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TOXICITY OF HEAVY METALS AND ITS MANAGEMENT THROUGH PHYTOREMEDIATION

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Abstract: Heavy metal toxicity has proven to be a major threat and there is several health risks associated with it. The toxic effects of these metals, even though they do not have any biological role, remain present in some or the other form harmful for the human body and its proper functioning. They sometimes act as a pseudo element of the body while at certain times they may even interfere with metabolic processes. Few metals, such as aluminum, can be removed through elimination activities, while some metals get accumulated in the body and food chain, exhibiting a chronic nature. Various public health measures have been undertaken to control, prevent and treat metal toxicity occurring at various levels, such as occupational exposure, accidents and environmental factors. Metal toxicity depends upon the absorbed dose, the route of exposure and duration of exposure, *i.e.* acute or chronic. 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.
Keywords: Free radicals, Heavy metals, Metal toxicity, Oxidative stress.
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INTRODUCTION

Heavy metals are defined as metallic elements that have a relatively high density compared to water (Fergusson, 1990). With the assumption that heaviness and toxicity are inter-related, heavy metals also include metalloids, such as arsenic, that are able to induce toxicity at low level of exposure (Duffus, 2002). In recent years, there has been an increasing ecological and global public health concern associated with environmental contamination by these metals. Also, human exposure has risen dramatically as a result of an exponential increase of their use in several industrial, agricultural, domestic and technological applications (Brad, 2002). Reported sources of heavy metals in the environment include geogenic, industrial, agricultural, pharmaceutical, domestic effluents, and atmospheric sources (He *et al*., 2005). Environmental pollution is very prominent in point source areas such as mining, foundries and smelters, and other metal-based industrial operations. The most commonly found heavy metals in waste water include arsenic, cadmium, chromium, copper, lead, nickel, and zinc, all of which cause risks for human health and the environment (Lambert *et al*., 2000). Although heavy metals are naturally occurring elements that are found throughout the earth's crust, most environmental contamination and human exposure result from anthropogenic activities such as mining and smelting operations, industrial production and use, and domestic and agricultural use of metals and metal-containing compounds (He *et al*., 2005, Goyer, 2001, Herawati *et al*., 2000 and Shallari *et al*.,

1998). Environmental contamination can also occur through metal corrosion, atmospheric deposition, soil erosion of metal ions and leaching of heavy metals, sediment re-suspension and metal evaporation from water resources to soil and ground water (Nriagu, 1989). Natural phenomena such as weathering and volcanic eruptions have also been reported to significantly contribute to heavy metal pollution (Fergusson, 1990; Brad, 2002; He *et al*., 2005; Shallari *et al*., 1998 and Nriagu, 1989). Industrial sources include metal processing in refineries, coal burning in power plants, petroleum combustion, nuclear power stations and high tension lines, plastics, textiles, microelectronics, wood preservation and paper processing plants (Arruti *et al*., 2010 and Pacyna, 1996).

Heavy metals are also considered as trace elements because of their presence in trace concentrations (ppb range to less than 10ppm) in various environmental matrices (Kabata-Pendia, 2001). Their bioavailability is influenced by physical factors such as temperature, phase association, adsorption and sequestration. It is also affected by chemical factors that influence speciation at thermodynamic equilibrium, complexation kinetics, lipid solubility and octanol/water partition coefficients (Hamelink *et al*, 1994). Biological factors such as species characteristics, trophic interactions, and biochemical/physiological adaptation, also play an important role (Verkleji, 1993). Although these metals have crucial biological functions in plants and animals, sometimes their chemical coordination and oxidation-reduction properties have given them an additional benefit so that they can escape control mechanisms such as homeostasis, transport, compartmentalization and binding to required cell constituents. These metals bind with protein sites which are not made for them by displacing original metals from their natural binding sites causing malfunctioning of cells and ultimately toxicity. Previous research has found that oxidative deterioration of biological macromolecules is primarily due to binding of heavy metals to the DNA and nuclear proteins (Flora *et al.*, 2008).

Copper for example serves as an essential co-factor for several oxidative stress-related enzymes including catalase, superoxide dismutase, peroxidase, cytochrome c oxidases, ferroxidases, monoamine oxidase, and dopamine β-monooxygenase (Stern, 2010; Harvey and McArdle, 2008 and ATSDR, 2002). Hence, it is an essential nutrient that is incorporated into a number of metalloenzymes involved in hemoglobin formation, carbohydrate metabolism, catecholamine biosynthesis, and cross-linking of collagen, elastin, and hair keratin. The ability of copper to cycle between an oxidized state, Cu(II) and reduced state, Cu(I), is used by cuproenzymes involved in redox reactions (Stern, 2010; Harvey and McArdle, 2008 and ATSDR, 2002). However, it is this property of copper that also makes it potentially toxic because the transitions between Cu(II) and Cu(I) can result in the generation of superoxide and hydroxyl radicals (Stern, 2010; Harvey and McArdle, 2008; ATSDR, 2002 and Tchounwou *et al.,* 2008). Also, excessive exposure to copper has been linked to cellular damage leading to Wilson disease in humans (ATSDR, 2002 and Tchounwou *et al.*, 2008). Similar to copper, several other essential elements are required for biological functioning; however, an excess amount of such metals produces cellular and tissue damage leading to a variety of adverse effects and human diseases. For some including chromium and copper, there is a very narrow range of concentrations between beneficial and toxic effects (Tchounwou *et al*., 2008 and Chang, 1996). Other metals such as aluminium (Al), antinomy (Sb), arsenic (As), barium (Ba), beryllium (Be), bismuth (Bi), cadmium (Cd), gallium (Ga), germanium (Ge), gold (Au), indium (In), lead (Pb), lithium (Li), mercury (Hg), nickel (Ni), platinum (Pt), silver (Ag), strontium (Sr), tellurium (Te), thallium (Tl), tin (Sn), titanium (Ti), vanadium (V) and uranium (U) have no established biological functions and are considered as non-essential metals (Chang, 1996). Several laboratory studies have demonstrated that reactive oxygen species (ROS) production and oxidative stress play a key role in the toxicity and carcinogenicity of metals such as arsenic

(Yedjou and Tchounwou, 2006; Yedjou and Tchounwou, 2007 and Tchounwou *et al*., 2004), cadmium (Tchounwou *et al*., 2001), chromium (Patlolla *et al.,* 2009 and Patlolla *et al.,* 2009), lead (Yedjou and Tchounwou, 2008 and Tchounwou *et al*., 2004), and mercury (Sutton and Tchounwou, 2007 and Sutton and Tchounwou, 2002). Because of their high degree of toxicity, these five elements rank among the priority metals that are of great public health significance. They are all systemic toxicants that are known to induce multiple organ damage, even at lower levels of exposure. Heavy metal-induced toxicity and carcinogenicity involves many mechanistic aspects, some of which are not clearly elucidated or understood. However, each metal is known to have unique features and physico-chemical properties that confer to its specific toxicological mechanisms of action. This review provides an analysis of the environmental occurrence, production and use, potential for human exposure, and molecular mechanisms of toxicity, genotoxicity, and carcinogenicity of arsenic, cadmium, chromium, lead, and mercury.

