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PAPER FOR
PRACTITIONERS AND
POLICY MAKERS

Blood lead levels for Australians

Introduction

Lead is a heavy metal used extensively in the manufacture of storage batteries, various alloys including solder and ammunition, some plastics and protective coatings. It is not required for human health, and even small amounts of lead and lead compounds can be toxic when ingested or inhaled.

Based on the research evidence on the effects of low-level exposure to lead, it is not possible to make a definitive statement on what constitutes a 'safe level' or 'level of concern' for blood lead concentrations. Bearing this in mind, this Information Paper examines the evidence on the effects of lead exposure and makes recommendations on what should be strived for in terms of blood lead concentrations in Australia.

How humans are exposed to lead

Although lead occurs naturally in the environment, human activities have increased levels of lead in the biosphere more than a thousand-fold in the past 300 years. Much of this increase occurred in the second half of the twentieth century because of

RECOMMENDATIONS

- All Australians should have a blood lead level below 10 µg/dL (micrograms per decilitre).¹
- All children's exposure to lead should be minimised.
- All women are advised to minimise their exposure to lead both before and during pregnancy and also while breastfeeding.

This Information Paper is intended for health care practitioners and policy makers. It provides background to lead exposure and articulates goals and recommendations for Australia.

the use of lead compounds in petrol, a practice which has ceased in Australia (except under special licence). Lead also enters the environment as a consequence of the mining and refining of lead, industrial and manufacturing activities associated with products which contain lead, and the inappropriate disposal of waste materials containing lead (see Figures 1 and 2).

¹ Throughout this document blood lead concentrations are given as micrograms of lead per decilitre of blood (µg/dL). There are strong historical reasons for preferring this choice of units to SI units of micromoles of lead per litre of blood, but interested readers can always convert to SI units by dividing the figure for micrograms per decilitre by 20.7



Industrial and hobby activities which can generate lead dust and/or fumes include:

- restoration of homes, boats, cars and furniture coated with lead-based paints (probably the most important source of lead exposure in communities which are not industrially exposed)
- pottery (glazing and firing)
- soldering (radiators, stained glass, electronics)
- lead casting (to make ammunition, fishing sinkers and toy soldiers)
- Burning of lead-stabilised plastics or materials coated with lead-based paints
- Recycling of objects containing, or coated with, lead products, e.g. motor vehicle bodies, batteries, electronic equipment.

Other potential sources include:

- ceramic cookware, especially if it was improperly fired (e.g. imported tagines) and then used to cook or store acidic, alcoholic or hot foods
- imported toys containing lead, or coated with lead-based paints
- some 'traditional' medicines
- some cosmetics
- ingestion of soil or dust contaminated with lead (a problem in small children).

Although cigarettes contain only small amounts of lead, studies conducted both in Australia and overseas have consistently found elevated blood lead concentrations in adults who smoke, and in children who live in households where there is a smoker (Baghurst et al, 1987, Mannino et al, 2003). The mechanism of this elevation is not well understood.

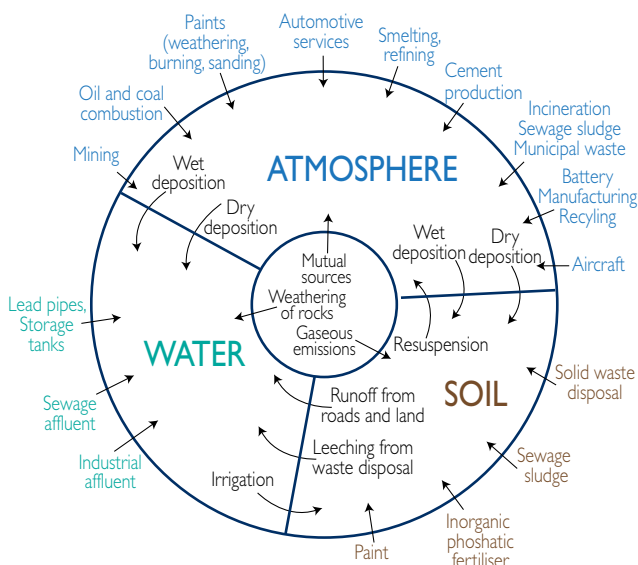


Figure 1: Transport pathways for lead in the environment (as presented in EPA, 2006, pp.2-84, which in turn acknowledges that the diagram is modified from Zabel, 1993).

