

ACCUMULATION OF HEAVY METALS IN RESOURCES OF LIVING AND ITS HAZARDOUS EFFECT ON HUMAN HEALTH

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ABSTRACT

Heavy metals not only have major impacts on aquatic organisms, but also seriously threaten human health. However. the current environmental criteria refer to the maximum value limitations of environmental factors in environmental media where harmful or detrimental effects are not produced on specific protected objects. These metals causes many health aspects with all organisms like human, plants, animals and even microbes. They causes acute as well as chronic diseases. This research deals with the effect of hazardous heavy metals on human This includes new health and plants. mechanism of phytoremediation to reduce the rate of these metals from the soil and water. Heavy metals constitute a group of about 40 elements with a density greater than five (Passow et al. 1961). 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 (Järup, 2003). Heavy metals have many adverse health effects and last for a long period of time, heavy metal exposure continues and is increasing in many parts of the world.

INTRODUCTION

Our environment get polluted by different types of particles which damages our internal metabolic rules of the body and also causes various diseases which are non curable upto now.These metals come in the food chain and get biomagnified.In living organisms including humans, metals and other chemical toxicants bio accumulate as a result of consuming contaminated drinking water and food, particularly fish and molluscs, with toxicant concentrations increasing as they pass along the food chain (Gruber 1989). Metal ions must be bioavailable (i.e. able to move across biological membranes) before they can initiate an adverse effect (Batley et al. 2004), and low pH typically increases metal bioavailability (Hyne et al. 2005). Double-strand RNA (dsRNA) has 11 base pairs (BPs) per turn (compared to 10 in β -DNA) with its bases tilted 30° with respect to the helical axis (Lescrinier et al. 2003).Under conditions environmental with high concentrations of heavy metals, the ingestion of excessive amounts of heavy metals will lead to effects with possible poisoning serious consequences .Metal toxicity depends upon the absorbed dose, the route of exposure and duration of exposure, i.e. acute or chronic.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 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). Multiple heavy metals exist

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in the earth's crust, and the myriad undertakings of human activities results in practically everyone being exposed to these elements in the air, water, and food supply

Anjum et al.,2014 reported that Most plants have gradually formed an avoidance mechanism and tolerance mechanism in the heavy metal stress environment. Avoidance mechanisms include plants affecting mobility of heavy metals and microbial activity through root exudates, fine isolation and regionalization of cell walls, cell membranes and vacuoles.

METHOD

Heavy metals, with soil residence times of subject of study are Arsenic, I thousands of years, pose numerous health Cadmium, Chromium, Aluminium Table representing heavy metals recently found in huge amount in our surroundings-

dangers to higher organisms. They are also known to have effect on plant growth, ground cover and have a negative impact on soil microflora. Heavy metals disrupt metabolic functions in two ways: They accumulate and thereby disrupt function in vital organs and glands such as the heart, brain, kidneys, bone, liver, etc. They displace the vital nutritional minerals from their original place, and copy their biological function. The aim of study is the collection of review regarding heavy metal effect on human health. Seven most dangerous heavy metals are taken granted as the main subject of study are Arsenic, Lead, Mercury, Cadmium, Chromium, Aluminium & Iron.

HEAVY METAL	EFFECT ON ANIMAL	REFERENCES
Arsenic	Carcinogenic	Singh <i>et al.</i> , 2007
Lead	birth defects, psychosis,	Martin & Griswold, 2009
	autism	
Mercury	Neurotoxic	Patrick, 2002
Cadmium	Premature birth	Henson & Chedrese, 2004
Chromium	damage to DNA and proteins	Stohs & Bagchi, 1995
Aluminium	Alzheimer disease	WHO, 1997.
Iron	oxidation of DNA molecules	Bhasin et al., 2002

Arsenic

Arsenic is one of the most important heavy metals causing disquiet from both ecological and individual health standpoints (Hughes et al., 1988). It has a semimetallic property, is prominently toxic and carcinogenic, and is extensively available in the form of oxides or sulfides or as a salt of iron, sodium, calcium, copper, etc. (Singh et al., 2007).In arsenic biotransformation, harmful inorganic arsenic compounds get methylated by bacteria, algae, fungi and humans to give monomethylarsonic (MMA) and dimethylarsinic acid acid (DMA)Monomethylarsonic acid (MMA III), an intermediate product, is found to be highly toxic compared to other arsenicals, potentially accountable for arsenic-induced carcinogenesis (Singh et al., 2007). Most of the reports of chronic arsenic toxicity in man focus on skin manifestations because of its specificity in diagnosis. Pigmentation and keratosis are the

specific skin lesions that indicate chronic arsenic toxicity (Martin & Griswold, 2009). Lower levels of arsenic exposure can cause nausea and vomiting, reduced production of erythrocytes and leukocytes, abnormal heart beat, pricking sensation in hands and legs, and damage to blood vessels.