Environmental Occurrence, Industrial Production and Use of Arsenic

Arsenic is a ubiquitous element that is detected at low concentrations in virtually all environmental matrices (ATSDR, 2000). Humans may encounter arsenic by natural means, industrial source, or from unintended sources. Drinking water may get contaminated by use of arsenical pesticides, natural mineral deposits or inappropriate disposal of arsenical chemicals. Deliberate consumption of arsenic in case of suicidal attempts or accidental consumption by children may also result in cases of acute poisoning (Mazumder, 2008; Saha *et al*., 1999). Arsenic is a protoplastic poison since it affects primarily the sulphydryl group of cells causing malfunctioning of cell respiration, cell enzymes and mitosis (Gordon and Quastel, 1948). In arsenic biotransformation, harmful inorganic arsenic compounds get methylated by bacteria, algae, fungi and humans to give monomethylarsonic acid (MMA) and dimethylarsinic acid (DMA). In this biotransformation process, these inorganic arsenic species (iAs) are converted enzymatically to methylated arsenicals which are the end metabolites and the biomarker of chronic arsenic exposure.

iAs(V)→iAs(III)→MMA(V)→MMA(III)→DMA(V)

Lead

Lead is a highly toxic metal whose widespread use has caused extensive environmental contamination and health problems in many parts of the world. The sources of lead exposure include mainly industrial processes, food and smoking, drinking water and domestic sources. The sources of lead were gasoline and house paint, which has been extended to lead bullets, plumbing pipes, pewter pitchers, storage batteries, toys and faucets (Thürmer *et al.*, 2002). Lead is an extremely toxic heavy metal that disturbs various plant physiological processes and unlike other metals, such as zinc, copper and manganese, it does not play any biological functions. A plant with high lead concentration fastens the production of reactive oxygen species (ROS), causing lipid membrane damage that ultimately leads to damage of chlorophyll and photosynthetic processes and suppresses the overall growth of the plant. The ionic mechanism of lead toxicity occurs mainly due to the ability of lead metal ions to replace other bivalent cations like Ca^{2+}, Mg^{2+}, Fe^{2+} and monovalent cations like $Na^{+,}$ which ultimately disturbs the biological metabolism of the cell. The ionic mechanism of lead toxicity causes significant changes in various biological processes such as cell adhesion, intra- and inter-cellular signaling, protein folding, maturation, apoptosis, ionic transportation, enzyme regulation, and release of neurotransmitters. Lead can substitute calcium even in picomolar concentration affecting protein kinase C, which regulates neural excitation and memory storage (Flora *et al*., 2012).

Mercury

The metallic mercury is a naturally occurring metal which is a shiny silver-white, odourless liquid and becomes colourless and odourless gas when heated. Mercury is very toxic and exceedingly bioaccumulative. Its

presence adversely affects the marine environment and hence many studies are directed towards the distribution of mercury in water environment. Major sources of mercury pollution include anthropogenic activities such as agriculture, municipal wastewater discharges, mining, incineration, and discharges of industrial wastewater (Chen *et al*., 2012). Mercury exists mainly in three forms: metallic elements, inorganic salts and organic compounds, each of which possesses different toxicity and bioavailability. These forms of mercury are present widely in water resources such as lakes, rivers and oceans where they are taken up by the microorganisms and get transformed into methyl mercury within the microorganism, eventually undergoing biomagnification causing significant disturbance to aquatic lives. Consumption of this contaminated aquatic animal is the major route of human exposure to methyl mercury (Trasande *et al*., 2005). Mercury is extensively used in thermometers, barometers, pyrometers, hydrometers, mercury arc lamps, fluorescent lamps and as a catalyst. It is also being used in pulp and paper industries, as a component of batteries and in dental preparations such as amalgams.

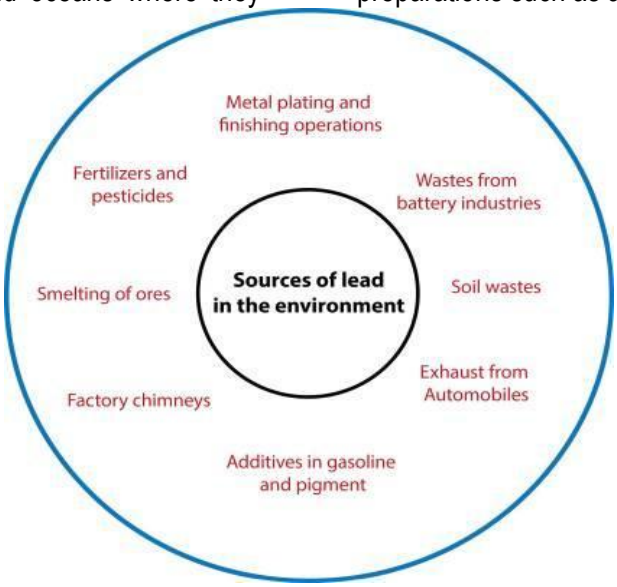

Figure 1. Various sources of Lead Pollution in Environment (Adapted from Sharma & Dubey, 2005)

