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Metal toxicity: Adverse Effect of E-waste on Human Health and Bioremediation

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ABSTRACT

In this modern era, enormous E waste is generated, improper dismantling and processing of which leads to accumulation of various heavy metals in the ecosystem. Heavy metal toxicity is the major concern as it has adverse impact on human health. The toxicity of heavy metals depends upon the absorbed dose, the route of exposure and duration of exposure, i.e. acute or chronic. Most of the heavy metals are carcinogenic in nature. This can lead to various disorders and can also result in excessive damage due to oxidative stress induced by free radical formation. This review gives details about some heavy metals and their toxicity mechanisms, along with their health effects. Microbial bioremediation could be the economical and promising solution to detoxify the toxic heavy metals.

Key words *: Heavy metal, Bioremediation, Toxicity, Microbial*

Introduction

E -waste is famous, informal name for electronic products at the end of their life. E waste is defined as 'electrical and electronic equipment, discarded as waste by the consumer. Disposal of e-wastes is a particular problem faced in many regions across the globe. The toxicants commonly found in E-waste include toxic metals and metalloids such as lead, mercury, arsenic, barium, beryllium, cadmium, chromium, etc. Out of these toxicants found in the e-waste lead, mercury, cadmium all accumulate in the environment as well as in the human body and are categorized under persistent bio-accumulative toxins. Other than these, arsenic in electronics, chromium, selenium and nickel in dyes and semiconductors, beryllium in motherboards, cobalt in insulators, copper in cables and circuitry, lithium in cell phones and batteries and silver and zinc in batteries and alloys are among the leading pollutants in the environment. (Deval *et al*., 2015)

The main objective of this review is to provide insight into the sources of heavy metals and their harmful effects on the environment and living organisms. Heavy metals are generally referred to as those metals which possess a specific density of more than 5 g/cm3 and adversely affect the environment and living organisms.

Metal ions are very reactive and interact with biological systems. Each human cell has many metal-binding sites. Binding of essential metals to these receptors is necessary for enzymatic activity. The mechanism of metal toxicity is mimicry of indispensable metals; toxic metals bind to the receptors and disrupt the vital metal-mediated cellular functions. For example, mimicry of zinc is a mechanism of toxicity for cadmium, copper, and nickel. Other mechanisms for metal toxicity are formation of cross

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linkages with DNA, proteins, abnormal gene expression & by forming reactive oxygen species etc. (Klaassen, 2018).

Some heavy metals and toxicity mechanisms

Lead

Lead is one of the major toxicants in e-waste. An old CRT television contains about 1.5-2kg Pb, and a CRT computer monitor contains of 0.5kg Pb (Deval *et al*., 2015). Thus, analyzing the health impacts of bio-accumulation of the element caused by improper disposal becomes essential. Lead can induce a wide range of adverse effects in the human body depending on the duration of exposure and amount of ingestion. Children are more susceptible because of the impairment of cognitive and behavioral development caused by the element (Ryan *et al*., 2004), while in adults, Iead toxicity produces peripheral neuropathy and hypertension (Navas-Acien *et al*., 2007). Lead has various hematologic effects, ranging from increased urinary porphyrins, leading to microcytic and hypochromic anemia (Klaassen, 2018). Acute lead nephrotoxicity leads to proximal tubular dysfunction, whereas chronic lead nephrotoxicity leads to interstitial fibrosis and progressive nephron loss, azotemia, and renal failure (Bellinger, 2011). There is evidence of relationship between lead exposure and hypertension (Navas-Acien *et al*., 2007). Lead alters calcium-activated contraction and functioning of vascular smooth muscle cells. It increases contractility by inhibiting Na+/K+-ATPase activity and stimulating the $Na+/Ca^{2+}$ activity.

The developing immune system is sensitive to toxic effects of lead (Bellinger, 2011). Lead-associated immunologic changes include altered T-cell subpopulations, reduced immunoglobulin levels, and reduced polymorphonuclear leukocyte chemotactic activity. All these changes in turn lead to a malfunctioning immune system (Luebke *et al.*, 2006). Lead can affect bone by interfering with calcium metabolism. This occurs as lead substitutes calcium in bone. It is associated with osteoporosis and delays in fracture healing (Carmouche *et al.*, 2005).