Once lead has been ingested and absorbed, it circulates in the blood, with a high proportion attached to specific proteins in red blood cells. Much of the lead we ingest is excreted, mostly in faeces and urine, but some of it is absorbed in soft tissues such as the kidney, liver and brain, and some is incorporated into 'hard' or calcified tissue such as the bones and teeth. Our bodies therefore accumulate lead over time. More than 90% of lead in the adult human body is found in bone; in children it is more than 70%.

Most people are exposed to lead through breathing air which contains very fine particles of lead and/or through swallowing dust or dirt that is contaminated with lead. Children's rapid neurological development in their early years combined with hand-to-mouth activities and the ease with which they absorb and retain lead, makes them more vulnerable to lead exposure than adults. Other routes of absorption (through the eye or skin) are slower and less important, and the US Agency for Toxic Substances and Disease Registry (ATSDR) has argued that few studies have provided quantitative estimates of dermal absorption of inorganic lead in humans (ATSDR, 2007). The quantitative significance of the dermal absorption pathway as a contributor to lead body burden in humans is therefore uncertain.

Despite removal of lead from petrol, lead is still ubiquitous in the environment and exposure continues (see Figures 1 and 2). There are minute quantities of lead in the food and public water supply, although lead in drinking water in Australia is generally well within the limit of 10 µg/L recommended in the Australian Drinking Water Guidelines (2004).

Less is known about lead contamination of private water supplies, especially rainwater tanks. In localised areas around lead-related industries, surveys of lead in tank rainwater have resulted in strong advice to residents not to use this water for drinking or food preparation. The enHealth Council concluded from recent surveys in Adelaide and Newcastle that there does not appear to be any significant impact of urban and non-lead related industrial emissions on the lead content of tank water in these cities, although lead flashing on roofs has been identified as a potential source of lead exposure. The Council called for further studies in Australia's largest cities, i.e. Melbourne and Sydney (enHealth, 2004).