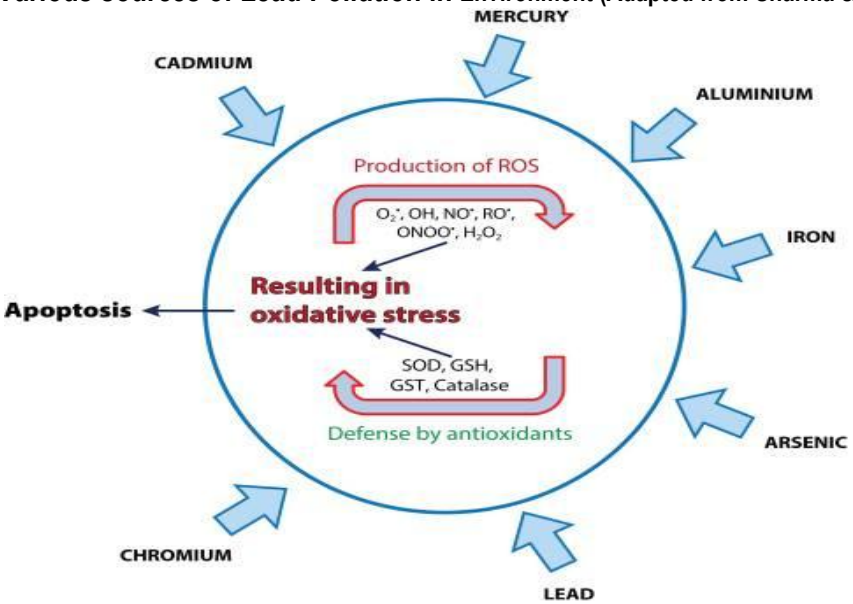

Figure 2. Attack of heavy metals on a cell and the balance between ROS production and the subsequent defense presented by antioxidants

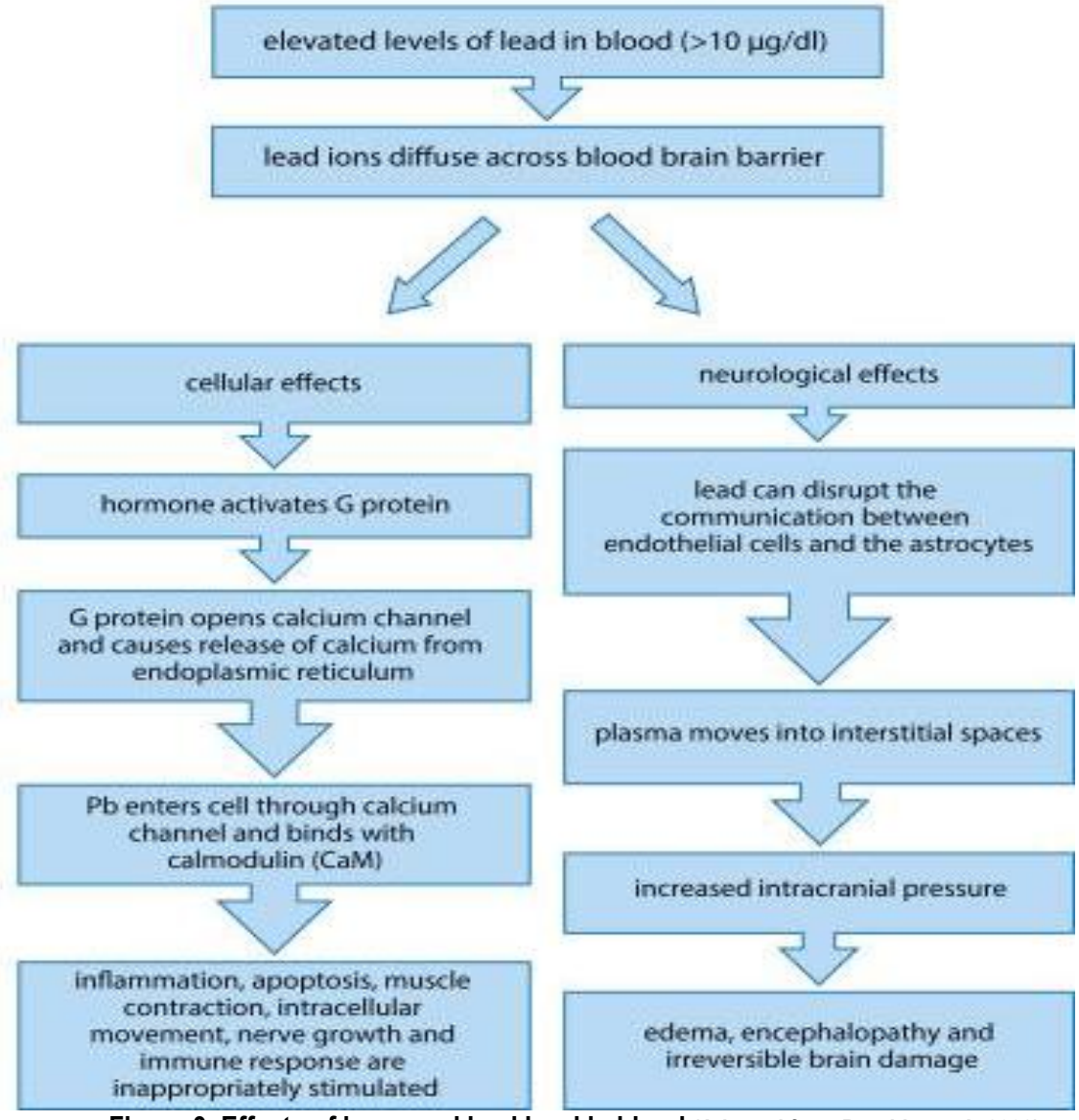

Figure 3. Effects of increased lead level in blood (Adapted from Brochin *et al.*, 2008)

Mechanism of mercury toxicity

Mercury is well known as a hazardous metal and its toxicity is a common cause of acute heavy metal poisoning. Methylmercury is a neurotoxic compound which is responsible for microtubule destruction, mitochondrial damage, lipid peroxidation and accumulation of neurotoxic molecules such as serotonin, aspartate, and glutamate (Patrick, 2002). The brain remains the target organ for mercury, yet it can impair any organ and lead to malfunctioning of nerves, kidneys and muscles. It can cause disruption to the membrane potential and interrupt with intracellular calcium homeostasis. Mercury binds to freely available thiols as the stability constants are high (Patrick, 2002). Mercury vapours can cause bronchitis, asthma and temporary respiratory problems. Mercury plays a key role in damaging the tertiary and quaternary protein structure and alters the cellular function by attaching to the selenohydryl and sulfhydryl groups which undergo reaction with methyl mercury and hamper the cellular structure. It also intervenes with the process of transcription and translation resulting in the disappearance of ribosomes and eradication of endoplasmic reticulum and the activity of natural killer cells. The cellular integrity is also affected causing free radical formation. The basis for heavy metal chelation is that even though the mercury sulfhydryl bond is stable and divided to surrounding sulfhydryl consisting ligands, it

also contributes free sulfhydryl groups to promote metal mobility within the ligands.

