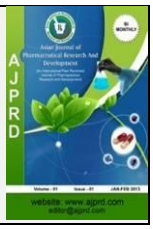
Available online on 15.08.2026 at http://ajprd.com

Asian Journal of Pharmaceutical Research and Development

Open Access to Pharmaceutical and Medical Research





Open Access

Review Article

# Toxicological Scenario of Heavy Metals on Human Health: Current Status

**Manoj Kumar*[1], Dr. Rubal Rathi[2], Ankita Kumari[3], Sanjana[3], Abhinay Gupta[1], Dr. Priyanka[1], Aslam[1], Triloki Prasad[1]**

[1] Kalka Institute for research and advanced studies Partapur bypass, Meerut

[2] Principal, MIIT college of Pharmacy, Ghat Road Meerut.

[3,] Krishak college of Pharmacy, Rajgarh, Mirzapur, Uttar Pradesh

## ABSTRACT

Heavy metals are persistent environmental contaminants and naturally occurring elements of the Earth's crust. There are several ways that humans might be exposed to heavy metals, such as through the food chain, consuming contaminated water or soil, or breathing in air or dust particles. Their bioaccumulation may have a variety of harmful impacts on various organ systems and bodily tissues. The characteristics of the particular metal, dose, route, length of exposure (acute or chronic). The ability of heavy metals to disrupt antioxidant defense mechanisms, mainly through their interaction with intracellular glutathione (GSH) or sulfhydryl groups (R-SH) of antioxidant enzymes like superoxide dismutase (SOD), catalase, and other enzyme systems. Although arsenic (As) is believed to bind directly to critical thiols, alternative hydrogen peroxide production processes have also been postulated. Numerous biological processes, such as cell development, proliferation, survival, metabolism, and apoptosis, are known to be impacted by heavy metals, which also disrupt signaling pathways. It has been demonstrated that nuclear factor erythroid 2-related factor 2 (Nrf2) functions as a double-edged sword in response to arsenic-induced oxidative stress. Nrf2 is an essential regulator of antioxidant enzymes, the degree of oxidative stress, and cellular tolerance to oxidants.

**Keywords:** Heavy metals, toxicity, lead poisoning, cancer, environmental distortion.

**ARTICLE INFO:** Received 19 Jan. 2025; Review Complete 22 March, 2026; Accepted 28 June. 2026; Available online 15 August 2026

**Cite this article as:**

Kumar M, Rathi R, Kumari A, Sanjana, Gupta A, Priyanka, Aslam, Prasad T, Toxicological Scenario of Heavy Metals on Human Health: Current Status, Asian Journal of Pharmaceutical Research and Development. 2026; 14(4): 82-88,
DOI: http://dx.doi.org/10.22270/ajprd.v14i4.1856

*Address for Correspondence:

Manoj Kumar, Kalka Institute for research and advanced studies Partapur bypass, Meerut

## INTRODUCTION:

The contemporary world grapples with a significant environmental challenge in the form of heavy metal pollution. The swift pace of industrialization, marked by expansive manufacturing growth, contributes to a continual rise in metal concentrations, giving rise to severe pollution issues. The accumulation of certain metals in living organisms is referred to as heavy metal toxicity causes detrimental effects in living organisms. These elements include lead, cadmium, nickel, arsenic, and mercury and are characterized by high densities and high atomic weights. In minute quantities, metals tend to play a significant role as essential nutrients, actively participating in diverse enzymatic and metabolic pathways while serving as cofactors. Their involvement is integral to the proper functioning of biological systems across various life forms. However, when these metals are present in high levels they may cause lethal effects on plants, animals, microorganisms, and humans [1-3].

Metals like cadmium, arsenic, and mercury, exhibit remarkable toxicity even at low concentrations, as documented by previous studies. The consequences of elevated metal concentrations become particularly evident when they accumulate in the soil, leading to adverse impacts on both productivity and fertility. Furthermore, the entry of these metals into the ecosystem through diverse pathways—be it through food, water, or air—can result in their bioaccumulation over extended periods. Bioaccumulation of metals exerts a potent threat not only to human health but also to the environment. The persistence of these harmful metals in the environment raises issues about their long-term negative impact on the ecosystems and the organisms living in that environment[4-7].