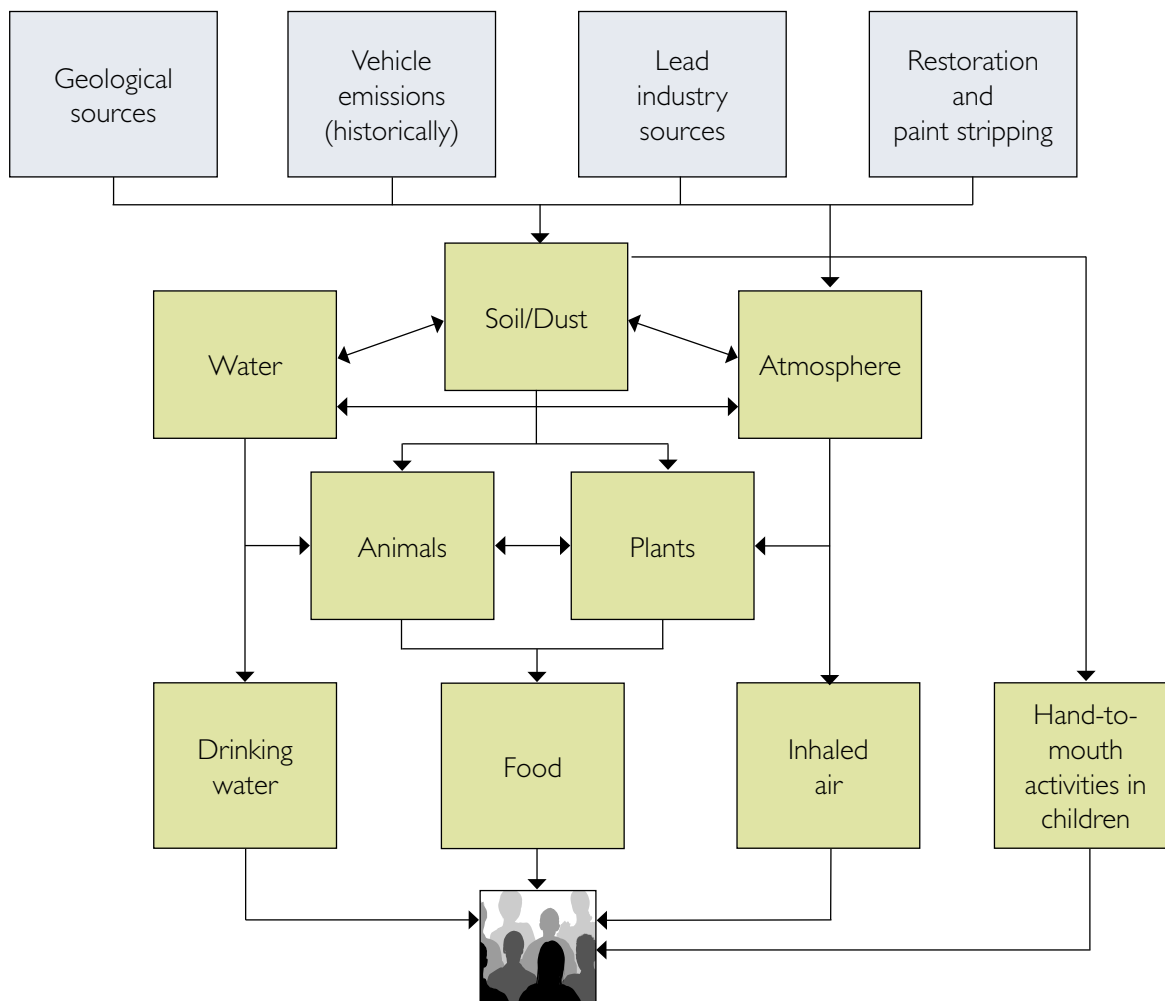


Figure 2 Principal pathways of lead from the environment to humans (EPA, 2006).

Health effects of lead exposure

A brief summary of health effects is given below. A more detailed account is available from the ATSDR, 2007, United States Department of Health and Human Services.

Acute effects

Immediate health effects of high-level exposure to lead resulting in blood lead values of 70-100 micrograms per decilitre or above are clinical emergencies, and may cause encephalopathy or severely affect neurological function. Symptoms include stomach pain, vomiting, convulsions (fitting), loss of consciousness and possibly death. Occurrences of this kind of lead poisoning are now extremely rare.

Longer-term health effects

Long-term exposure to lead at levels less than those required to cause acute symptoms can result in weakness in the fingers, wrists and ankles, general fatigue, headaches, anaemia, small increases in blood

pressure, and reduced kidney function. There is a paucity of studies examining past lead exposure and health outcomes over long periods of time in adults with most research focussing on possible effects of lead on cardiovascular health. A recent review of observational studies by Navas-Acien et al. (2008) concluded that while the apparent effect of lead on blood pressure was modest, the association could be causal, but there was not sufficient evidence to infer a causal relationship of lead exposure with clinical cardiovascular outcomes. In a 12–16-year follow-up of participants aged 30 to 74 in the National Health and Nutrition Examination Survey (NHANES II), individuals with blood lead levels of 20–29 µg/dL from 1976 to 1980 were found to have experienced significantly increased all-cause, circulatory and cardiovascular mortality (Lustberg & Silbergeld, 2002). NHANES III (Menke et al. (2006)) observed increasing mortality from myocardial infarction and stroke with increasing blood lead concentration after a 12 year follow-up of 13,946 adults whose initial blood lead concentrations (in 1988 to 1994) were below 10 µg/dL.

A study of 535 former organo-lead manufacturing workers whose occupational exposure had ceased an average of 16 years earlier found that their cognitive functioning was declining faster than that of community controls of similar age (Schwartz et al, 2000).

The World Health Organization (WHO) International Agency for Research on Cancer (IARC) recently concluded that there is limited evidence in humans for the carcinogenicity of inorganic lead compounds, and inadequate evidence in humans for the carcinogenicity of organic lead compounds (such as once used in leaded petrol). The ATSDR considers the evidence to be inconclusive (IARC, 2006; ATSDR, 2007).

Health effects on reproduction

High lead exposure may cause pregnant women to miscarry. Lead is transferred to the unborn infant during pregnancy, and a small amount passes into breast milk. There is evidence that lead adversely affects sperm motility, size, numbers and quality in occupationally exposed males.

Health effects in children

Children are considered to be more sensitive to the health effects of lead than adults. They absorb more lead into their bloodstream from the gut and airways and retain more in their body than adults. Only in the past 30 years has it been generally recognised that lead may impact adversely on the neurodevelopment of children at exposure levels well below those required to induce the acute signs and symptoms described above. Typically these effects have been assessed using the tools of psychologists and psychiatrists such as Ability Scales, Intelligence Quotients (IQ) and standardised reports of emotional and behavioural problems.