Cadmium

Cadmium is the seventh most toxic heavy metal. It is a by-product of zinc production which humans or animals may get exposed to at work or in the environment. Once this metal gets absorbed by humans, it will accumulate inside the body throughout life. This metal was first used in World War I as a substitute for tin and in paint industries as a pigment. In today's scenario, it is also being used in rechargeable batteries, for special alloys production and also present in tobacco smoke. About three-fourths of cadmium is used in alkaline batteries as an electrode component, the remaining part is used in coatings, pigments and platings and as a plastic stabilizer. Humans may get exposed to this metal primarily by inhalation and ingestion and can suffer from acute and chronic intoxications. Cadmium distributed in the environment will remain in soils and sediments for several decades. Plants gradually take up these metals which get accumulated in them and concentrate along the food chain, reaching ultimately the human body.

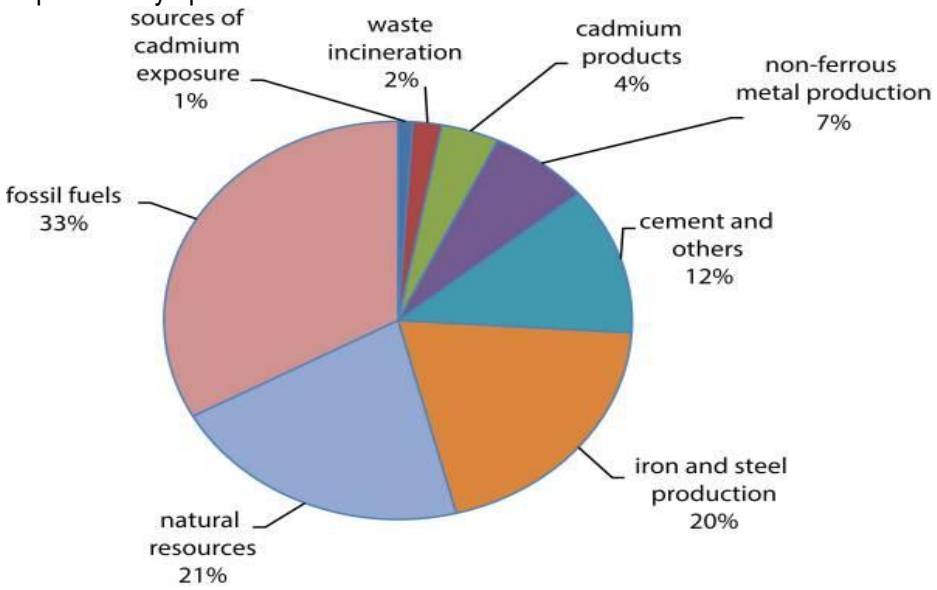

Figure 4. Relative contribution of different sources to human Cadmium exposure (Adapted from Regoli, 2005)

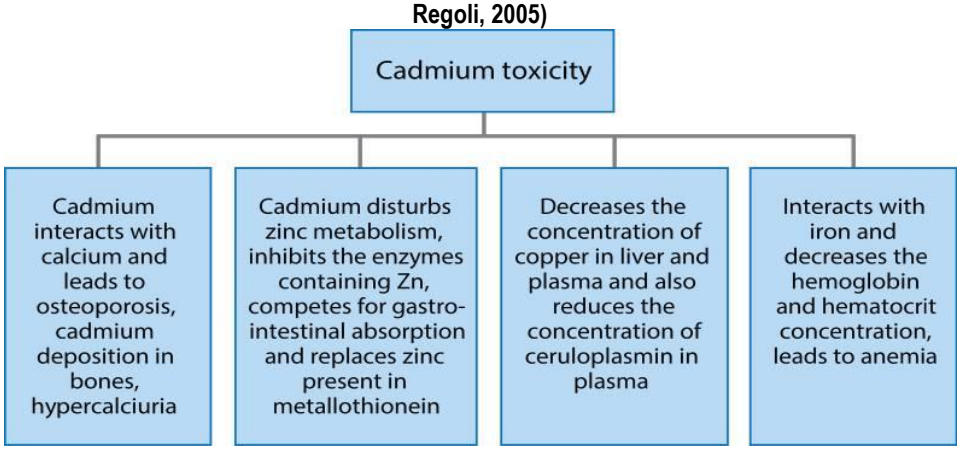

Figure 5. Values of Cadmium toxicity (Adapted from Flora *et al.*, 2008)

Mechanism of cadmium toxicity

The mechanism of cadmium toxicity is not understood clearly but its effects on cells are known (Patrick, 2003). Cadmium concentration increases 3,000 fold when it binds to cystein-rich protein such as metallothionein. In the liver, the cystein-metallothionein complex causes hepatotoxicity and then it circulates to the kidney and gets accumulated in the renal tissue causing nephrotoxicity. Cadmium has the capability to bind with cystein, glutamate, histidine and aspartate ligands and can lead to

the deficiency of iron (Castagnetto *et al.*, 2002). Cadmium and zinc have the same oxidation states and hence cadmium can replace zinc present in metallothionein, thereby inhibiting it from acting as a free radical scavenger within the cell.

