



Inorganic Poisons and Treatment: A Review of the Literature

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Abstract

In this review we have studied inorganic poisons that are one type of poisons highlighted the inorganic poisons for some metals such as arsenic, mercury and lead and showing their effects on health. © 2017 ijrei.com. All rights reserved

Keywords: Inorganic metals, mercury, lead, arsenic, poisons.

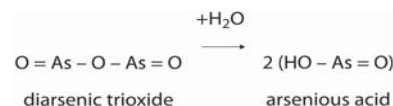
1. Introduction

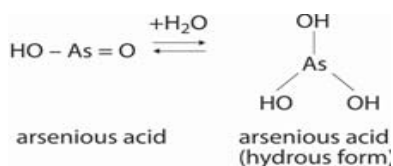
Heavy metals are natural components of the earth's crust and as such are the oldest toxins known to humans, have been used for thousands of years. There is high potential of exposing to heavy metals depending on the sources include the natural one such as groundwater, metal ores, industrial processes, commercial products, folk remedies, and contaminated food and herbal products. Generally, most heavy metals are toxic where sufficient quantities of these elements are reached.

Exposing human bodies via food, drinking water, or air, metals produce toxicity by forming complexes with cellular compounds containing sulfur, oxygen, or nitrogen. The complexes inactivate enzyme systems or modify critical protein structures leading to cellular dysfunction and death. The most commonly involved organ systems include central nervous, gastrointestinal (GI), cardiovascular, hematopoietic, renal, and peripheral nervous systems. The nature and severity of toxicity vary with the heavy metal involved, its exposure level, chemical and valence states (inorganic versus organic), mode of exposure (acute versus chronic), and the age of the individual. Children, with their developing nervous systems, are particularly vulnerable to heavy metal intoxication (especially lead) and deserve special consideration. We may mention some inorganic poisons such as

1.1 Arsenic

Arsenic compounds are available in three oxidation states: trivalent arsenite, pentavalent arsenate, and elemental. Arsenite is ten times more toxic than arsenate; elemental is nontoxic. Arsenic also exists in three chemical forms: organic, inorganic, and arsine gas, with organic arsenic has little acute toxicity in compare with inorganic arsenic and arsine gas which they are toxic [1]. Exposure to arsenic primarily occurs by ingestion, but inhalation and absorption through the skin are possible. Arsenic occurs naturally in seafood as nontoxic organic compounds, such as arsenobetaine, which can cause elevated urine arsenic levels [2, 3]. Diarsenic trioxide (As₂O₃, arsenic (III) oxide) is a trivalent arsenic compound and thus highly toxic, in contrast to less toxic pentavalent arsenates which being widely distributed in nature. When diarsenic trioxide is dissolved in water, it is immediately converted into the arsenious acid, exerts its toxicity in humans [4].





1.1.1 Effects of Arsenic on Human

1.1.1.1 Toxicological Effects

The clinical picture of chronic poisoning with arsenic varies widely. It is usually dominated by changes in the skin, mucous membranes, by neurological, vascular and haematological lesions. Involvement of the gastrointestinal tract, increased salivation, irregular dyspepsia, abdominal cramps and loss of weight may also occur. Reports of diminished sexual activity in persons with chronic arsenic exposure are frequent [5]. Arsenic and its inorganic compounds have long been known to be neurotoxic. Peripheral neuropathy in arsenic smelter workers has been reported, where by chronic exposure to arsenic dust caused a decrease in peripheral nerve conduction velocities. The skin is a common critical organ in people exposed to inorganic arsenical compounds. Eczematoid symptoms can be developed with varying degrees of severity. Hyperkeratosis, warts and melanosis of the skin are the most commonly observed lesions in chronic exposure. Increased mortality from cardiovascular diseases has been observed in epidemiological investigations of smelter workers exposed to high levels of airborne arsenic [6]. A peripheral vascular disorder leads to gangrene of the extremities, known as Blackfoot disease, has been observed [7]. Inorganic arsenic has an inhibitory effect on haematopoiesis, giving rise to anaemia, most commonly of the hypoplastic type. In severe cases of arsenical poisoning, agranulocytosis or thrombopenia may develop. An increasing of spontaneous abortions rate and lower mean birth weights was been reported among Swedish smelter workers and among subjects living in the vicinity of the smelter [8]. The rate of congenital malformations in the offspring for women working at the smelter was higher as well. However, it was not possible to prove these recorded cases with exposure to any specific compound in the smelter environment. The United States Environmental Protection Agency (EPA), is considering that hyperpigmentation, keratosis and possible vascular complications (blackfoot disease) as the critical effects, and stated that the accepted the value of 0.3 $\mu\text{g}/\text{kg}$ per day (no-observed-adverse-effect level (NOAEL) 0.009 mg/liter ; (converted to 0.0008 mg/kg per day); as the reference dose in the case of human chronic oral exposure.

