more widely researched than some alternatives.) Thus, some parameters used in the computations are not known, and must be estimated - and these estimates (often termed default assumptions) can be inaccurate.
- Calculations should take into account the natural background levels in soils, which vary considerably between locations, and where possible the forms of the lead present which will influence its solubility and bioavailability.

Though the technique is being developed to improve predictions, as yet risk assessment is not an exact science.

### **Precautionary Principle**

The Precautionary Principle is defined as an approach to risk management that is applied in circumstances of scientific uncertainty, reflecting the need to take

action in the face of a potentially serious risk, without awaiting the full results of scientific research. This is a political approach, exercised in order to protect citizens or the environment from a threat. When scientific data are incomplete and there is a significant danger to human, animal or plant life, decision-makers are justified in taking action. Such action could involve refusing to license or allow a potentially hazardous activity (for example, an industrial process) or substance (such as lead), unless it can be demonstrated that any risks involved would be acceptably small. The Precautionary Principle is enshrined in international law, including European Community legislation and the Rio Declaration of 1992.

Recent guidance on the implementation of the Precautionary Principle, states clearly that:

- implementation of an approach based on the Precautionary Principle should begin with an objective risk assessment, identifying the degree of uncertainty at all stages,
- all the stakeholders involved should be involved in the decision to study the various management options that may be envisaged once the results of risk assessment are available, and the procedure should be as transparent as possible,
- measures based on the Precautionary Principle must be proportionate to the risk which is to be limited or eliminated,
- measures based on the Precautionary Principle must include an overall cost benefit assessment (advantages /disadvantages) with a view to reducing the risk to a level which is acceptable to all stakeholders,
- measures based on the Precautionary Principle must be able to establish responsibility as to who must furnish the scientific proof needed for a full risk assessment, and
- measures based on the Precautionary Principle must always be of a provisional nature, pending the results of scientific research performed to furnish the missing data and perform a more objective risk assessment.

(*Source*: European Commission DG XXIV Consumer Policy and Consumer Health, “Guidelines on the Application of the Precautionary Principle” (draft), 17th October 1998).

### **Summary**

The two above approaches are not contradictory, but should be used together when making policy decisions. The Precautionary Principle is justified when knowledge is incomplete and there is significant potential for harm. Policies based on this Principle should take account of all available information and be reviewed as more detailed knowledge emerges. All risk assessments must include an account of their uncertainties and limitations in order to be useful.