Chromium

Chromium is the seventh most abundant element on earth (Mohanty and Kumar Patra, 2013). Chromium occurs in several oxidation states in the environment ranging from Cr^{2+} to Cr^{6+} (Rodríguez *et al*., 2007). The most commonly occurring forms of Cr are trivalent-Cr^{+3} and hexavalent Cr^{+6} , with both states being toxic to animals, humans and plants (Mohanty and Kumar Patra, 2013). Chromium occurs naturally by the burning of oil and coal, petroleum from ferro cromate refractory material, pigment oxidants, catalyst, chromium steel, fertilizers, oil well drilling and metal plating tanneries. Anthropogenically, chromium is released into the environment through sewage and fertilizers (Ghani, 2011). The Cr(III) is immobile in its reduced form and is insoluble in water whereas Cr(VI) in its oxidized state is highly soluble in water and thus mobile (Wolińska *et al*., 2013). In order to determine the activities of the metal ions in the environment, metal speciation is very important where in case of chromium the oxidative form of Cr(III) is not an essential contaminant of the ground water but Cr(VI) has been found to be toxic for humans. Cr(III) resides in the organic matter of soil and aquatic environment in the form of oxides, hydroxides and sulphates (Cervantes *et al*., 2001). Chromium is extensively used in industries such as metallurgy, electroplating, production of paints and pigments, tanning, wood preservation, chemical production and pulp and paper production. These industries play a major role in chromium pollution with an adverse effect on biological and ecological species (Ghani, 2011). A wide range of industrial and agricultural practices increase the toxic level in the environment causing concern about the pollution caused by chromium. Pollution of the environment by chromium, particularly hexavalent chromium, has been the greatest concern in recent years (Zayed and Terry, 2003). Tanneries discharge numerous polluting heavy metals and compounds into the water streams (Nath *et al*., 2008). Due to the presence of excess oxygen in the environment, Cr (III) is oxidized to Cr (VI), which is extremely toxic and highly soluble in water (Cervantes *et al*., 2001). In India, the chromium level in underground water has been witnessed to be more than 12 mg/L and 550–1,500 ppm/L. The mechanism of ultrastructural organization, biochemical changes and metabolic regulations has not been clarified since the process of phytotoxicity in the aquatic environment by chromium has not been concentrated on in detail (Chandra and Kulshreshtha, 2004).

Mechanism of Chromium Toxicity

In the environment, trivalent chromium Cr(III) is generally harmless due to its weak membrane permeability. Hexavalent chromium Cr(VI), on the other hand, is more active in penetrating the cell membrane through passages for isoelectric and isostructural anions such as SO_4^{2-} and HPO_4^{2-} channels and these chromates are taken up through phagocytosis. Cr(VI) is a strong oxidizing agent and can be reduced to give ephemeral species of pentavalent and tetravalent chromium that are different from that of Cr(III). Stabilization of the pentavelent form is carried out by glutathione and hence intracellular reduction of Cr[VI] is considered a detoxification mechanism when reduction occurs away from the target region. However if intracellular reduction of Cr[VI] occurs near the target site, it may serve to activate Cr. The reactions between Cr(VI) and biological reductants like thiols and ascorbate result in the production of reactive oxygen species such as superoxide ion, hydrogen peroxide, and hydroxyl radical, ultimately leading to oxidative stress in the cell causing damage to DNA and proteins (Stohs and Bagchi, 1995). According to literature surveys, Cr(VI) has been found to be much more dangerous than Cr(III), since Cr(VI) enters the cells more readily than does Cr(III) and is eventually reduced to Cr(III). Because of its mutagenic properties, Cr(VI) is categorized as a group 1 human carcinogen by the International Agency for the Research on Cancer.

Aluminum

Aluminum is the third most abundant element found in the earth's crust (Gupta *et al*., 2013). Aluminum occurs naturally in the air, water and soil. Mining and processing of aluminum elevates its level in the environment. Recent investigations on environmental toxicology revealed that aluminum may present a major threat for humans, animals and plants in causing many diseases (Barabasz *et al*., 2002). Many factors, including pH of water and organic matter content, greatly influence the toxicity of aluminium. With decreasing pH its toxicity increases in the city and distracts the physical and cellular processes in plants. The Enzymes such as hexokinase, phosphodiesterase, alkali phosphatase and phosphoxidase are inhibited by aluminium since it has a greater affinity to DNA and RNA. Metabolic pathways in the living organism involving calcium, phosphorous, fluorine and iron metabolism are affected by aluminium. Aluminium has been found to be very harmful to nervous, osseous and hemopoietic cells (Barabasz *et al*., 2002).

Mechanism of Aluminum toxicity

Aluminum interferes with most physical and cellular processes. The exact mechanism of absorption of aluminum by the gastrointestinal tract is not understood completely. Based on literature surveys, it is difficult to give a proper time period for aluminum toxicity since some symptoms of aluminum toxicity can be detected in seconds and others in minutes after exposure to aluminum (WHO, 1997). Aluminum toxicity probably results from the interaction between aluminum and plasma membrane, apoplastic and symplastic targets (Kochian *et al*., 2005). In humans Mg^{2+} and Fe^{3+} are replaced by Al^{3+}, which causes many disturbances associated with intercellular communication, cellular growth and secretory functions. The changes that are evoked in neurons by aluminum are similar to the degenerative lesions observed in Alzheimer patients. The greatest complications of aluminum toxicity are neurotoxicity effects such as neuronal atrophy in the locus ceruleus, substantia nigra and striatum.

Iron

Iron is the second most abundant metal on the earth's crust (EPA, 1993). Iron occupies the 26th elemental position in the periodic table. Iron is a most crucial element for growth and survival of almost all living organisms (Valko *et al*., 2005). It is one of the vital components of organisms like algae and of enzymes such as cytochromes and catalase, as well as of oxygen transporting proteins, such as hemoglobin and myoglobin (Vuori, 1995). Iron is an attractive transition metal for various biological redox processes due to its inter-conversion between ferrous (Fe^{2+}) and ferric (Fe^{3+}) ions. The source of iron in surface water is anthropogenic and is related to mining activities. The production of sulphuric acid and the discharge of ferrous (Fe^{2+}) takes place due oxidation of iron pyrites (FeS_2) that are common in coal seams (Valko *et al*., 2005). The following equations represent the simplified oxidation reaction for ferrous and ferric iron:

$$2FeS_2 + 7O_2 \rightarrow 2FeSO_4 + H_2SO_4 \text{ (ferrous)}$$
$$4FeSO_4 + O_2 + 10H_2O \rightarrow 4Fe(OH)_3 + 4H_2SO_4 \text{ (ferric)}$$

The concentration of dissolved iron in the deep ocean is normally 0.6 nM or 33.5×10^{-9} mg/L. In freshwater the concentration is very low with a detection level of 5 µg/L – ICP, whereas in groundwater the concentration of dissolved iron is very high with 20 mg/L (EPA, 1993). Acid soils restrict rice production and together with Zn deficiency cause a macronutrient disorder in wetland rice. The production of lowland rice was greatly affected by high concentrations of reduced iron (Fe^{2+}) in the flooded soils. The features of iron toxicity in rice include high uptake of Fe^{2+} by roots, acropetal translocation into leaves, bronzing of rice leaves and yield loss (Becker and Asch, 2005).