1.1.1.2 Carcinogenic Effects

There are sufficient evidences that inorganic arsenic compounds are skin and lung carcinogens in humans [10].

Several studies showed that exposure to inorganic compounds can increase the risk of lung cancer in smelter workers, those involved in the production of arsenic-containing pesticides and metal ore miners [11]. The data often indicates positive dose-response relationships. Both trivalent and pentavalent arsenic compounds have been occurred in these exposure situations and at present the possibility cannot be ruled out that any form of inorganic arsenic may cause carcinogenic. Results of studies on the interaction between inorganic arsenic and smoking are conflicting: one of the studies, been done, provided evidence of a multiplicative interaction according to another [12]. The interaction between arsenic and smoking was intermediate between additives and multiplicative and appeared to be less pronounced among heavy smokers [13]. Some other investigations of populations living near copper smelters and other point sources of arsenic emission to the air have revealed moderate increases in lung cancer mortality. However, other studies have failed to detect an effect in such situations. Significantly elevated standard mortality ratios for cancer of the bladder, lung, liver, kidney, skin and colon were found to be in the population living in an area of Taiwan, China where arsenic contamination of the water supply was endemic. Lung cancer is considered as the critical effect following exposure via inhalation. Consequently, cancer at other sites, e.g. skin cancer, will not be discussed in detail here. A frequent elevation of chromosomal aberrations has been found in peripheral blood lymphocytes of wine-growers exposed to arsenic, in psoriasis patients treated with arsenic, and in arsenic-exposed copper smelter workers. Sodium arsenate inhibits DNA repair in human skin biopsy cells and in lymphocytes

1.2 Mercury

Elemental mercury is one of only two metals that are known to be liquid at room temperature [14]. It is a heavy, non-wetting liquid that is able to volatilize to an odorless gas in quantities sufficient to cause clinical toxicity at room temperature. Its uniform expansion over a wide range of temperatures and its ability to alloy easily with other metals led to mercury's use in several commercial applications, including thermometers, barometers, thermostats, electronics, batteries, dental amalgams, home folk remedies, and a host of other uses. Because of recent concerns regarding exposure and toxicity, the use of mercury in the manufacture of most of these products has been abandoned in favor of less toxic substances [15]. Many older mercury-containing products are still in use, however, and continue to be a source for potential toxicity. Inorganic mercury occurs naturally as mercuric and mercurous salts, the most common being mercury (II) sulfide (HgS), also known commonly as cinnabar and vermilion. This red, earthy-appearing ore also is found in a crystal form prized for its rich red color, and was equally prized as a source of mercury throughout its history [16]. Other common mercurial salts include mercuric chloride,