The toxic effect of these metal ions in human bodies is quite severe and multifaceted. Several researches have demonstrated the adverse effects of these metals such as carcinogenesis, growth and developmental issues, mental illness, disruption in metabolic pathways, and neuromuscular defects. Heavy metals are reported to be omnipresent and have raised concerns regarding their negative Eco toxicological effects on human health and the environment.

These are released into the environment via mining activities, industrial activities, and several other anthropogenic activities.

Their physicochemical properties like solubility, ionization states, and redox behavior, contribute to their unique toxicological profiles. These metals pose a high level of toxicity to organisms since these are persistent in nature and they may undergo complex transformations and can accumulate in the tissues of organisms leading to bio magnification that in turn result in severe negative impacts on organisms of higher trophic levels.

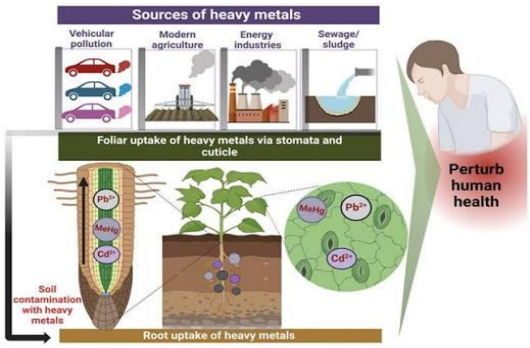



**Figure: 1** Different sources of heavy metals

The various methods that heavy metals enter the environment contribute to the complexity of heavy metal toxicity. These routes include air deposition, mining operations, agricultural runoff, and industrial discharge. Heavy metals can spread through contaminated soil, water, and air, endangering people, animals, and plants. When heavy metals enter biological systems, they often cause oxidative stress, alter cellular functions, and interfere with enzymatic processes. Depending on the type of heavy metal, exposure duration, and chemical reactivity, prolonged exposure to heavy metals at a significant level has been shown to impact brain functions, developmental activities, and ultimately carcinogenesis[8,9].The toxicity of heavy metals is significantly impacted by their speciation or chemical forms, making heavy metal pollution a major environmental and public health concern. Lead, mercury, cadmium, and arsenic are examples of metals that can exist in elemental, ionic, and organic compound forms with varying solubility, mobility, and toxicity properties.

For example, inert, water-insoluble, and biologically less active mercury in its elemental form becomes extremely poisonous, bio accumulating in aquatic species and posing a serious risk to human health due to neurological damage on methylation.

In the case of lead (II) nitrate, among others, exposure will be higher through drinking water since the soluble form provides an easy way to enter the water. However, depending on their solubility, less soluble lead sulphate and lead chloride may leak into water from a solid structure, releasing lead ions that could be harmful to human health. Therefore, it is crucial to comprehend toxicity in relation to chemical speciation in order to evaluate the impact of heavy metals on the environment and their corresponding remediation techniques[10-13].

Numerous mechanistic factors contribute to the toxicity and carcinogenicity caused by heavy metals. According to Kim et al. (2019), each metal is known to have unique physicochemical traits and features that contribute to its unique toxicological modes of action. An interdisciplinary approach encompassing chemistry, biology, environmental science, and medicine is necessary to comprehend heavy metal toxicity. Researchers look at how metals are absorbed, transported, and accumulated in living things as well as how metals interact with biological constituents. Furthermore, when assessing the total influence on ecosystems, the mobility and destiny of heavy metals in the environment are crucial factors.

## Heavy metals and their toxicity mechanisms

One of the most significant heavy metals that is concerning from an ecological and personal health perspective is arsenic. It is widely accessible as oxides or sulphides, or as a salt of iron, sodium, calcium, copper, etc., and has a semi-metallic quality. It is also very poisonous and carcinogenic .The inorganic forms of arsenic, such as arsenite and arsenate compounds, are deadly to both the environment and living things[14-18]. Arsenic is the tenth most abundant element on Earth. Arsenic can come into contact with humans through industrial, natural, or accidental sources. Natural mineral deposits, improper disposal of arsenical compounds, and the use of arsenical insecticides can all contaminate drinking water.

Acute poisoning can also occur from youngsters inadvertently consuming arsenic or intentionally consuming it during suicidal attempts. According to Gordon and Quastel (1948), arsenic is a proto-plastic toxin since it mostly affects the sulphydryl group of cells, impairing respiration, cell enzymes, and mitosis. Arsenic toxicity mechanism Monomethylarsonic acid (MMA) and dimethylarsinic acid (DMA) are produced during arsenic biotransformation when bacteria, algae, fungus, and humans methylate hazardous inorganic arsenic molecules[19,20].