Mechanism of Iron Toxicity

A wide range of harmful free radicals are formed when the absorbed iron fails to bind to the protein, which in turn severely affects the concentration of iron in mammalian cells and biological fluids. This circulating unbound iron results in corrosive effect of the gastrointestinal tract and biological fluids. An extremely higher level of iron enters into the body crossing the

rate-limiting absorption step and becomes saturated. These free irons penetrate into cells of the heart, liver and brain. Due to the disruption of oxidative phosphorylation by free iron, the ferrous iron is converted to ferric iron that releases hydrogen ions, thus increasing metabolic acidity. The free iron can also lead to lipid peroxidation, which results in severe damage to mitochondria, microsomes and other cellular organelles (Albretsen, 2006). The toxicity of iron on cells has led to iron mediated tissue damage involving cellular oxidizing and reducing mechanisms and their toxicity towards intracellular organelles such as mitochondria and lysosomes. A wide range of free radicals that are believed to cause potential cellular damage are produced by excess intake of iron. The iron produced hydrogen free radicals attack DNA, resulting in cellular damage, mutation and malignant transformations which in turn cause an array of diseases (Grazuleviciene *et al*., 2009).

Management of Contaminated Soil

Soil and crop management methods can help prevent uptake of pollutants by plants, leaving them in the soil. The soil becomes the sink, breaking the soil-plant animal or human cycle through which the toxin exerts its toxic effects. The following management practices will not remove the heavy metal contaminants, but will help to immobilize them in the soil and reduce the potential for adverse effects from the metals. Note that the kind of metal (cation or anion) must be considered:

i. Increasing the soil pH to 6.5 or higher: Cationic metals are more soluble at lower pH levels, so increasing the pH make them less available to plants and therefore less likely to be incorporated in their tissues and ingested by humans. Raising pH has the opposite effect on anionic elements.

ii. Draining wet soils: Drainage improves soil aeration and will allow metals to oxidize, making them less soluble. Therefore when aerated, these metals are less available. The opposite is true for chromium, which is more available in oxidized forms. Active organic matter is effective in reducing the availability of chromium.

iii. Applying phosphate: Heavy phosphate applications reduce the availability of cationic metals, but have the opposite effect on anionic compounds like arsenic. Care should be taken with phosphorus applications because high levels of phosphorus in the soil can result in water pollution.

iv. Carefully selecting plants for use on metal-contaminated soils: Plants translocate larger quantities of metals to their leaves than to their fruits or seeds. The greatest risk of food chain contamination is in leafy vegetables like lettuce or spinach. Another hazard is forage eaten by livestock.

Plants for Environmental Cleanup

Research has demonstrated that plants are effective in cleaning up contaminated soil (Wenzel *et al.,* 1999). Phytoremediation is a general term for using plants to remove, degrade, or contain soil pollutants such as heavy metals, pesticides, solvents, crude oil, polyaromatic hydrocarbons, and landfill leacheates For example, prairie grasses can stimulate breakdown of petroleum products. Wildflowers were recently used to degrade hydrocarbons from an oil spill in Kuwait. Hybrid poplars can remove ammunition compounds such as TNT as well as high nitrates and pesticides.

Plants for Treating Metal Contaminated Soils

Plants have been used to stabilize or remove metals from soil and water. The three mechanisms used are *phytoextraction*, *rhizofiltration*, and *phytostabilization*. Here we are defining rhizofiltration and phytostabilization but will mainly focus on phytoextraction. Rhizofiltration is the adsorption onto plant roots or absorption into plant roots of contaminants that are in solution surrounding the root zone (rhizosphere). Rhizofiltration is used to decontaminate groundwater. Plants are grown in greenhouses in water instead of soil. Contaminated water from the site is used to acclimate the plants to the environment. The plants are then planted on the site of contaminated ground water where the roots take up the water and contaminants. Once the roots are saturated with the contaminant, the

plants are harvested including the roots. In Chernobyl, Ukraine, sunflowers were used in this way to remove radioactive contaminants from groundwater. Phytostabilization is the use of perennial, non-harvested plants to stabilize or immobilize contaminants in the soil and groundwater. Metals are absorbed and accumulated by roots, adsorbed onto roots, or precipitated within the rhizosphere. Metal-tolerant plants can be used to restore vegetation where natural vegetation is lacking, thus reducing the risk of water and wind erosion and leaching. Phytostabilization reduces the mobility of the contaminant and prevents further movement of the contaminant into groundwater or the air and reduces the bioavailability for entry into the food chain.

Phytoextraction

Phytoextraction is the process of growing plants in metal contaminated soil. Plant roots then translocate the metals into aboveground portions of the plant. After plants have grown for some time, they are harvested and incinerated or composted to recycle the metals. Several crop growth cycles may be needed to decrease contaminant levels to allowable limits. If the plants are incinerated, the ash must be disposed of in a hazardous waste landfill, but the volume of the ash is much smaller than the volume of contaminated soil if dug out and removed for treatment. Phytoextraction is done with plants called hyperaccumulators, which absorb unusually large amounts of metals in comparison to other plants. Hyperaccumulators contain more than 1,000 milligrams per kilogram of cobalt, copper, chromium, lead, or nickel; or 10,000 milligrams per kilogram (1 %) of manganese or zinc in dry matter (Baker and Brooks, 1989). One or more of these plant types are planted at a particular site based on the kinds of metals present and site conditions.

CONCLUSION

Phytoremediation has been studied extensively in research and small-scale demonstrations, but in only a few full-scale applications. Phytoremediation is moving into the realm of commercialization. Given the current effectiveness, phytoremediation is best suited for cleanup over a wide area in which contaminants are present at low to medium concentrations. For phytoremediation to get fully commercialized, further research is needed to assure that tissues of plants used for phytoremediation do not have adverse environmental effects if eaten by wildlife or used by humans for things such as mulch or firewood. Research is also needed to find more efficient bioaccumulators, hyperaccumulators that produce more biomass, and to further monitor current field trials to ensure a thorough understanding. There is the need for a commercialized smelting method to extract the metals from plant biomass so they can be recycled. Phytoremediation is slower than traditional methods of removing heavy metals from soil but much less costly. Prevention of soil contamination is far less expensive than any kind of remediation and much better for the environment.

