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Lead (Pb): cheap metal – costly to health status

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INTRODUCTION

Lead is one of the most studied occupational and environmental agents and is known to have a broad range of effects on multiple organ systems. The diverse physical properties of this metal resulted in the extensive use in industry.

Metals like zinc, chromium and manganese are known to have properties essential to human physiology and health impairments are more difficult to diagnose as only work related. Whereas lead, mercury, arsenic and cadmium serve no recognised biological purpose and are considered highly toxic at concentrations above normal background levels.

Most metal exposures are ubiquitous, generally arising from air, water and food sources. They can vary considerably and may contribute to or have a synergistic effect that may be greater than the sum of their separate effects.

This widespread exposure and the array of health hazard that non-essential metals pose are of particular importance in occupational and environmental medicine as they can be and in many cases are very costly to our health status.

LEAD: BIOLOGICAL MARKERS AND BIOMONITORING OF METAL TOXICITY

The information available on toxicity of lead is enormous. However, to summarise, lead is still one of the more prevalent metals in our system that takes considerable effort to be eliminated and this can only be achieved by limiting or controlling the source and duration of the exposure.

Exposure

In an occupational setting humans are normally exposed to lead through inhalation, contaminated food and tobacco. Heavy exposure may occur in lead smelters, metal strippers, spray painting, storage, manufacture of batteries and in the industry where lead is a contaminant of the mineral extraction industry.

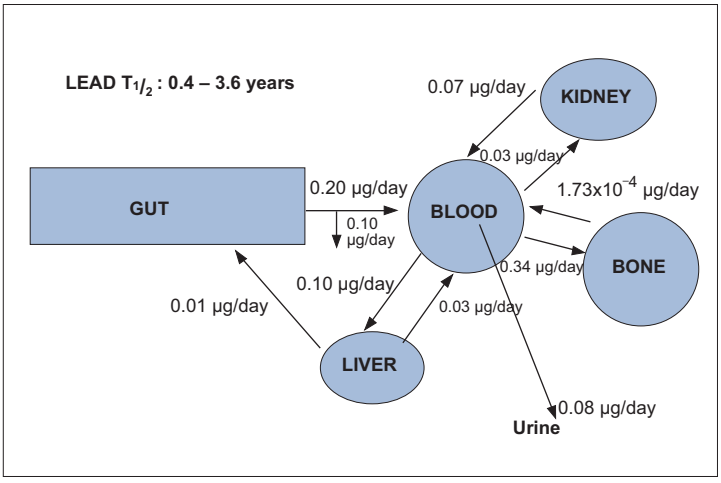

Figure 1. Multi-component model demonstrating the target organs and the effect of accumulation of lead. (Applied to adult humans exposed to 5 µg Pb/kg/day for 30 years – Mallon RP)

Metabolism

If exposed, 10–60% of the smaller lead particles (0,01 – 5 µm) are deposited in the alveolar region of the respiratory tract. Particles are also deposited in the nose, mouth and upper part of the airways. These are then swallowed or cleared. Lead is absorbed from the gastrointestinal tract and the absorption range can vary from 37–70% depending on the health status and dietary intake of that individual.

Distribution

Lead is absorbed in the blood plasma and readily equilibrates between plasma and the extracellular fluid. From the tissues, the lead is distributed to the bone marrow, liver, kidneys and, to some extent, passes through the blood-brain barrier into the nervous system. Such passage is probably higher in infants than in adults. A large portion is absorbed into the skeleton, which is known to accumulate 90% of the body burden. This in itself is very problematic for any worker who has been exposed to lead for long periods of time as the continuous mobilisation of lead from the accumulation in the skeleton causes considerable "endogenous" lead exposure.

Elimination

Elimination of lead is through glomerular filtration in the kidneys, probably followed by partial tubular reabsorption. Some of the lead is excreted in the bile and pancreatic juices and to some extent, excreted in sweat, seminal fluid, hair and nails. Lactating and child bearing age mothers are especially vulnerable to the transplacental passage of lead to the fetus and the lead secretion in the milk.

METABOLIC MODEL

Evidence of health effects

Over the years new research has demonstrated that lead exposure at concentrations of 5 µg/kg/day is sufficient to cause physiological and psychological changes and that the effects have been highly underrated. Health effects at low levels of exposure may only be evident years after chronic exposure and are mostly the result of lead body burden effect (see Figure 2). Body burden effect, the accumulation of lead in the skeleton, begins to play a significant role at the lower concentration end of the curve. Thus, to achieve levels below 15 µg/dL will require much longer time and for historically exposed individuals this might never be reached.

In both environmental (general public) and occupational settings the "no effect" level for lead is constantly being lowered as more sensitive analytical methods of the physiological and mental effects of lead are being developed.