These inorganic arsenic species (iAs) undergo enzymatic conversion during this biotransformation process to methylated arsenicals, which are the final metabolites and a biomarker of long-term exposure to arsenic. Methylated inorganic arsenic, such as MMA (V) and DMA (V), which are eliminated through urine, are bioindicators of long-term exposure to arsenic. Bio methylation is a detoxifying process. MMA (III), on the other hand, stays inside the cell as an intermediate product and is not eliminated. Compared to other arsenicals, monomethylarsonic acid (MMA III), an intermediate product, is discovered to be extremely hazardous and may be responsible for arsenic-induced carcinogenesis [21].

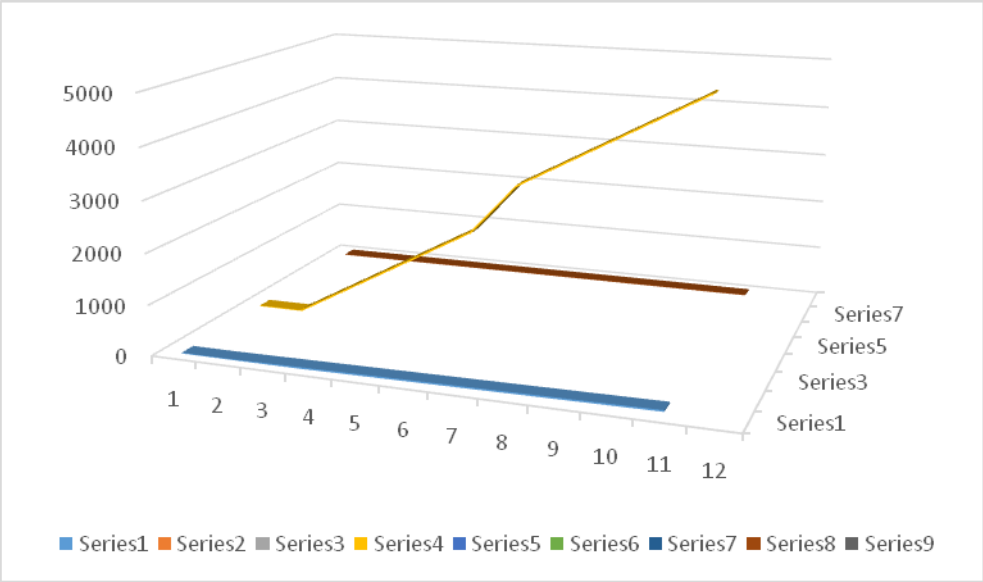



**Figure: 2** The global production and consumption of selected toxic metals during 1850–1990 (Adapted from Nriagu, 1996).

## Lead

Lead is a highly toxic metal whose widespread use has caused extensive environmental contamination and health problems in many parts of the world. Lead is a bright silvery metal, slightly bluish in a dry atmosphere. It begins to tarnish on contact with air, thereby forming a complex mixture of compounds, depending on the 90 80 70 60 50 40 30 20 Production (million tones/year) 10 0 4500 4000 3500 3000m 2500 2000 1500 1000 500 0 Emissions (thousands tones/year). The global production and consumption of selected toxic metals during 1850–1990.Metal plating and finishing operations Fertilizers and pesticides Smelting of ores Wastes from battery industries Sources of lead in the environment Factory chimneys Soil wastes Exhaust from Automobiles Additives in gasoline and pigment various sources of lead pollution in the environment given conditions. Figure 3 shows various sources of lead pollution in the environment .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.