The mechanisms proposed for lead-induced carcinogenesis, include regenerative repair, inhibition of DNA synthesis or repair, generation of ROS with oxidative damage to DNA, substitution of lead for zinc in transcriptional regulators, interaction with DNA-binding proteins, and aberrant gene expression (Silbergeld, 2003).

Mercury

Mercury (Hg) is used in laptop monitors, cold cathode fluorescent lamps, cell phones, and printed circuit boards. The improper recycling of e-waste may release Hg in its elemental vapor form into the environment causing life threats to humans and many animals (Deval *et al*., 2015). Mercury is the most prevalent toxic metal found in E-waste.

Hg is present as a stable monoatomic gas (vapor form) in the environment. It evaporates from the Earth's surface and is emitted by volcanoes. Anthropogenic sources include emissions or improper E-waste recycling. After approximately 1 year, mercury vapor is converted to soluble form and returned to the earth by rainwater. Hg dissolved in water may be converted back to the vapor by microorganisms and this is followed by release into the atmosphere. Thus, mercurial recirculation may take place for long periods. Mercury attached to aquatic sediments is converted to methylmercury by microbes. This biomethylation is followed by biomagnification starting with plankton, then herbivorous fish, and finally ascending to carnivorous fish and sea mammals. Ingestion of these methylated fish can cause health hazards in humans, and eventually in developing foetus if consumed by pregnant women (Klaassen 2018).Mercury has the potential to cause neurotoxicity in humans and especially developing fetus (Dietz 2013). This ionic mercury can also damage the renal tubules during excretion. In the Central nervous system, mercury acts mainly upon cerebellum, temporal lobe, basal ganglia, and corpus callosum (Pilay, 2018).

Cadmium

Cadmium is used in nickel-cadmium (Ni-Cd) batteries, surface mount devices, chip resistors, infrared detectors, and semiconductor chips.

Its deleterious effects include deficits in cognition, learning, behavior and neuromotor skills in children. Cadmium accumulation in the body can cause kidney damage and fragile bones, affecting joints. Injury to the spine may lead to Itai-itai disease (Deval *et al.*, 2015).

Cadmium's toxic effects may be due to the displacement of zinc by cadmium. Cadmium may also cause apoptosis (programmed cell death). Cadmium is toxic to renal tubular cells and glomeruli, markedly impairing renal function (Pilay, 2013). This cadmium-induced renal toxicity is reflected by proteinuria (protein in urine) and hypercalciuria, i.e.

excessive calcium excretion in urine which attribute to bone remodeling (Kazantzis, 2004).

Cadmium exposure may induce obstructive lung disease in humans. The blood–brain barrier severely restricts cadmium access to the central nervous system. Thus, cadmium toxicity occurs only in case of cadmium exposure prior to blood–brain barrier formation (young children), or with blood–brain barrier dysfunction under certain pathological conditions (Klaassen, 2018). Prenatal exposure to cadmium is associated with adverse birth outcomes, impaired child health, and developmental errors (Vilahur *et al*., 2015). In humans, exposure to cadmium has been most clearly associated with lung, renal and pancreatic cancer (Djordjevic *et al.,* 2019).

Arsenic

Arsenic is found in the microchips of many electronic devices including mobile phones. Low levels of exposure affect the skin, liver, nervous and respiratory systems, while high levels of exposure can be fatal (Deval *et al.,* 2015).

Sensory loss in the peripheral nervous system is the most common neurological effect. Anemia and leucopenia occur within a few days of high arsenic exposure (Klaassen, 2018).