mercuric oxide, mercurous chloride, mercuric iodide, ammoniated mercury, and phenylmercuric salts [17-18]. Historically, these compounds have been used in cosmetics and skin treatments, particularly as skin-lightening agents [19]. Mercurial teething powders containing calomel (mercuric chloride) were in common use until the mid-twentieth century and were prescribed to infants to soothe the discomfort of teething [20]. Although these treatments no longer are prescribed, some dermatologic preparations still are available over the counter in the United States. Patients also may develop local or systemic toxicity after using mercurial compounds found in old topical antiseptics, skin creams, and folk remedies. Currently, most exposures in the United States occur from exposure to germicides, pesticides, and antiseptics mercury contains. Recently, mercury pollution has resurfaced, vapor of inorganic mercury easily pass through the blood-brain barrier and enter into the brain tissue [21]. A large quantity of inorganic mercury has been used to make amalgam in gold mines. Basically, when the mercury is heated up, it vaporizes and spreads into the atmosphere. The mercury vapor is inhaled and absorbed in the lung and reaches to the brain, and passing through the blood-brain barrier, when gold miners are exposed, causing inorganic mercury poisoning [22]. Mercury chloride ($HgCl_2$), a form of inorganic mercury, affects the epithelial cells of proximal tubuli of the kidney. Takahata et al (1970) reported two cases of inorganic mercury poisoning. Both, individually, had very high mercury level in the brain, higher than those in common other cases of inorganic mercury poisoning after short-term exposure to mercury vapor. However, no lesions were authenticated. Clinically, these patients showed gingivitis, tremor, erethism, disturbance of speech, delirium, and rigidity. In August 1993, three fatal cases of acute mercury vapor poisoning among twenty seven patients who worked in a refinery (Toho Zinc Co., LTD) were autopsied [23]. The two cases that studied by Takahata et al [24] showed slightly elevated mercury and mild but non-specific pathologic changes in the brain. In contrast, the kidneys showed high levels of mercury, with necrosis of the renal tubules.

In other hand, four autopsy cases of inorganic mercury poisoning were examined, including two cases of acute inorganic mercury poisoning from short-term exposure to mercury vapor and two cases from prolonged exposure that resulted in long survival. In first man patient (IKH K-31) report, the average value of total mercury in the brain was $1.08 \mu g/g$ and that of methyl mercury was $0.025 \mu g/g$. While with the second man (IKH K-32) case, the average value of total mercury was $0.73 \mu g/g$ and that of methyl mercury was $0.077 \mu g/g$. In the brain of the third man (HU 594), the average value of total mercury was $11.38 \mu g/g$, and that in the brain of the fourth man (HU 602) was $10.82 \mu g/g$, as measured by Akagi's method [25-26].

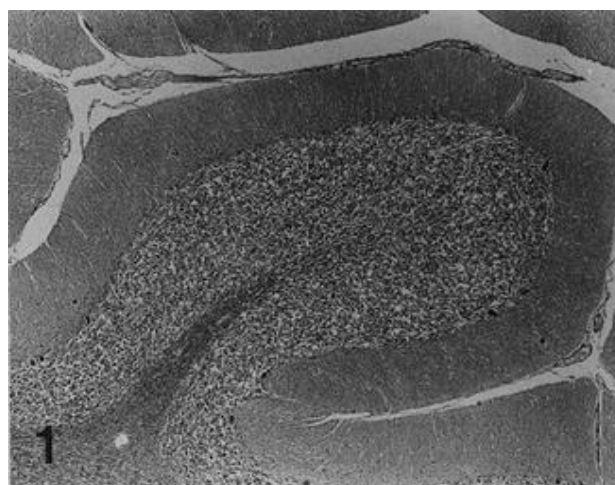


Figure 1: Decreased number of Purkinje and granule cells in the cerebellum of a man (IKH K-31) with acute inorganic mercury poisoning. Note bullous lesions, which are postmortem changes H&E. X60 [24].

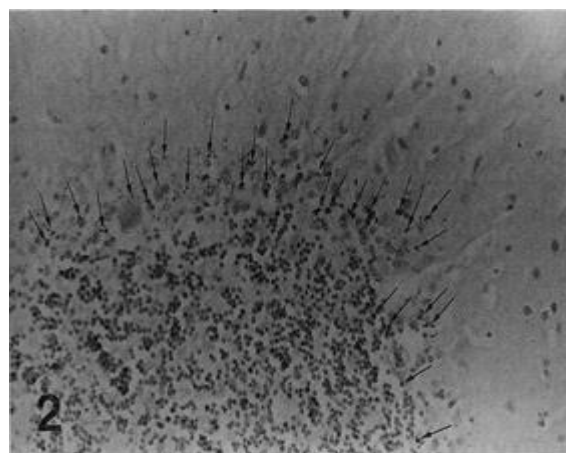


Figure 2. Black-brown granules represent mercury deposition in the cerebellum (IKH K-31). Histochemistry. X 150.