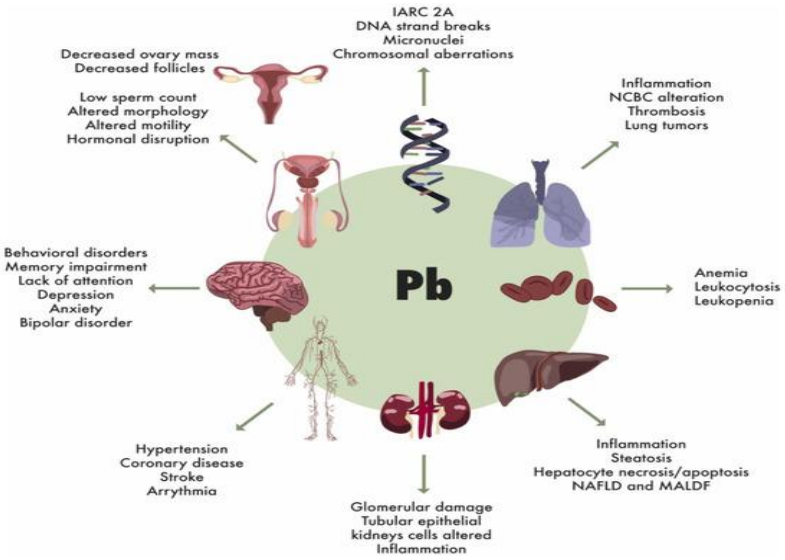



**Figure: 3** Lead toxicity of in various organs.

In the US, more than 100 to 200,000 tons of lead per year is being released from vehicle exhausts. Some is taken up by plants, fixation to soil and flow into water bodies, hence human exposure of lead in the general population is either due to food or drinking water. 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 bio logical functions[22,23,24]. 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. 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. Even at low concentrations, lead treatment was found to cause huge instability in ion uptake by plants, which in turn leads to significant metabolic changes in photosynthetic capacity and ultimately in a strong inhibition of plant growth. Mechanisms of lead toxicity Lead metal causes toxicity in living cells by following ionic mechanism and 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. Figure 3 shows the attack of heavy metals on a cell and the balance between ROS production and the subsequent defense presented by antioxidants. Antioxidants, as e.g. glutathione, present in the cell protect it from free radicals such as H2O2. Under the influence of lead, however, the level of the ROS increases and the level of antioxidants decreases. Since glutathione exists both in reduced (GSH) and oxidized (GSSG) state, the reduced form of glutathione gives its reducing equivalents (H+ + e–) from its thiol groups of cystein to ROS in order to make them stable. In the presence of the enzyme glutathione peroxidase, reduced MERCURY CADMIUM Resulting in Apoptosis oxidative stress SOD, GSH, GST, Catalase Defense by antioxidants ALUMINIUM Production of ROS O2 , OH, NO , RO ONOO , H2 O2 , IRON ARSENIC CHROMIUM LEAD Figure 3. The attack of heavy metals on a cell and the balance between ROS production and the subsequent defense presented by antioxidants. glutathione readily binds with another molecule of glutathione after donating the electron and forms glutathione disulfide (GSSG). The reduced form (GSH) of glutathione accounts for 90% of the total glutathione content and the oxidized form (GSSG) accounts for 10% under normal conditions. Yet under the condition of oxidative stress, the concentration of GSSG exceeds the concentration of GSH. Another biomarker for oxidative stress is lipid peroxidation, since the free radical collects electron from lipid molecules present inside the cell membrane, which eventually causes lipid peroxidation[25-30]. 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.

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.

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[31-33].

Lead can substitute calcium even in Pico molar concentration affecting protein kinase C, which regulates neural excitation and memory storage[34-37].

## Cadmium

Cadmium is a metal of the 20th century. It is a byproduct of zinc production. Soils and rocks, including coal and mineral fertilizers, contain some amount of cadmium. Cadmium has many applications, e.g. in batteries, pigments, plastics and metal coatings and is widely used in electroplating. Figure 9 presents a relative contribution of different sources to human cadmium exposure. Cadmium and its compounds are classified as Group 1 carcinogens for humans by the International Agency for Research on Cancer. Cadmium is released into the environment through natural activities such as with fish age and with increasing trophic levels. EPA has declared mercuric chloride and methyl mercury to be highly carcinogenic. The nervous system is very sensitive to all types of mercury. Increased exposure of mercury can alter brain functions and lead to shyness, tremors, memory problems, irritability, and changes in vision or hearing. 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[39,40].

