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Study of Heavy Metals as Environmental Toxins

Chemistry

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ABSTRACT

Toxicity in Environment is a global concern. All of us are surrounded by environmental toxins. Substances that may cause distress or disease to our bodies can be found in everything that we eat, in everything that we drink and even in the air we breathe. Toxins, including chlorofluorocarbons, heavy metals, pesticides, herbicides, toxic waste, PCB(polychlorinated biphenyl), DDT, bioaccumulation, endocrine disruptors, asbestos and poorly implemented hazardous waste management. Some of these compounds are a by-product of an industrialized world. Generally Heavy metals are released from factories or are produced as waste substances in the industry. The objective of this paper is to investigate environmental occurrence, impact and remedial methods of some toxic heavy metals.

Introduction:

We are surrounded by environmental toxins. Substances that may cause distress or disease to our bodies can be found in everything that we eat, in everything that we drink and even in the air we breathe. Some of these compounds are a by-product of an industrialized world. Heavy metals like lead and cadmium are released from factories or are produced as waste substances in the industry. We are also exposed to many naturally-occurring toxic substances.

The main threats to human health from heavy metals are associated with exposure to lead, cadmium, mercury and arsenic. These metals have been extensively studied and their effects on human health regularly reviewed by international bodies such as the WHO[1]. exposure to heavy metals continues and is even increasing in some areas. For example, mercury is still used in gold mining in many parts of Latin America. Arsenic is still common in wood preservatives, and tetraethyl lead remains a common additive to petrol, although this use has decreased dramatically in the developed countries. Since the middle of the 19th century, production of heavy metals increased steeply for more than 100 years, with concomitant emissions to the environment (Figure 1). At the end of the 20th century, however, emissions of heavy metals started to decrease in developed countries: in the UK, emissions of heavy metals fell by over 50% between 1990 and 2000.

People may be exposed to potentially harmful chemical, physical and biological agents in air, food, water or soil. However, exposure does not result only from the presence of a harmful agent in the environment. The key word in the definition of exposure is contact. There must be contact between the agent and the outer boundary of the human body, such as the airways, the skin or the mouth. Exposure is often defined as a function of concentration and time: "an event that occurs when there is contact at a boundary between a human and the environment with a contaminant of a specific concentration for an interval of time. For exposure to happen, therefore, co-existence of heavy metals and people has to occur.

Cadmium: Occurrence, exposure and dose

Cadmium occurs naturally in ores together with zinc, lead and copper. Cadmium compounds are used as stabilizers in PVC products, colour pigment, several alloys and, now most commonly, in re-chargeable nickel–cadmium batteries. Metallic cadmium has mostly been used as an anticorrosion agent (cadmiation). Cadmium is also present as a pollutant in phosphate fertilizers. EU cadmium usage has decreased considerably during the 1990s, mainly due to the gradual phase-out of cadmium products other than Ni-Cd batteries and the implementation of more stringent EU environmental legislation (Directive 91/338/ECC). Notwithstanding these reductions in Europe, however, cadmium production, consumption and emissions to the environment worldwide have increased dramatically during the 20th century. Cadmium containing products are rarely re-cycled, but frequently dumped together with household waste, thereby contaminating the environment, especially if the waste is incinerated.

Health effects

Inhalation of cadmium fumes or particles can be life threatening, and although acute pulmonary effects and deaths are uncommon, sporadic cases still occur. Cadmium exposure may cause kidney damage. The first sign of the renal lesion is usually a tubular dysfunction, evidenced by an increased excretion of low molecular weight proteins [such as β_2-microglobulin and α_1-microglobulin (protein HC)] or enzymes [such as N-Acetyl-β-D-glucosaminidase (NAG)]. It has been suggested that the tubular damage is reversible, but there is overwhelming evidence that the cadmium induced tubular damage is indeed irreversible.