REFERENCES

Agency for Toxic Substances and Disease Registry (ATSDR). (2002). Toxicological Profile for Copper. Atlanta, GA: Centers for Disease Control.

Agency for Toxic Substances and Disease Registry (ATSDR). (2000). Toxicological Profile for Arsenic TP-92/09. Georgia: Center for Disease Control, Atlanta.

Albretsen J. (2006). The toxicity of iron, an essential element; Veterinary medicine; pp. 82–90.

Arruti A, Fernández-Olmo I, Irabien A. (2010). (Evaluation of the contribution of local sources to trace metals levels in urban PM2.5 and PM10 in the Cantabria region (Northern Spain) *J Environ Monit.* 12(7):1451–1458.

Baker, A.J.M., and R.R. Brooks. (1989). Terrestrial plants which hyperaccumulate metallic elements – a review of their distribution, ecology and phytochemistry. Biorecovery 1:81:126.

Barabasz W, Albinska D, Jaskowska M, Lipiec J. (2002). Ecotoxicology of Aluminium. *Pol J Environ Stud*. 11(3):199–203.

Becker M, Asch F. (2005). Iron toxicity in rice–conditions and management concepts. *J Plant Nutr Soil Sci.* 168:559–553.

Brochin R, Leone S, Phillips D, Shepard N, Zisa D, Angerio A. (2008). The cellular effect of lead poisoning and its clinical picture. GUJHS. 5(2):1–8.

Castagnetto JM, Hennessy SW, Roberts VA, Getzoff ED, Tainer JA, Pique ME. (2002). MDB: the metalloprotein database and browser at the Scripps Research Institute. *Nucleic Acids Res.* 30(1):379–382.

Cervantes C, Campos-García J, Devars S, Gutiérrez-Corona F, Loza-Tavera H, Torres-Guzmán JC, Moreno-Sánchez R.(2001). Interactions of chromium with microorganisms and plants. *FEMS Microbiol Rev*. 25(3):335–347.

Chandra P, Kulshreshtha K. (2004). Chromium accumulation and toxicity in aquatic vascular plants. *Botanical Rev*. 70(3):313–327.

Chen CW, Chen CF, Dong CD. (2012). Distribution and Accumulation of Mercury in Sediments of Kaohsiung River Mouth, Taiwan. APCBEE *Procedia*. 1:153–158.

Duffus JH. (2002). Heavy metals-a meaningless term? *Pure Appl Chem*. 74(5):793–807.

Fergusson JE, (1990). The Heavy Elements: Chemistry, Environmental Impact and Health Effects. Oxford: Pergamon Press.

Flora SJS, Mittal M, Mehta A. (2008). Heavy metal induced oxidative stress and its possible reversal by chelation therapy. *Indian J Med Res*. 128:501–523.

Ghani A. (2011). Effect of chromium toxicity on growth, chlorophyll and some mineral nutrients of *Brassica juncea* L. *Egyptian Acad J Biol Sci*. 2(1):9–15.

Gordon JJ, Quastel GH. (1948). Effect of organic arsenicals on enzyme system. Biochem J. 42:337–350.

Goyer RA. (2001). Toxic effects of metals. *In:* Klaassen CD, editor. Cassarett and Doull's Toxicology: The Basic Science of Poisons. New York: McGraw-Hill Publisher; pp. 811–867.

Grazuleviciene R, Nadisauskiene R, Buinauskiene J, Grazulevicius T. (2009). Effects of Elevated Levels of Manganese and Iron in Drinking Water on Birth Outcomes. *Polish J of Environ Stud.* 18(5):819–825.

Gupta N, Gaurav SS, Kumar A. (2013). Molecular Basis of Aluminium Toxicity in Plants: A Review. *Am J of Plant Sci.* 4:21–37.

Hamelink JL, Landrum PF, Harold BL, William BH, editors. (1994). Bioavailability: Physical, Chemical, and Biological Interactions. Boca Raton, FL: CRC Press Inc.

Harvey LJ, McArdle HJ. (2008). Biomarkers of copper status: a brief update. *Br J Nutr.* 99(S3):S10–S13.

He ZL, Yang XE, Stoffella PJ. (2005). Trace elements in agroecosystems and impacts on the environment. *J Trace Elem Med Biol.* 19(2–3):125–140.

Herawati N, Suzuki S, Hayashi K, Rivai IF, Koyoma H. (2000). Cadmium, copper and zinc levels in rice and soil of Japan, Indonesia and China by soil type. *Bull Env Contam Toxicol*. 64:33–39.

Kabata- Pendia A. (2001). Trace Elements in Soils and Plants. Boca Raton, FL: CRC Press.

Kochian LV, Piñeros MA, Hoekenga OA. (2005). The physiology, genetics and molecular biology of plant aluminum resistance and toxicity. *Plant and Soil*. 274:175–195.

Mathew BB, Tiwari A, Jatawa SK.(2011). Free radicals and antioxidants: A review. *Journal of Pharmacy Research.* 4(12):4340–4343.

Mazumder G. (2008). Chronic arsenic toxicity and human health. *Indian J Med Res*. 128(4):436–447.

Mohanty M, Kumar Patra H. (2013). Effect of ionic and chelate assisted hexavalent chromium on mung bean seedlings (Vigna Radiata l. Wilczek. Var k-851) during seedling growth. JSPB. 9(2):232–241.

Nath K, Shyam S, Singh D, Shanna YK. (2008). Effect of chromium and tannery effluent toxicity on metabolism and growth in cowpea (Vigna sinensis L. Saviex Hassk) seedling. *Res Environ Life Sci.* 1:91–94.

Nriagu JO. (1989). A global assessment of natural sources of atmospheric trace metals. *Nature*. 338:47–49.

Pacyna JM. (1996). Monitoring and assessment of metal contaminants in the air. In: Chang LW, Magos L, Suzuli T, editors. Toxicology of Metals. Boca Raton, FL: CRC Press. pp. 9–28.