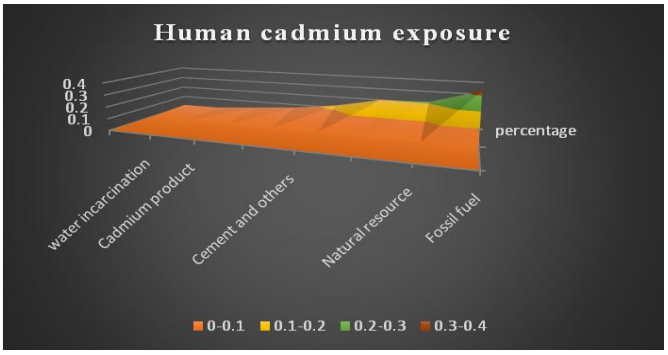



**Figure: 4** cadmium exposure and relatable sources.

### Aluminum

Decreases the concentration of copper in liver and plasma and also reduces the intestinal absorption and replaces zinc present in metallothionein concentration of ceruloplasmin in plasma Figure 10. Values of cadmium toxicity. Interacts with iron and decreases the hemoglobin and hematocrit concentration, leads to anemia Aluminum is the third most common element found on the earth's crust. It exists in only one oxidation state (3+) in the environment[41-43]. The main routes of aluminum consumption by humans are through inhalation, ingestion and dermal contact and sources of exposure are drinking water, food, beverages, and aluminum containing drugs. Aluminum is naturally present in food. Aluminum and its compounds are poorly absorbed in humans, although the rate at which they get absorbed has not been clearly studied. Symptoms that indicate the presence of higher amounts of aluminum in the human body are nausea, mouth ulcers, skin ulcers, skin rashes, vomiting,diarrhea and arthritic pain. These symptoms have however been reported to be mild and short lived (Clayton, 1989). Aluminium exposure is probably a risk factor for the onset of Alzheimer disease (AD) in humans, as hypothesized by the WHO, 1997. Contact dermatitis and irritant dermatitis were seen in persons who were exposed to aluminium in their place of work. Aluminium showed adverse effects on the nervous system and resulted in loss of memory, problems with balance and loss of coordination[44-47]

Because patients with kidney diseases have difficulty eliminating the metal, accumulation of aluminium in the body damages bones and the brain. Aluminium poisoning may develop as a result of living in dusty settings, receiving long-term intravenous nourishment, having reduced kidney function, receiving hemodialysis, drinking or ingesting substances high in aluminium content, and working in an environment with high levels of aluminium. Patients undergoing kidney dialysis may come into contact with aluminium from contaminated dialysates and phosphate binders. In addition to causing other disorders including aluminum-induced osteomalacia and aluminum-induced adynamic bone disease, which are both characterized by low-bone remodeling, increased exposure to aluminium can change the course of secondary hyperparathyroidism[48-50].Lung issues, anaemia, poor iron absorption, nervous system issues, etc. are some additional symptoms linked to aluminium toxicity. The most prevalent transition metal in the earth's crust is iron. Since it is a cofactor for numerous essential proteins and enzymes, it is the most significant nutrient for the majority of living things. The majority of aerobic organisms rely on iron-mediated processes to support their respiration. It can catalyse reactions that result in the creation of radicals, which can harm biomolecules, cells, tissues, and the entire organism if it is not adequately insulated. The issue of iron poisoning has always been of particular interest to pediatricians. Due to their increased exposure to iron-containing items, children are particularly vulnerable to iron poisoning. . Iron toxicosis occurs in four stages. The first stage which occurs after 6 hrs of iron overdose is marked by gastrointestinal effects such as gastro intestinal bleeding, vomiting and diarrhea. The second stage progresses within 6 to 24hrs of overdose and it is considered as the latent period, a period of apparent medical recovery. The third stage occurs between 12 to 96 hrs after the onset of certain clinical symptoms[51-53]. This stage is characterized by shocks, hypotension, lethargy, tachycardia, hepatic necrosis, metabolic acidosis and sometimes death. The fourth stage occurs within 2–6 weeks of iron overdose. This stage is marked by the formation of gastrointestinal ulcerations and development of strictures. Excess iron uptake is a serious problem in developed and meat eating countries and it increases the risk of cancer. 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. 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[54].

Iron salts such as iron sulfate, iron sulfate monohydrate and iron sulfate heptahydrate are of low acute toxicity when exposure is through oral, dermal and inhalation routes and hence they have been placed in toxicity category 3. Furthermore, iron salts are considered to be safe by the Food and Drug Administration and their toxic effects are very much negligible. Formation of free radicals is the outcome of iron toxicity. Superoxide and hydrogen peroxide are byproducts of normal and pathological cell processes that are regarded as free radicals. Enzymes like superoxide dismutase, catalase, and glutathione peroxidase actually neutralize these free radicals. However, the superoxide molecule can liberate iron from ferritin, and that free iron reacts with increasing amounts of superoxide and hydrogen peroxide to form extremely toxic free radicals like hydroxyl radical. Because they can depolymerize polysaccharides, start lipid peroxidation, inactivate certain enzymes, and break DNA strands, hydroxyl radicals are harmful. Cell death may occasionally follow from this[55-57].