The skin is a major target organ in chronic inorganic arsenic exposure. The characteristic changes in skin epithelium include diffuse or spotted hyperpigmentation and, alternatively, hypopigmentation (Alain *et al.,* 1993). Cardiovascular disease like coronary artery disease are the most common noncancer diseases associated with chronic toxicity (Nigra *et al.,* 2016). Chronic exposure to arsenic is also associated with a range of nonmalignant respiratory effects including respiratory symptoms, chronic obstructive pulmonary disease, and respiratory disease mortality (Parvez *et al.,* 2013). Liver injury, being a characteristic of chronic arsenic exposure, manifests as jaundice, abdominal pain, and hepatomegaly (Mazumderand Dasgupta, 2011). Arsenic exposures, especially in utero and during early developmental, may affect central cognition and memory behaviors (Wasserman *et al.,* 2011). This type of exposure results in immunotoxicity and potentially increases risk of infections and inflammatory-like diseases during childhood and in adulthood (Schulz *et al.,* 2002).

Chromium

Hexavalent Chromium (Cr+6) is used for protecting ungalvanized steel plates from corrosion. Researches have proved that Cr exposure can damage the kidneys and liver and also cause bronchial anomalies such as lung cancer and asthmatic bronchitis(Deval *et al*., 2015).

Hexavalent chromium also causes chronic ulceration of skin surfaces (Travis *et al.,* 2003). It causes contact dermatitis among previously sensitized individuals. Occupational exposure to chromium may cause asthma. (Bright *et al*., 1997). Hexavalent chromium compounds are toxic to genetic material. (De Flora, 1990). Once hexavalent chromium enters cells, it is reduced by various intracellular reductants. During the reduction process, various genetic lesions can be generated, including chromium–DNA adducts, DNA–protein cross-linkages, DNA–chromium intra-strand cross-linkages, DNA strand breaks, and oxidized DNA bases (Travis *et al*., 2003; Macfie *et al.,* 2010).

Selenium

Selenium is found in various electronic components such as circuit boards and photosensitive drums in photocopiers. It is a semiconductor and is used in photocells as well.

Symptoms of fatal selenium intoxication include nausea and vomiting, followed by pulmonary edema and cardiovascular collapse (Fairweather-Tait *et al.,* 2011). Chronic selenium toxicity also known as selenosis, can occur with environmental exposure when the intake exceeds the excretory capacity. Toxicity of selenium causes mainly dermal and neurological effects. These include hair and fingernail loss, tooth discoloration, numbness, paralysis, and occasional hemiplegia. Selenium forms complexes with copper, and toxicity of either selenium or copper is influenced by the intake of the other elements. Selenium sulfide is anticipated to be a human carcinogen based on multiple positive rodent studies (Bethesda, 1980).

Nickel

Nickel is used in batteries, including rechargeable nickel cadmium batteries and nickel-metal hydride batteries used in hybrid vehicles.

Nickel induced contact dermatitis is the most common deleterious health effect caused due to nickel exposure. It is found in 10% to 20% of the population. Nickel is a respiratory tract carcinogen (Prueitt *et al*., 2020). There is a high risk of lung and nasal cancers due to nickel intoxication. The carcino-

genic nature of nickel is thought to be due to the generation of ionic nickel, which is the active and genotoxic form (Qiao *et al.*, 2017).

Beryllium

Beryllium is used as a component of motherboards and connectors and has been classified as a human carcinogen (Deval *et al.*, 2015).

The primary route of exposure to beryllium compounds is inhalation. It is then deposited in the lung and slowly absorbed into the blood. Exposure to soluble beryllium compounds may result in conjunctivitis and papulovesicular dermatitis of the skin. (Costa *et al.*, 2008). Beryllium exposure may also cause a delayed-type hypersensitivity reaction in the skin, which is an immune response. If insoluble beryllium-containing materials become embedded under the skin, a granulomatous necrotizing and ulcerative lesion may develop. Inhalation of beryllium can initiate an inflammatory response of the entire respiratory tract. A number of epidemiology studies in U.S. beryllium workers found that deaths due to lung cancer were increased , along with increased incidence of respiratory diseases in these workers (Paolo *et al.*, 2012). The carcinogenic mechanism being under research, several molecular events including oncogene activation and tumor suppressor gene dysregulation have been suggested. (Klaassen, 2018).

Cobalt

Cobalt is mainly used in lithium-ion batteries, and in the manufacture of magnetic, wear-resistant and high-strength alloys. Cobalt alloyed with aluminium and nickel to make particularly powerful magnets.

