

LEAD

Introduction

Lead is one of the “heavy metals”, an ill-defined subset of elements which includes transition metals, metalloids, lanthanides, and actinides. The alternative term is “toxic metal”, for which also no consensus of exact definition exists. Such metals are environmental pollutants which come from a number of sources, including lead in petrol, industrial effluent and acid rain-leaching of metal ions from the soil into rivers. There are more than 10,000 scientific publications on the internet dealing with lead toxicity [1], making it the most extensively studied toxic agent. The recent statement by the European Food Safety Authority “Scientific opinion on lead in food” [2] states that, for average adult consumers, dietary lead exposure in Europe ranges from 0.36 up to 2.43 µg/kg body weight per day. Exposure in infants ranges from 0.21 to 0.94 µg/kg per day and in children from 0.80 to 5.51 µg/kg body weight per day. Looked at from the cellular level these apparently low levels of exposure represent a very high dose of this toxic metal; an average adult exposure of 1.0 µg/kg body weight per day, with an average GIT absorption of 10% of ingested lead, translates into approximately 100 lead atoms reaching each cell of the human body per day [1].

Lead is absorbed more readily by children than by adults and is well recognized as a neurotoxin, with adverse effects on fertility and foetal development. Lead accumulates in soft tissues and, over time, in bones, while excretion is primarily via the urine and faeces. Based on current evidence, EFSA has determined that there is no known safe dose of lead, i.e., it is not possible to identify a level of lead exposure which is without harmful effect in humans. Nevertheless, there are “reference levels” for lead in blood, urine, hair and water, which are quoted on laboratory reports to give guidance on the potential magnitude of the toxic hazard posed by the presence of lead in the tissues.

The EFSA study [2] concludes that the current provisional tolerable weekly intake (PTWI) of 25 µg/kg body weight is no longer appropriate as there is no evidence of a threshold for critical lead-induced effects. The margins of exposures are such that the possibility of an effect from lead in some subjects, particularly in children from 1-7 years of age, cannot be excluded (note the higher levels of exposure in children mentioned above). Protection of children against the potential risk of neurodevelopmental effects would be protective for all other adverse effects of lead, in all populations.

Sources of lead

The EFSA study [2] states that **cereal products** currently contribute most to dietary lead exposure, while dust and soil can be important sources in children. Lead has a variety of uses and can still be found in the home, for example in old paint [3]. Petrol containing anti-knock lead is still available in the U.K. for use in lawnmower engines.

Activities with a high risk of lead exposure include:

- 1) Radiator repair - solder (60% lead, 40% tin) is used; after repair the excess solder is buffed off using a grinder, creating dust. In addition radiators were formerly painted with lead based paint but this practice has been discontinued.
- 2) Smelting - the heat generated in the furnace leads to production of lead fumes. Lead (2-3%) is added to brass (copper/zinc alloy) and some bronzes (copper/tin alloy) e.g. gunmetal, as it gives the metal a lubrication quality.

- 3) Car exhaust repair - exhaust systems may have deposits of lead oxide from lead petrol. This creates fine dust and lead fumes during welding.
- 4) Scrap metal recovery – which includes burning off the covering from old cables, cutting lead enamelled baths, handling lead batteries and smelting down lead using crude furnaces.
- 5) Container repair - welding, brushing and sandblasting of lead paint in the confined spaces of containers produces dusts and fumes.
- 6) Small bore rifle shooting - lead fumes may arise from both the bullet and the primer. There is additional lead in the dust in the shooting range and older ranges may not be properly ventilated.
- 7) Lead battery manufacture - used lead batteries are recycled by smelting down into lead ingots. Manufacture of new lead batteries involves forming lead plates, pasting them with lead oxide and assembling them in to the battery.

Activities with a medium risk of lead exposure include:

- 1) Metal machining - dust is created by the cutting and spinning of metal alloys containing lead (for example, brass).
- 2) Ceramics/arts - lead based glazes may be used; some artists still use lead-based paints.
- 3) Plastic production - PVC contains tri-basic lead sulphate as an acid scavenger to slow the degradation of PVC and improve thermal stability. Exposure can occur where the bags of powder are opened and mixed to give a free-flowing powder that is approximately 2% lead.