Patlolla A, Barnes C, Field J, Hackett D, Tchounwou PB. (2009). Potassium dichromate-induced cytotoxicity, genotoxicity and oxidative stress in human liver carcinoma (HepG2) cells. *Int J Environ Res Public Health*. 6:643–653.

Patlolla A, Barnes C, Yedjou C, Velma V, Tchounwou PB. (2009). Oxidative stress, DNA damage and antioxidant enzyme activity induced by hexavalent chromium in Sprague Dawley rats. *Environ Toxicol.* 24(1):66–73.

Patrick L. (2002). Mercury toxicity and antioxidants: Part 1: role of glutathione and alpha-lipoic acid in the treatment of mercury toxicity. *Altern Med Rev.* 7(6):456–471.

Regoli L. (2005). The Relative Contributions of Different Environmental Sources to Human Exposure and the EU Cadmium Risk Assessment Meeting of UNECE Task Force on Heavy Metals. Presentation for the UNECE Long-Range-Trans-boundary Air Pollutants – Task Force on Heavy Metals; 16–18 March, Berlin.

Rodríguez MC, Barsanti L, Passarelli V, Evangelista V, Conforti V, Gualtieri P. (2007). Effects of chromium on photosynthetic and photoreceptive apparatus of the alga *Chlamydomonas reinhardtii*. *Environ Res.* 105(2):234–239.

Saha JC, Dikshit AK, Bandyopadhyay M, Saha KC. (1999). A review of arsenic poisoning and its effects on human health. *Crit Rev Env Sci Technol.* 29(3):281–313.

Shallari S, Schwartz C, Hasko A, Morel JL.(1998). Heavy metals in soils and plants of serpentine and industrial sites of Albania. *Sci Total Environ*. 19209:133–142.

Sharma P, Dubey RS.(2005). Lead toxicity in plants. *Brazilian Journal of Plant Physiology*. 17(1):35–52.

Stern BR. (2010). Essentiality and toxicity in copper health risk assessment: overview, update and regulatory considerations. Toxicol Environ Health A. 73(2):114–127.

Stohs SJ, Bagchi D. (1995). Oxidative mechanisms in the toxicity of metal ions. *Free Radic Biol Med.* 18(2):321–336.

Sutton D, Tchounwou PB, Ninashvili N, Shen E.(2002). Mercury induces cytotoxicity, and transcriptionally activates stress genes in human liver carcinoma cells. *Intl J Mol Sci.* 3(9):965–984.

Sutton DJ, Tchounwou PB. (2007). Mercury induces the externalization of phosphatidylserine in human proximal tubule (HK-2) cells. *Intl J Environ Res Public Health.* 4(2):138–144.

Tchounwou P, Newsome C, Williams J, Glass K. (2008). Copper-induced cytotoxicity and transcriptional activation of stress genes in human liver carcinoma cells. *Metal Ions Biol Med.* 10:285–290.

Tchounwou PB, Centeno JA, Patlolla AK. (2004). Arsenic toxicity, mutagenesis and carcinogenesis - a health risk assessment and management approach. *Mol Cell Biochem.* 255:47–55.

Tchounwou PB, Ishaque A, Schneider J. (2001). Cytotoxicity and transcriptional activation of stress genes in human liver carcinoma cells (HepG2) exposed to cadmium chloride. *Mol Cell Biochem*. 222:21–28.

Tchounwou PB, Yedjou CG, Foxx D, Ishaque A, Shen E. (2004). Lead-induced cytotoxicity and transcriptional activation of stress genes in human liver carcinoma cells (HepG2) *Mol Cell Biochem*. 255:161–170.

Thürmer K, Williams E, Reutt-Robey J. (2002). Autocatalytic oxidation of lead crystallite surfaces. *Science*. 297(5589):2033–2035.

Trasande L, Landrigan PJ, Schechter C. (2005). Public health and economic consequences of methyl mercury toxicity to the developing brain. *Environ Health Perspect.* 113(5):590–596.

U.S. EPA. (1993). Standard Methods for the Examination of Water and Wastewater; US: American Public Health Assoc.

Valko MMHCM, Morris H, Cronin MTD. (2005). Metals, toxicity and oxidative stress. *Curr Med Chem*. 12(10):1161–1208.

Verkleji JAS. (1993). The effects of heavy metals stress on higher plants and their use as biomonitors *In*: Plant as Bioindicators: Indicators of Heavy Metals in the Terrestrial Environment. Markert B, editor. New York: VCH; pp. 415–424.

Vuori K-M. (1995). Direct and Indirect effects of iron on river eco systems. *Annal Zoo Fennici.* 32:317–329.

Wadhwa N, Mathew BB, Jatawa S, Tiwari A. (2012). Lipid peroxidation: mechanism, models and significance. *Int J Curr Sci*. 3:29–38.

Wenzel, W.W., Adriano, D.C., Salt, D., and Smith, R. (1999). Phytoremediation: A plant-microbe based remediation system. p. 457-508. In D.C. Adriano et al. (ed.) Bioremediation of contaminated soils. American Society of Agronomy, Madison, WI.

WHO. (1997). Aluminium; Geneva: World Health Organization, International Programme on Chemical Safety (Environmental Health Criteria 194).

Wolińska A, Stępniewska Z, Włosek R. (2013). The influence of old leather tannery district on chromium contamination of soils, water and plants. *Nat Sci.* 5(2A):253–258.

Yedjou CG, Tchounwou PB.(2006). Oxidative stress in human leukemia cells (HL-60), human liver carcinoma cells (HepG2) and human Jerkat-T cells exposed to arsenic trioxide. *Metal Ions Biol Med.* 9:298–303.

Yedjou GC, Tchounwou PB. (2007). *In vitro* cytotoxic and genotoxic effects of arsenic trioxide on human leukemia cells using the MTT and alkaline single cell gel electrophoresis (comet) assays. *Mol Cell Biochem*. 301:123–130.

Yedjou GC, Tchounwou PB. (2008). N-acetyl-cysteine affords protection against lead-induced cytotoxicity and oxidative stress in human liver carcinoma (HepG2) cells. *Intl J Environ Res Public Health*. 4(2):132–137.

Zayed AM, Terry N. (2003). Chromium in the environment: factors affecting biological remediation. *Plant Soil.* 249(1):139–156.

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