Exposure limits of lead: inorganic, dust and fumes

ACLIH TLV: 0.5 mg/m^3 TWA
OSHA PEL: 0.05 mg/m^3 TWA
ACLIH BEI: 30 µg/dL, whole blood

HEALTH EFFECTS

Mechanisms of toxicity

Rather little is known about the basic mechanism behind lead toxicity. We know that lead binds to the sulphydryl groups of proteins and if this occurs on the enzyme, its function may be inhibited and eventually will result in a toxic response. Evidence exists that lead alters calcium mediated cellular processes which in turn may result in organ failure response.

Kidneys: Lead may cause kidney damage and in acute lead toxicity proximal tubular damage which can result in a reversible Fanconi syndrome-like condition (aminoaciduria, glucouria and hyperphosphaturia). Years of heavy exposure may result in interstitial nephritis, with interstitial fibrosis, tubular atrophy and arteriosclerotic damage. Such changes seem to occur at blood lead levels of 31.2 – 62.4 µg/dL or higher.

Blood and blood-forming organs: Lead has an inhibitory effect on stops in the chain reaction that lead to the formation of haeme. Lead also inhibits the activity of the enzyme pyrimidine-5-nucleotidase (P5N) in the red cells. This may lead to anaemia which is normocytic and sideroblastic. Anaemia ("lead pallor") occurs at whole blood lead concentrations of 62.4 µg/dL and higher.

Nervous system: Both peripheral and central nervous system (CNS) symptoms and signs have been documented at concentrations of 30.2 – 41.6 µg/dL. In subjects without obvious clinical signs of encephalopathia, subjective and nonspecific symptoms (fatigue, impaired concentration, loss of memory, insomnia, anxiety and irritability) can be observed or may occur.

Gastrointestinal tract: Lead has a negative effect on the gastrointestinal tract, causing diarrhoea, epigastric pain, nausea, indigestion, loss of appetite and colic, which normally occurs at whole blood lead levels of higher than 72.8 µg/dL.

Cardiovascular system: There is significant evidence linking high blood pressure to the increase in whole blood lead levels. However, secondary hypertension resulting from lead-associated kidney damage has been documented.

Genotoxicity: Lead acetate and lead sub-acetate can cause kidney and brain tumours, whereas the exposure to lead phosphate may result in kidney tumours at very high levels and chronic exposure.

Reproductive effects: The reproductive effects of lead exposure are well documented. Recent studies have shown that sperm vitality, motility and morphology impairment at whole blood lead concentrations greater than 41.6 µg/dL have been observed. In some studies, very low levels of lead exposure in women at child bearing age have resulted in stillbirth, preterm delivery, reductions of gestational age and birth weight, as well as sudden death syndrome. In many cases developmental impairment of these children has been documented. This has been observed at levels of 10.4 – 15.6 µg/dL and even lower in some cases.

BIOLOGICAL MONITORING OF EXPOSURE

Monitoring of whole blood lead in humans is fairly easy. However, the non-linear relationship between lead exposure/uptake makes any comparison to environmental levels very difficult. Low exposure and the accumulation effect of lead is a great concern. It can only be diagnosed from historical data and generally requires a few measurements over time. Thus, monitoring lead workers on an ongoing basis will be essential in determining whether any accumulation of the lead has occurred and whether occupational hygiene protocols and engineering procedures have been effective (see Figure 2).

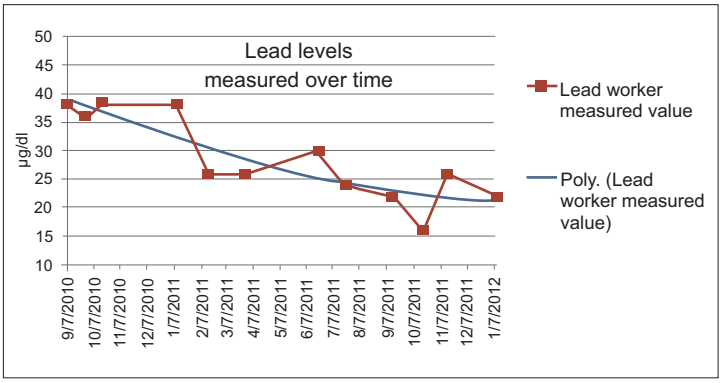

Figure 2. Demonstration of the body burden effect of a worker exposed to lead dust and the time it took to reach acceptable levels

CONCLUSION

Metals are rarely used in the pure form, usually being present in alloys. Therefore health care providers should be alert to complex symptomatology and unravel the effects of multiple exposures, to ensure the correct diagnosis. Exposure to lead or any other heavy metals that essentially have no functional metabolic properties does exert biological effects and may lead to long- or short-term health impairment or toxicity. Familiarising ourselves with the potential health effects of metals within different occupational settings is critical, not only for the occupational health and safety professionals, but also for the general medical practitioner.

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