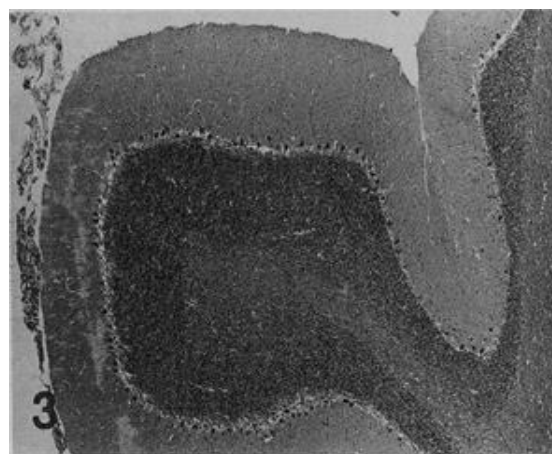


Figure 3. No lesions are found in the cerebellum of a man (HU 594) with chronic inorganic mercury poisoning. H&E. X 60.

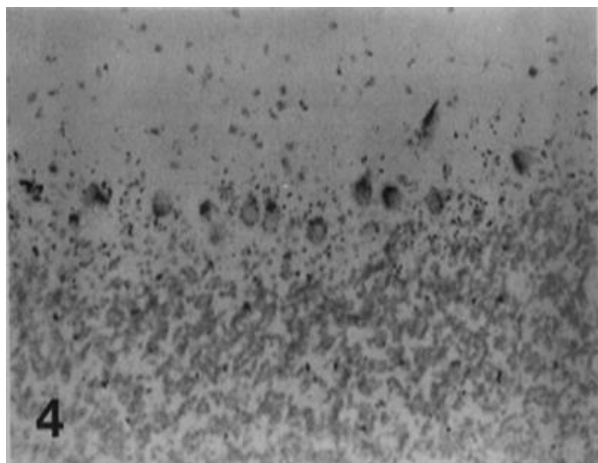


Figure 4. Mercury granules are clearly identified in the Purkinje and Bergman's glial cells (HU 594). *Histochemistry. X 150.*

In the brain of the first man (IKH K-31), the neuronal architecture of the cerebral cortices was well preserved with deposition of lipofuscin pigment in the neuronal cytoplasm. Ischemic changes were prominent in the cerebellum, which also showed postmortem bullous changes (Figure 1). Mercury granules were identified in the Bergman's glial cells and macrophages in the granular cell layer (Figure 2). Congestion and widened Virchow-Robin spaces were found in the brain stem. With the second man (IKH K-32), the changes were similar except that there were recent infarcts in the occipital lobe and the putamen. The brain of the third man (HU 594) showed nonspecific changes, including lipofuscin within the neurons and corpora amylacea in the brain stem and in the spinal cord. The histological features of the cerebellum are shown in Figure 3. However, there was conspicuous deposition of mercury granules predominantly in the Purkinje cells and Bergman glia (Figure 4). The fourth man (HU 602) showed no specific changes except for chromatolysis of neurons.

1.3 Lead

Lead poisoning, particularly its negative impact on children during the early growth years, is a public health problem in continuing importance. As an understanding of the ramifications of lead poisoning has continued to evolve, public health advocates have pushed for legislation that has decreased the amount of lead in gasoline, residential paint, metal solder and plumbing components. As a result, few children suffer from the most serious effects of lead poisoning such as seizures, comas or death. However, a great deal of old leaded paint still exists in older housing, and thousands of children continue to be exposed to lower doses of lead that can result in subtle but serious health problems.

