

LEAD - TRACE ELEMENT

Introduction

Atomic mass 207. A ubiquitous constituent of the environment, lead has been used as a component of paints and as a petrol additive. Lead has no physiological role in the body. Its toxicity arises from its capacity to mimic other biologically important metals, becoming incorporated into the relevant metalloenzymes to interfere with their normal function.

Exposure

Settings that may result in exposure are lead mining and smelting, battery manufacturing and recycling, through pigments, solder, ammunitions, car radiators, cables and ceramics use, brass foundries, glass industries and plastics industries. Significant exposure to lead may occur in some hobbies. Lead is present in paint manufactured prior to 1970 and thus may contaminate soil adjacent to older houses. Pica (the ingestion of non-food substances, particularly soil) may lead to toxicity in children. There have been instances of lead poisoning from Ayurvedic preparations.

Acute and chronic poisoning may occur in places of heavy contamination. These places are typically near to mines and smelters. Heavy, localised contamination may result from stripping leaded paint or be associated with lead using industries. General exposure to lead is related to airborne fume emitted from motor vehicles and industrial emissions. Airborne lead eventually drops-out to contaminate soil, dust, sediments and waterways. Lead particles in dust and soil are small, requiring high performance vacuum devices to ensure effective removal. Where the contamination is related to vehicle exhaust, gross elevation of soil and dust lead concentration is restricted to the area within 25 metres of the road. Water and food contamination has not been found as a widespread problem in Australia.

Absorption

Human exposure is almost always associated with inhalation or ingestion. Approximately 40-50% of particles of less than 1 micron are retained in the lung and absorbed. Children and fasted adults absorb more lead from the gut than normally fed adults. Iron deficiency is thought to enhance absorption.

Distribution

Lead accumulates in bone and teeth where it displays a half-life of years. A much smaller proportion of the body burden is distributed to the soft tissues including red blood cells. The half-life of this compartment is 1 month. Therefore, the determination of blood lead content demonstrates recent exposure to the metal with levels rising within hours of exposure. 95% of lead in whole blood is associated with the red cells.

Excretion

Lead is mainly eliminated from the body in the urine by glomerular filtration and tubular secretion.

Pathology

Altered haem synthesis is found early after lead exposure and is due to inhibition of delta aminolevulinic acid dehydratase. Lead is also a neurotoxin. Neurobehavioral deficits have been described in children with blood leads in the range 0.5 to 1.4 $\mu\text{mol/L}$.

Patients with acute toxicity (blood lead $>3.5 \mu\text{mol/L}$) may have abdominal pain, constipation, arthralgia, myalgia, headache, anorexia, decreased libido, difficulty concentrating, short-term memory deficits, anaemia, peripheral neuropathy (frequently 'wrist/ankle drop').



Adults with lower level, chronic exposure (blood lead 1.5 to 3.5 $\mu\text{mol/L}$) may be asymptomatic or present with vague non-specific symptoms such as myalgia, fatigue, irritability, insomnia, anorexia, impaired short-term memory, difficulty concentrating.

Prolonged exposure may result in progressive renal disease and/or hypertension, abdominal colic and disturbance in reproductive function.

Monitoring

Whole blood lead is the sample of choice for the investigation of inorganic lead exposure. It is a short-term marker of exposure and also reflects redistribution of lead from the skeleton.

Urine lead (24-hour or spot) is the test of choice for organic lead exposure, which results in only modest elevation in blood levels. However, organic lead toxicity is much less of an issue given the cessation of its use in petrol. Urine lead levels are not recommended for monitoring exposure to inorganic lead as at low concentrations results are highly variable, even when corrected for creatinine concentration. Urine lead determination is, however, valuable in determining the efficacy of chelation therapy. The use of a variant of chelation, the EDTA mobilisation test, is not recommended by this site due to the toxicity of the drugs, the potential for eliciting acute lead toxicity and the difficulty in interpreting the result of the test.

The investigation of the lead content of deciduous teeth allows the retrospective estimation of lead exposure. Lead accumulates at different rates within the tooth. Analysis of secondary dentine is the most valuable test and this requires careful preparation of the tooth, restricting the determination to specialist laboratories.

Determination of lead in hair is not recommended because of the confounding variable of environmental contamination.

The erythrocyte morphology on peripheral blood smear may give a clue to acute toxicity when basophilic stippling is evident.

Historically, zinc protoporphyrin or free erythrocyte protoporphyrin have been used as measures of the effect of lead on haemoglobin synthesis over the preceding three-month period. However, these analytes are not sensitive at concentrations now known to cause neurobehavioral deficits or with acute exposure and, furthermore, may be elevated by other conditions so cannot be recommended.

Treatment

Various groups have established action guidelines related to various blood lead concentrations in different scenarios. In occupational exposure each state promotes its own guidelines. Workcover NSW guidelines for Lead Risk Work can be found on their web-site at <http://www.workcover.nsw.gov.au/>.

For exposure in children, the National Health Medical Research Council (NHMRC) recommends the following responses to lead determinations.

Lead $<0.7 \mu\text{mol/L}$ - no response.

Lead 0.7 to $1.2 \mu\text{mol/L}$ - identify exposure source and remediate, counselling, repeat test to evaluate effect of actions.

Lead $>1.2 \mu\text{mol/L}$ - take medical history; identify exposure source and remediate, counselling, repeat test in 3 months.

Lead $>2.6 \mu\text{mol/L}$ - as for $>1.2 \mu\text{mol/L}$ and include urgent clinical assessment.

The NHMRC promotes a goal blood lead for all Australians of $<0.5 \mu\text{mol/L}$.

Analysis

Collection of blood for the determination of lead should be made with care to avoid environmental contamination. Capillary sampling is not recommended. Routine blood lead determination at this site is made by inductively coupled plasma mass spectrometry. Determinations in other biological and environmental matrices in support of investigation and remediation are also available at this site.

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