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 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).Some research revealed that lead is capable of inhibiting the growth of tea plant by reducing biomass and debases the tea quality by changing the quality of its components (Yongsheng *et al.*, 2011). Lead metal causes toxicity in living cells by

ionic mechanism and following that of oxidative stress. Many researchers have shown that oxidative stress in living cells is caused by the imbalance between the production of free radicals and the generation of antioxidants to detoxify the reactive intermediates or to repair the resulting damage.Under the influence of lead, however, the level of the ROS increases and the level of antioxidants decreases. At very high concentrations, ROS may cause structural damage to cells, proteins, nucleic acid, membranes and lipids, resulting in a stressed situation at cellular level (Mathew et al., 2011). The ionic mechanism of lead toxicity occurs mainly due to the ability of lead metal ions to replace other bivalent cations like Ca²⁺, Mg²⁺, Fe²⁺ and monovalent cations like Na^{+,} which ultimately disturbs the biological metabolism of the cell.

Toxicity of lead, also called lead poisoning, can be either acute or chronic. Acute exposure can cause loss of appetite, headache, hypertension, abdominal pain, renal dysfunction, fatigue, sleeplessness, arthritis, hallucinations and vertigo. Chronic exposure of lead can result in mental retardation, birth defects, psychosis, autism, allergies, dyslexia, weight loss, hyperactivity, paralysis, muscular weakness, brain damage, kidney damage and may even cause death (Martin & Griswold, 2009)

Mercury

The metallic mercury is a naturally occurring metal which is a shiny silver-white, odorless liquid and becomes colorless and odorless gas when heated.Consumption of this contaminated aquatic animal is the major route of human exposure to methyl mercury (Trasande et al., 2005. Methylmercury is a neurotoxic compound which is responsible for microtubule mitochondrial damage, lipid destruction, peroxidation and accumulation of neurotoxic molecules such as serotonin, aspartate, and glutamate (Patrick, 2002). Animals which are exposed to toxic mercury have shown adverse neurological and behavioral changes. Rabbits when exposed to 28.8 mg/m³ mercury vapor for 1 to 13 weeks have shown vague pathological changes, marked cellular degeneration and brain necrosis (Ashe et al., 1953). 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.

Exposure to metallic mercury vapors at higher levels for shorter periods of time can lead to lung damage, vomiting, diarrhea, nausea, skin rashes, increased heart rate or blood pressure. Symptoms of organic mercury poisoning include depression, memory problems, tremors, fatigue, headache, hair loss, *etc.* Since these symptoms are common also in other conditions, it may be difficult to diagnose such cases (Martin & Griswold, 2009).

Cadmium

Cadmium is the seventh most toxic heavy metal as per ATSDR ranking. 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.In the US, more than 500,000 workers get exposed to toxic cadmium each year as per The Agency for Toxic Substances and Disease Registry (Bernard, 2008; Mutlu et al., 2012).In Japan and China, environmental cadmium exposure is comparatively higher than in any other country (Han et al., 2009). Cadmium concentration increases 3,000 fold when it binds to cystein-rich protein such as metallothionein. liver. the cystein-metallothionein In the complex causes hepatotoxicity and then it circulates to the kidney and gets accumulated in renal tissue causing the nephrotoxicity.Premature birth and reduced birth weights are the issues that arise if cadmium exposure is high during human pregnancy (Henson & Chedrese, 2004). Cadmium can cause both acute and chronic intoxications (Chakraborty *et al.*, 2013). Cadmium is highly toxic to the kidney and it accumulates in the proximal tubular cells in higher concentrations.

Chromium

Chromium is the seventh most abundant element on earth (Mohanty & Kumar Patra, 2013). Chromium occurs in several oxidation states in the environment ranging from Cr^{2+} to Cr^{6+} (Rodríguez *et al.*, 2009). 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). Pollution of the environment by chromium, particularly hexavalent chromium, has been the greatest concern in recent years (Zayed & Terry, 2003) Chromium toxicity greatly affects the biological processes in various plants such as maize, wheat, barley, cauliflower, citrullus and in vegetables. Chromium toxicity causes chlorosis and necrosis in plants (Ghani, 2011)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. 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 & Bagchi, 1995).