Activities with a low risk of lead exposure include:

- 1) Plumbing - plumbers may still use red lead putty as a sealant, but most risk is from old lead-based solder joints and during preparation of lead flashings on roofs.
- 2) Bright soldering (used in jewellery manufacture).
- 3) Motor car assembly and repair - lead is used in car assembly lines as a gap filler on seams (it is illegal to use synthetic fillers). The excess lead metal is removed with a hand file. Restorers of vintage cars still use lead. In the past anti-rust paint from cars also contained lead.

Indications

Background exposure to lead in the UK has greatly declined since the practice of using lead as an “anti-knock” additive in petrol was discontinued. However, occupational exposure and accidental exposure remains a problem. An annual blood test is recommended if a risk assessment shows there is potential exposure to lead. Subjects with low level toxicity can present with ill-defined symptoms including general aches and pains, especially abdominal pains, without having developed clinical microcytic anaemia and basophilic stippling. Acute lead poisoning in adults is characterized by abdominal pain, tiredness, aching limbs and joints, and irritability. Nerve palsy and wrist drop are rarely observed. In children and animals lead poisoning is accompanied by CNS signs such as convulsions, irritability and vomiting, as well as anaemia [4].

Current results suggest that water is still a significant source of lead exposure in the U.K. Unlike most water contaminants, lead is predominantly a “home” contaminant, sometimes as a result of water treatments aimed at improving water quality. These treatments can destabilize lead-bearing scale that coats the inside of pipes and also cause corrosion to solder, pipes and fixtures that are lead-containing.

Lead in blood

Reports quote < 0.50 $\mu\text{mol/L}$ as the reference interval for blood lead, which is the blood level below which there is no evidence of excess exposure as compared to the rest of the population. Population reference ranges from the literature are shown below (PCV is packed cell volume) [5,6]:

Age group	Whole blood lead ($\mu\text{mol/L}$)	PCV
adult male	0.0-0.50	0.40-0.55
adult female	0.0-0.35	0.38-0.45
child 5-16 yrs	0.0-0.55	0.35-0.40
child 9 mths - 4 yrs	0.0-0.55	0.33-0.37
child < 9 mths	0.0-0.50	0.30-0.35

Lead in urine

Reports for urine lead quote < 2.20 $\mu\text{mol/mol}$ creatinine as the reference interval for urinary lead excretion.

Lead in hair

Reports quote < 2.00 $\mu\text{g/gm}$ (ppm) of hair as the reference interval for hair lead. This is a useful measure of lead exposure since sequestration in hair represents a de-toxification mechanism rather than evidence of an adverse health effect for lead.

Patient preparation

No special preparation is required and the patient can continue to take nutritional supplements and medication before the collection of the sample.

Specimen requirements

Blood collected into an 8 ml trace element-free potassium EDTA tube is required for blood lead analysis. Collection tubes are available from the laboratory. If a number of blood tubes are being taken at the same collection, the trace element free tubes should be filled first to avoid cross-contamination.

A 24-hour urine collection is preferred for urine lead determination, but a 6-hour urine collection is acceptable. The total volume of urine collected should be recorded and, after mixing, 15 mL of urine in a plastic screw-top universal container should be sent to Biolab. A postal sample kit can be supplied.

Hair for lead analysis should be cut from the back of the head, or nape of the neck, as close to the scalp as possible. At least 0.25gm of hair is required (about one heaped tablespoon). Only hair up to 1½" (4cm) from scalp should be used.

Methodology

Lead is measured by inductively coupled plasma - mass spectrometry (ICPMS).

Turn around time

5 working days.

References:

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2. European Food Safety Authority. Scientific Opinion on Lead in Food. EFSA panel on contaminants in the food chain (CONTAM). *EFSA J* 2010;8:1570, 1-146.
3. Srianjata S. Lead - the toxic metal to stay with humans. *J Toxicol Sci* 1998;23S 2:237-240.
4. Rogan WJ, Dietrich KN, Ware JH et al. The effect of chelation therapy with succimer on neuropsychological development in children exposed to lead. *NEJM* 2001;344:1421-1426.
5. Soldin OP, Hanak B, Soldin SJ. Blood lead concentrations in children: new ranges. *Clin Chim Acta* 2003;327:109-113.
6. Zierold KM, Anderson H. Trends in blood lead levels among children enrolled in the Special Supplemental Nutrition Program for Women, Infants, and Children from 1996 to 2000. *Am J Public Health* 2004;94:1513-5.
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