Mercury: Occurrence, exposure and dose

The mercury compound cinnabar (HgS), was used in prehistoric cave paintings for red colours, and metallic mercury was known in ancient Greece where it (as well as white lead) was used as a cosmetic to lighten the skin. In medicine, apart from the previously mentioned use of mercury as a cure for syphilis, mercury compounds have also been used as diuretics [calomel (Hg_2Cl_2)], and mercury amalgam is still used for filling teeth in many countries.

Inorganic mercury is converted to organic compounds, such as methyl mercury, which is very stable and accumulates in the food chain. Until the 1970s, methyl mercury was commonly used for control of fungi on seed grain.

Inorganic mercury

Acute mercury exposure may give rise to lung damage. Chronic poisoning is characterized by neurological and psychological symptoms, such as tremor, changes in personality, restlessness, anxiety, sleep disturbance and depression. The symptoms are reversible after cessation of exposure. Because of the blood–brain barrier there is no central nervous involvement related to inorganic mercury exposure. Metallic mercury may cause kidney damage, which is reversible after exposure has stopped. It has also been possible to detect proteinuria at relatively low levels of occupational exposure.

Metallic mercury is an allergen, which may cause contact

eczema, and mercury from amalgam fillings may give rise to oral lichen. It has been feared that mercury in amalgam may cause a variety of symptoms. This so-called 'amalgam disease' is, however, controversial, and although some authors claim proof of symptom relief after removal of dental amalgam fillings, there is no scientific evidence of this.

Organic mercury

Methyl mercury poisoning has a latency of 1 month or longer after acute exposure, and the main symptoms relate to nervous system damage. The earliest symptoms are paresthesias and numbness in the hands and feet. Later, coordination difficulties and concentric constriction of the visual field may develop as well as auditory symptoms. High doses may lead to death, usually 2–4 weeks after onset of symptoms. The Minamata catastrophe in Japan in the 1950s was caused by methyl mercury poisoning from fish contaminated by mercury discharges to the surrounding sea. In the early 1970s, more than 10,000 persons in Iraq were poisoned by eating bread baked from mercury-polluted grain, and several thousand people died as a consequence of the poisoning. However, the general population does not face significant health risks from methyl mercury exposure with the exception of certain groups with high fish consumption.

Lead: Occurrence, exposure and dose

The general population is exposed to lead from air and food in roughly equal proportions. Earlier, lead in foodstuff originated from pots used for cooking and storage, and lead acetate was previously used to sweeten port wine. During the last century, lead emissions to ambient air have further polluted our environment, over 50% of lead emissions originating from petrol. Over the last few decades, however, lead emissions in developed countries have decreased markedly due to the introduction of unleaded petrol. Subsequently blood lead levels in the general population have decreased (Figure 2).

Health effects

The symptoms of acute lead poisoning are headache, irritability, abdominal pain and various symptoms related to the nervous system. Lead encephalopathy is characterized by sleeplessness and restlessness. Children may be affected by behavioural disturbances, learning and concentration difficulties. In severe cases of lead encephalopathy, the affected person may suffer from acute psychosis, confusion and reduced consciousness.

Recent research has shown that long-term low-level lead exposure in children may also lead to diminished intellectual capacity. The combined evidence suggests a weighted mean decrease in IQ of 2 points for a 0.48 μmol/l (10 μg/dl) increase in blood lead level (95% confidence interval from −0.3 points to −3.6 points).

Arsenic: Occurrence, exposure and dose

Arsenic is a widely distributed metalloid, occurring in rock, soil, water and air. Inorganic arsenic is present in groundwater used for drinking in several countries all over the world (*e.g.* Bangladesh, Chile and China), whereas organic arsenic compounds (such as arsenobetaine) are primarily found in fish, which thus may give rise to human exposure.

Smelting of non-ferrous metals and the production of energy from fossil fuel are the two major industrial processes that lead to arsenic contamination of air, water and soil, smelting activities being the largest single anthropogenic source of atmospheric pollution. Other sources of contamination are the manufacture and use of arsenical pesticides and wood preservatives.