Schroeder *et al.* (<u>1970</u>) reported that cigarettes contained 390 g/kg of Cr, but there has been no significant report published on the amount of chromium inhaled through smoking.

Aluminium

Aluminium is the third most abundant element found in the earth's crust (Gupta et al., 2013). Aluminium occurs naturally in the air, water and soil. Mining and processing of aluminium elevates its level in the environment (ATSDR, 2010). Recent investigations on environmental toxicology revealed that aluminium may present a major threat for humans, animals and plants in causing many diseases (Barabasz et al., 2002). Aluminium has no biological role and is a toxic nonessential metal to microorganisms (Olaniran et al., 2013). Enzymes such as hexokinase, phosphodiesterase, alkalic 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 aluminiumAluminium interferes with most physical and cellular processes. The exact mechanism of absorption of aluminium by the gastrointestinal tract not understood is completely.Aluminium exposure is probably a risk factor for the onset of Alzheimer disease (AD) in humans, as hypothesized by the WHO, 1997.Aluminium toxicity probably results from the interaction between aluminium 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.

Iron

Iron is the second most abundant metal on the earth's crust. Iron occupies the 26^{th} 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). The production of lowland rice was greatly affected by high concentrations of reduced iron (Fe²⁺) in the flooded soils. The features of iron toxicity in rice include high uptake of Fe²⁺ by roots, acropetal translocation into leaves, bronzing of rice leaves and yield loss (Becker & Asch, 2005). The following equations represent the simplified oxidation reaction for ferrous and ferric iron (Phippen *et al.*, 2008):

 $\begin{array}{l} 2FeS_2 + 7O_2 \rightarrow 2FeSO_4 + H_2SO_4(ferrous) \\ 4FeSO_4 & + O_2 & + 10H_2O \rightarrow 4Fe(OH)_3 \\ + 4H_2SO_4(ferric) \end{array}$

In freshwater the concentration is very low with a detection level of 5 μ g/L – ICP, whereas in groundwater the concentration of dissolved iron is very high with 20 mg/L .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). Workers who are highly exposed to asbestos that contains almost 30% of iron are at high risk of asbestosis, which is the second most important cause for lung cancer (Nelson, 1992). It is said that asbestos associated cancer is linked to free radicals. Loose intracellular iron can also promote DNA damage. Iron can initiate cancer mainly by the process of oxidation of DNA molecules (Bhasin et al., (2002).

RESULT AND DISCUSSION

At the present time, experts and researchers around the world have completed a great deal of valuable research regarding the toxic effects and mechanisms of heavy metals. As a result, major achievements have been reached in the field of effective water environment quality standard systems. Lead can cause nerve damage even when exposed to low levels of lead, especially in infants and children.Kumar et al.,2012 reported that as a nonessential element in plant metabolism, the toxicity of lead is usually related to physiological processes, in which it interferes with the normal functions of cells to organs, including seed germination and delayed growth, water deficiency, nutritional disorders, and reduced photosynthesis, respiration, and transpiration processes.Rengaraj et al.,2007 dicussed that excessive exposure to chromium may result in serious damage to many aquatic organisms, carcinogenic effects in humans, and skin irritations and corrosion

CONCLUSION

Thus finally, this paper illuminated the hazardous role of heavy metals which are responsible for different health anomalies which are caused due to exposure to these chemical diseases like Alzheimer. Parkinson's. Depression, Headaches, Thyroid problems, Skin diseases. Cardio vascular diseases. Few digestive problems are also related with exposure or consumption of heavy metals. The role of plants are now taken as a weapon for the treatment of heavy metals in ground. The plants resistance to heavy metals reflects their ability to reduce the toxicity of heavy metals. Plants that are resistant to heavy metals mainly use methods such as blocking the absorption of heavy metals, extracellular complexation, cytoplasmic complexation and chelation. With the development of molecular biology, molecular biology is more widely used in tolerance studving the plant to heavy metals. Thus, phytoremediation is now exploited for reduction of heavy metals from the ground.

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REFERENCES

Järup L. Hazards of heavy metal contamination. Br Med Bull. 2003;68(1):167–182

Lambert M, Leven BA, Green RM. New methods of cleaning up heavy metal in soils and

water; Environmental science and technology briefs for citizens; Manhattan, KS: Kansas State University; 2000

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

Hughes JP, Polissar L, Van Belle G. Evaluation and synthesis of health effects studies of communities surrounding arsenic producing industries. Int J Epidemiol. 1988;17:407–413.