Lead enters the body primarily through inhalation and ingestion of lead containing dust. Once in the body, lead travels in the blood to soft tissues such as the liver, kidneys, lungs, brain, spleen, muscles, and heart. The body does not

change lead into any other form. Once it is taken in and distributed to the organs, the lead that is not stored in the bones is eliminated slowly from the body by the kidneys and gastrointestinal tract; negligible amounts of lead are lost through perspiration. About 99% of the amount of lead taken into the body of an adult will leave in the waste within a couple of weeks, but only about 32% of the lead taken into the body of a child will leave in the waste [27-28]. The half-life of lead varies from about a month in blood, 1-1.5 months in soft tissue, and about 25-30 years in bone [29-31]. Lead in bone is considered a biomarker of cumulative exposure because lead accumulates in bone over the lifetime and most of the lead body burden resides in bone. Some of the lead can stay in the bones for decades; however, some lead can leave the bones and reenter the blood and organs under certain circumstances, for example, during pregnancy and periods of breast-feeding, after a bone is broken, and during advancing age.

1.3.1 Sources of Lead

Now the main sources of lead poisoning are components of many colors used in the home, the water flowing through pipes coated with lead. It is desirable in the first place to avoid these factors. Other sources are listed below: Sources:

- Emissions of aircraft engines
- Oil of lead-based paint
- Car batteries
- Bone meal fertilizer from
- Ceramic coating on porcelain
- Cigarette smoke
- Dust and particles from lead-based paints
- Insecticides
- Pipes made of lead or lead-coated
- The process of lead ore from motor fuel with a high content of lead (exhaust)
- Vegetables grown near highways

1.3.2 Poisonous Effect of Lead

Lack of toxicity of lead means that there is no clearly defined symptoms, but there may be more subtle signs of poisoning, because, like radiation, lead is a cumulative poison. Once in the body, it accumulates in bones, liver and kidneys. Even moderate levels can lead to kidney impairment and immune suppression. Obvious symptoms of lead poisoning are severe weakness, spasms in the abdominal area, and paralysis. Asymptomatic, but also dangerous is the constant presence of lead in blood. It affects the formation of hemoglobin and causes anemia. There may be disturbances of the psyche. Lead poisoning in children of lead workers: 38 in 91 children (41.8%) presented lead blood levels up to 300 ug/L and 10 with lead blood levels higher than 800 ug/L. The source of contamination was lead dust carried home on parents contaminated work clothing. Lead In humans, Long term exposure can occur acute or chronic damage to the nervous

system on humans. Heavy metals can enter our bodies via food (fish, meat, and produce) and drinking water, and children can be exposed to lead through ingesting paint chips. The Environmental Protection Agency ranks lead, mercury, and cadmium as three of the top six toxins. Leaded gasoline is responsible for the proliferation of lead across the planet, even into Greenland's ice. More than 90 percent of houses built before 1975 still have lead paint. Lead also exists in everything from plumbing to calcium supplements, hair dye, mini-blinds, and leaded crystal. Urban areas with high levels of traffic or trash incinerators, as well as areas containing battery plants or industrial facilities that burn fuel, may still have high lead levels in the air [32-34]. Lead damages the kidneys, brain, blood (it enters the red blood cells), muscles and bones. Symptoms of Lead toxicity may include chronic kidney disease, hypertension, encephalopathy, anaemia, gout, sterility, abortion, fatigue, irritability, ADD, hyperactivity, memory loss, decreased sensory and motor reaction times, and abdominal pain.

2. Conclusion

Poison is define as a substance that when introduced in to a living organism causes injury, illness, or death by chemical reaction or other activity on the molecular scale. Poisons are classified according to chemical agents into organic poisons and inorganic poisons. In this review we show some inorganic poisons such as arsenic, mercury and lead and have been found that because of their high degree of toxicity arsenic, lead, and mercury rank among the priority metals that are of public health significance. These metallic elements are considered systemic toxicants that are known to induce multiple organ damage, even at lower levels of exposure.

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