Occupational inhalation of cobalt-containing dust may cause respiratory irritation at air concentrations between 0.002 and 0.01 mg/m^3. Occupational dermal exposure is said to be associated with an allergic dermatitis. There are increasing reports of cobalt leaching from cobalt containing nanoparticle wear products causing fatal cardiomyopathies (Packer, 2016). Chronic cobalt exposure targets mitochondrial enzymes causing mild effects on respiration and a minimal lowering of ATP levels (Clyne *et al.*, 2001). Inhalation of cobalt sulfate induces lung tumors. (Bucher *et al.*, 1999). Potential carcinogenic mechanisms include the release of cobalt ions, increased production of ROS resulting in oxidative stress response, and by causing cytotoxicity, genotoxicity, inflammation, and apoptosis (Cappellini *et al.*, 2016)

Copper

Copper is mainly used in power transmission, telecommunications, electronics circuitry, and innumber of electrical equipment.

The most commonly reported adverse health effects of copper intoxication are gastrointestinal distress, nausea, vomiting, and abdominal pain. Ingestion of large amounts of copper salts, most frequently copper sulfate, may produce hepatic necrosis and death. Studies have also found out copper toxicity to be carcinogenic (Lisa *et al.*, 2014).

Barium

Barium is used as a protective coating on the front panel of Cathode Ray Tubes to protect the users from harmful radiations coming out from the screens. Some researches claim that barium exposure can lead to brain swelling, muscle weakness and can also have an adverse effect over heart, liver and spleen (Deval *et al.*, 2015)

Barium intoxication blocks the exit channel for potassium ions in skeletal muscle cells, thus resulting in their accumulation inside the cell. This results in rapid onset of marked hypokalaemia. Profound hypokalemia and muscle weakness progressing to flaccid paralysis are the hallmarks of barium poisoning (Phelan *et al.*, 1984). Muscular cramps may also be seen. Metabolic and respiratory acidosis, and renal failure are commonly reported (Pilay, 2013).

Microbial Detoxification of Heavy Metal

Microorganisms have potential to detoxify the metal by various mechanisms like oxidation of metal, methylation, metal sequestration, production of metal chelators and exopolysaccharide etc. (Dixit *et al.*, 2015). In Bio Sorption mechanism, heavy metals are adsorbed on the surface of the cell and this can be carried out either by living cells or by dead Biomass (Fomina and Gadd, 2014).

Ability of microbial cell to accumulate the metal ions intracellularly is known as Intracellular Sequestration, *Rhizobium leguminosarum and P. putida* have ability of intracellular sequestration of heavy metals like Zn, Cu and Cadmium etc. (Lima *et al*., 2006)

Microorganisms have ability to alter the oxidation state of metal ion which can mitigate the toxicity of metal ions (Barkay *et al.*, 2003). Abioye has reported the use of *B. megaterium, Bacillus subtilis,Aspergillus niger, and Penicillium* for biosorption of heavy metals

like lead, cadmium and chromium from effluent (Abioye *et al*., 2018). There are reports on use of dead biomass of fungus *Aspergillus niger, Rhizopus oryzae, Saccharomyces cerevisiae, and Penicillium chrysogenum* for conversion of toxic Cr (VI) to nontoxic Cr (III) (Park *et al.*, 2005). Biofilm can be used for removal of toxic heavy metals. Phycoremediation, means use of algal biomass for bioremediation. There are various functional groups on the surface of algae which attribute to the metal binding. New concept of use of genetically modified microorganisms for detoxification of heavy metal has been reported by scientist. Bioremediation is ecofriendly and cost-effective mean for detoxification of heavy metals.

Conclusion

In this review we have emphasized on effect of heavy metals generally found in the E waste, Lead, Mercury, Cadmium Arsenic Chromium, Selenium, Nickel, Beryllium, Cobalt, Copper, Barium, on human beings. Negligence to exposure will lead to severe complications because of adverse effect of these metals on our body. Bioremediation is the promising solution to overcome the toxic effect of heavy metals.

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