Arsenic (or metabolites) concentrations in blood, hair, nails and urine have been used as biomarkers of exposure. Arsenic in hair and nails can be useful indicators of past arsenic exposure, if care is taken to avoid external arsenic contamination of the samples. Speciated metabolites in urine expressed as either inorganic arsenic or the sum of metabolites (inorganic arsenic + MMA + DMA) is generally the best estimate of recent arsenic dose. However, consumption of certain seafood may confound estimation of inorganic arsenic exposure, and should thus be avoided before urine sampling.

Health effects

Inorganic arsenic is acutely toxic and intake of large quantities leads to gastrointestinal symptoms, severe disturbances of the cardiovascular and central nervous systems, and eventually death. In survivors, bone marrow depression, haemolysis, hepatomegaly, melanosis, polyneuropathy and encephalopathy may be observed. Ingestion of inorganic arsenic may induce peripheral vascular disease, which in its extreme form leads to gangrenous changes (black foot disease, only reported in Taiwan).

The relationships between arsenic exposure and other health effects are less clear. There is relatively strong evidence for hypertension and cardiovascular disease, but the evidence is only suggestive for diabetes and reproductive effects and weak for cerebrovascular disease, long-term neurological effects, and cancer at sites other than lung, bladder, kidney and skin.

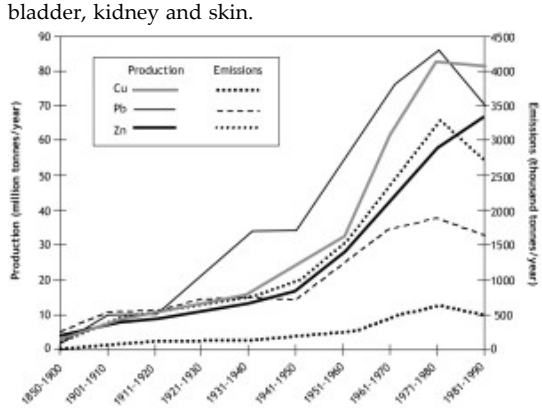

Figure 1: Global production and consumption of selected toxic metals [2].

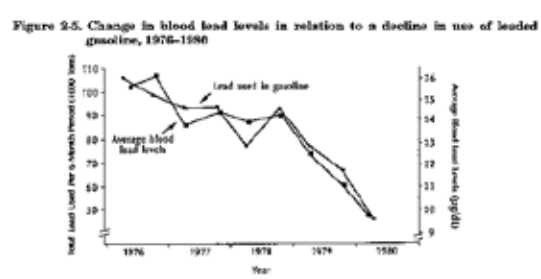

Figure 2: Lead concentrations in petrol and children blood (USA). (taken from cdc.gov)

Conclusions

Recent data indicate that adverse health effects of cadmium exposure, primarily in the form of renal tubular damage but possibly also effects on bone and fractures, may occur

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at lower exposure levels than previously anticipated. Many individuals in Europe already exceed these exposure levels and the margin is very narrow for large groups. Therefore, measures should be taken to reduce cadmium exposure in the general population in order to minimize the risk of adverse health effects.

The general population does not face a significant health risk from methyl mercury, although certain groups with high fish consumption may attain blood levels associated with a low risk of neurological damage to adults. Since there is a risk to the fetus in particular, pregnant women should avoid a high intake of certain fish, such as shark, swordfish and tuna. Fish, such as pike, walleye and bass, taken from polluted fresh waters should especially be avoided.

Long-term exposure to arsenic in drinking water is mainly related to increased risks of skin cancer, but also some other cancers, and other skin lesions such as hyperkeratosis and pigmentation changes. Occupational exposure to arsenic, primarily by inhalation, is causally associated with lung cancer. Clear exposure–response relationships and high risks have been observed.

References:

1) Lars Järup Oxford Journals Medicine & Health British Medical Bulletin Volume 68, Issue 1Pp. 167-182 (2003)
2) Image taken from oxford journals-medicine-british medical bulletin, vol. 68, issue 1.