While nearly all researchers agree that higher lead exposure is associated with poorer developmental performance and behaviour using these measures, there is uncertainty over the extent to which low-level lead exposure is actually causing poorer performance (CDC, 2005). This uncertainty arises partly because other factors such as genetics and early childhood experiences are also major determinants of children's abilities and behaviours. When these factors are all taken into account, the relationship between lead exposure and children's abilities and behaviour becomes less certain. Nevertheless, a working group commissioned by the United States Centers for Disease

Control and Prevention (CDC) (2005) to review evidence for effects of lead exposure at low blood lead levels concluded that:

...collectively, these concerns and limitations of the available evidence preclude definitive conclusions about causation and leave considerable uncertainty concerning the magnitude and form of causal relations that may underlie these associations. At the same time, available evidence does not refute the interpretation that these associations are, at least in part, causal.

The nature of the 'dose-response' relationship between lead exposure and children's intellectual abilities and behaviour is also contentious. No threshold of lead exposure below which any exposure might be considered 'safe' in respect of cognitive abilities has ever been identified. Earlier work has indicated that, at a population level, an increase in blood lead exposure from around 10 to 30 µg/dL is associated with an average decrease of 1 to 3 IQ points. But two US studies, one from a highly disadvantaged community (Canfield et al, 2003) and the other from an upper socioeconomic community (Bellinger et al, 1992), have reported larger decreases in IQ as blood lead levels increase from 3 to 10 µg/dL. Brown and Rhoads (2008) in an Editorial in *Environmental Health Perspectives* have cautioned against over-interpretation of the findings from these two studies. It must be emphasised that these estimates cannot be used to predict the responses of individual children to a given lead exposure.

Measuring and managing lead exposure

Measuring the concentration of lead in blood (blood lead levels) is the accepted biomarker for recent lead exposure (i.e. in the preceding 5–6 weeks). Long-term exposure is measured by analysis of teeth (in children) and X-ray fluorescence (XRF) of bones, but XRF measurements are not available in Australia and there are no guidelines for lead in teeth.

A report to the NHMRC in 2002, 'Child Health Screening and Surveillance: A Critical Review of the Evidence', suggests that representative samples of children aged 1–4 living in high and low lead exposure areas should be screened for lead exposure, but it does not recommend implementing universal screening programs to detect lead toxicity in healthy children (Centre For Community Child Health, 2002).

How much lead are Australians exposed to?

The only nationwide survey of blood lead concentrations was conducted on 1,575 children in 1995 (Donovan, 1996), nine years after all new cars were required to run on unleaded petrol, and when sales of unleaded petrol constituted 50–60% of all sales. The average (geometric) blood lead concentration in 1995 in 1–4-year-olds was 5.1 µg/dL, with 7.3% exceeding 10 µg/dL and 1.7% exceeding 15 µg/dL. Communities close to known sources of lead pollution would have contributed only a very small fraction to the national sample, but it is worth noting that a recent survey in Mt Isa (Queensland Health, 2008) found that 11% of children surveyed had blood lead concentrations in excess of 10 µg/dL. A recent five-year longitudinal study of 113 children living in Sydney, aged six months to 31 months at recruitment, showed a mean blood lead concentration of 3.1 µg/dL (Gulson et al, 2006).

Medical management of lead exposure

Acute lead poisoning must be managed in a clinical setting, but such events are now rare in Australia.

Clinically supervised therapy to reduce blood lead concentrations, using metal-chelating agents like EDTA (ethylene-diamine-tetra-acetic acid) or sulph-hydril compounds such as DMSA (dimercapto-succinic acid, also known as succimer) and d-penicillamine, is strongly recommended for adults with acute lead poisoning, and may be considered in asymptomatic adults with a blood lead concentration of 50 µg/dL or higher (AOEC, 2007).

A large study of the effects of chelation therapy using DMSA in US children, aged 12 to 33 months with an average blood lead concentration of around 26 µg/dL, did not yield sustainable changes in blood lead concentration or improve cognitive abilities, possibly because these compounds only remove a limited amount of lead from the skeleton, which may then release stored lead back into the bloodstream (Rogan et al, 2001).