Singh N, Kumar D, Sahu A. Arsenic in the environment: effects on human health and possible prevention. J Environ Biol. 2007;28(2 Suppl):359–365

M. M. Khan, E. Islam, S. Irem et al., "Pbinduced phytotoxicity in para grass (*Brachiaria mutica*) and Castorbean (*Ricinus communis* L.): antioxidant and ultrastructural studies," *Chemosphere*, vol. 200, pp. 257–265, 2018.

Anjum N A, Hasanuzzaman M, Hossain M A, et al. (2014) Jacks of metal/metalloid chelation trade in plants-an overview[J]. Frontiers in Plant Science, 6(192):192.

Wang Y, Shen H, Xu L, et al. (2015) Transport, ultrastructural localization, and distribution of chemical forms of lead in radish (Raphanus sativus L.)[J]. Frontiers in Plant Science, 6:293.

A. Kumar, M. N. V. Prasad, and O. Sytar, "Lead toxicity, defense strategies and associated indicative biomarkers in Talinum triangulare grown hydroponically," *Chemosphere*, vol. 89, no. 9, pp. 1056–1065, 2012

Martin S, Griswold W. Human health effects of heavy metals. Environmental Science and Technology Briefs for Citizens. 2009;(15):1–6

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

Yongsheng W, Qihui L, Qian T. Effect of Pb on growth, accumulation and quality component of tea plant. Procedia Engineering. 2011;18:214– 219

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

INTERNATIONAL JOURNAL OF CURRENT ENGINEERING AND SCIENTIFIC RESEARCH (IJCESR)

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

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

Ashe WF, Largent EJ, Dutra FR, Hubbard DM, Blackstone M. Behavior of mercury in the animal organism following inhalation. AMA Arch Ind Hyg Occup Med. 1953;7(1):19–43

Bernard A. Cadmium & its adverse effects on human health. Indian J Med Res. 2008;128(4):557–64

Mutlu A, Lee BK, Park GH, Yu BG, Lee CH. Long-term concentrations of airborne cadmium in metropolitan cities in Korea and potential health risks. Atmos Environ. 2012;47:164–173

Han JX, Shang Q, Du Y. Effect of environmental cadmium pollution on human health. Health. 2009;1(3):159–166

Henson MC, Chedrese PJ. Endocrine disruption by cadmium, a common environmental toxicant with paradoxical effects on reproduction. Exp Biol Med (Maywood) 2004;229(5):383–392

Chakraborty S, Dutta AR, Sural S, Gupta D, Sen S. Ailing bones and failing kidneys: a case of chronic cadmium toxicity. Ann Clin Biochem. 2013;50(5):492–495

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

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

Wolińska A, Stępniewska Z, Włosek R. The influence of old leather tannery district on

chromium contamination of soils, water and plants. Nat Sci. 2013;5(2A):253–258

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

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

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

S. Rengaraj, S. Venkataraj, J. W. Yeon et al., "Preparation, characterization and application of Nd–TiO₂ photocatalyst for the reduction of Cr (VI) under UV light illumination," *Applied Catalysis B Environmental*, vol. 77, no. 1-2, pp. 157–165, 2007

Schroeder HA, Nason AP, Tipton IH. Chromium deficiency as a factor in atherosclerosis. J Chron Dis. 1970;23(2):123– 142

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

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

Olaniran AO, Balgobind Pillay Β. А, Bioavailability of heavy metals in soil: impact biodegradation microbial of organic on compounds and possible improvement strategies. Int J Mol Sci. 2013;14(5):10197-10228

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

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

INTERNATIONAL JOURNAL OF CURRENT ENGINEERING AND SCIENTIFIC RESEARCH (IJCESR)

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

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

Phippen B, Horvath C, Nordin R, Nagpal N. Ambient water quality guidelines for iron: overview; Ministry of Environment Province of British Columbia; 2008. Grazuleviciene R, Nadisauskiene R, Buinauskiene J, Grazulevicius T. Effects of Elevated Levels of Manganese and Iron in Drinking Water on Birth Outcomes. Polish J of Environ Stud. 2009;18(5):819–825

Nelson RL. Dietary iron and colorectal cancer risk. Free Radic Biol Med. 1992;12(2):161–168

Bhasin G, Kauser H, Athar M. Iron augments stage-I and stage-II tumor promotion in murine skin. Cancer Lett. 2002;183(2):113–122