Animal studies have raised concerns that EDTA may actually increase brain lead levels, at least over the short term (Cory-Slechta et al, 1987) and that reductions in brain lead using DMSA may be much slower than reductions in blood lead (Stangle et al, 2004). Therefore it appears that more research is needed to identify optimal chelation strategies. However, treatment options were not considered within the scope of this document.

Intervention strategies

The major aim of governments worldwide is to implement risk management principles to minimise lead exposure in the home, community and workplace. In Australia, the removal of lead from petrol has been accompanied by a significant reduction in average blood lead levels. Better management of known sources of pollution in communities such as Port Pirie, Broken Hill, Mt Isa and the Lake Macquarie area has decreased average blood lead levels in those communities considerably.

The two strategies underlying interventions for minimising exposure to lead are:

- identifying the source of lead and implementing appropriate remediation and management techniques
- removing exposed individuals from the source.

Apart from site-specific communities like Port Pirie, Broken Hill and Mt Isa where management of lead exposure requires special care and lifestyle modifications, the most common non-occupational exposure to lead arises from renovation work which entails disturbing old paint containing lead. However, great care is still required in hobby activities such as those listed in section one. Many health authorities now provide information about the measures required to minimise exposure to lead in such circumstances (including advice that, where practicable, it may be safest to simply cover the old paint with a fresh coat of paint). Some links to practical advice for minimising lead exposure are provided at the end of this Information Paper.

Goals and recommendations

In the past 30 years, recommendations for limiting exposure to lead in the community have been largely driven by the need to protect children from the effects of lead on the developing brain. The setting of exposure targets, and the formulation of appropriate actions to be implemented when such targets are not met, is complex. This is because of the unusual shape of the exposure-response curve, the practicalities of achieving particular targets, and the difficulties associated with disentangling the apparent effects of lead from those of other determinants of children's neurodevelopment (CDC, 2005).

From 1993 to 2005, the NHMRC has recommended an overall goal of all Australians having a blood lead concentration below 10 µg/dL, together with a series of graduated responses depending on the proportion of children in a community who exceed blood lead concentrations of 15 and 25 µg/dL. It was never intended that this goal of 10 µg/dL be interpreted as either a 'safe' level of exposure or a 'level of concern'.

CDC, ATSDR and WHO also have a (non-occupational) goal of 10 µg/dL. While some groups have recently lobbied the US Government for a further reduction in this figure, CDC has argued for its retention (CDC, 2005; Brown & Rhoads, 2008).

Links to practical advice for minimising lead exposure

Please note: This list is not necessarily comprehensive.

NATIONAL

Guidelines for lead paint removal – aimed especially at workers: www.workershealth.com.au/facts057table.html

Guide to lead paint management – Australian Standards AS4361.1 (Industrial, 1995), and AS 4361.2 (Residential and Commercial, 1998). The contents pages of AS4361.2 can be viewed at: www.saiglobal.com/PDFTemp/Previews/OSH/As/as4000/4300/43612.pdf

NSW

A factsheet on lead exposure in children, from NSW Health: www.health.nsw.gov.au/factsheets/environmental/lead.html

A short guide, 'Lead and health – is your family at risk', from NSW Health: www.health.nsw.gov.au/mhcs/publication_pdfs/4465/BHC-4465-ENG.pdf

QUEENSLAND

Government information on lead paint exposure: http://access.health.qld.gov.au/hid/AccidentsInjuriesandPoisonings/SafetyintheHome/leadPaintExposure_ap.asp

SOUTH AUSTRALIA

Information on lead poisoning from the Women's and Children's Hospital: www.wch.sa.gov.au/services/az/divisions/labs/geneticmed/lead.html

Information on lead poisoning from Parenting and Child Health: www.cyh.com/HealthTopics/HealthTopicDetails.aspx?p=114&np=304&id=1895

Government advice on 'What to do about lead': www.dh.sa.gov.au/pehs/topics/topic-ptpirie-services.htm

'What can we do to minimise our child's exposure to lead?', from Ten by 10: www.tenby10.com.au/EveryChild/WhatCanWeDo.aspx

VICTORIA

A short guide, 'Lead and health – is your family at risk', from NSW Health but also relevant to Victoria: www.health.nsw.gov.au/mhcs/publication_pdfs/4465/BHC-4465-ENG.pdf

WESTERN AUSTRALIA

'Managing possible lead dust in and around the home': www.health.wa.gov.au/envirohealth/home/docs/Managing_lead_dust_in_and_around_the_home.pdf

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