## CONCLUSION

The impacts of several heavy metals, including arsenic, lead, cadmium, chromium, and aluminium, on the environment and living things, mostly humans, were examined in this review. Effective laws, regulations, and the identification of regions with elevated heavy metal concentrations are required. The negative impacts of heavy metals will cause serious problems in the future if the exposure is not controlled. Engineering solutions can reduce occupational exposure to heavy metals. Monitoring exposure and potential interventions to limit future exposure to heavy metals in persons and the environment can be a significant step in the direction of prevention. In order to develop effective strategies to reduce heavy metal toxicity, national and international cooperation is essential.Research on the various heavy metals that can interact with the body is crucial since occupational toxicology frequently has to deal with the issues of poisoning with multiple metals at once. Furthermore, the body's critical metals and harmful metals may interact in ways that are antagonistic, synergistic, or additive. An interdisciplinary approach including experts in toxicology, pharmacology, epidemiology, molecular biology, and other fields is necessary to address this and other issues pertaining to the toxicity of heavy metals and their consequences on health.

## REFERENCES:


1. Agency for Toxic Substances and Disease Registry (2008). Public Health Statement Aluminium. ATSDR Publication CAS#7429-90-5.
2. Albretsen J. The toxicity of iron, an essential element. Veterinary medicine 2006; 82–90
3. Alina M, Azrina A, Mohd Yunus AS, Mohd Zakiuddin S, MohdIzuan Effendi H, Muhammad Rizal R. Heavy metals (mercury, arsenic, cadmium, plumbum) in selected marine fish and shellfish along the Straits of Malacca. Int Food Res 2012; J 19(1): 135–140.
4. Andia JB. Aluminum toxicity: its relationship with bone and iron me tabolism. Nephrol Dial Transplant 11 Suppl 1996; 3:69–73.
5. 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.
6. Barabasz W, Albinska D, Jaskowska M, Lipiec J.Ecotoxicology of Alu minium. Pol J Environ Stud 2002; 11(3): 199–203.
7. Becker M, Asch F. Iron toxicity in rice – conditions and management concepts. J Plant Nutr Soil Sci 2005; 168:559–553.
8. Bernard A. Cadmium & its adverse eff ects on human health. Indian J Med Res 2008; 128(4):557–64.
9. A speciation analysis of aluminium in the River Silnica. Pol J Environ Stud 10(4): 263–268.
10. Bhasin G, Kauser H, Athar M. Iron augments stage-I and stage-II tu mor promotion in murine skin. Cancer Lett 2002; 183(2): 113–122.
11. Bielicka A, Bojanowska I, Wisniewski A. Two Faces of Chromium-Pol lutant and Bioelement. Pol J Environ Stud 2005; 14(1): 5–10.
12. Brochin R, Leone S, Phillips D, Shepard N, Zisa D, Angerio A. The cellular effect of lead poisoning and its clinical picture. GUJHS. 2008; 5(2): 1–8.
13. Castagnetto JM, Hennessy SW, Roberts VA, Getzoff ED, Tainer JA, Pique ME. MDB: the metalloprotein database and browser at the Scripps Research Institute. Nucleic Acids Res2002; 30(1): 379–382.
14. Cervantes C, Campos-García J, Devars S, Gutiérrez-Corona F, Loza-Tavera H, Torres-Guzmán JC, Moreno-Sánchez R. Interactions of chromium with microorganisms and plants. FEMS Microbiol Rev 2001; 25(3): 335–347.
15. 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.
16. Chandra P, Kulshreshtha K. Chromium accumulation and toxicity in aquatic vascular plants. Botanical Rev 70(3): 313–327. Chen CW, Chen CF, Dong CD. 2004; (2012).
17. Distribution and Accumulation of Mercury in Sediments of Kaohsiung River Mouth, Taiwan. APCBEE Procedia 1: 153–158.
18. Chowdhury UK, Biswas BK, Chowdhury TR, Samanta G, Mandal BK, Basu GC, Chakraborti D. Groundwater arsenic contamination in Bangladesh and West Bengal, India. Environ Health Perspect 2000; 108(5): 393–397.
19. Clayton DB. (1989). Water pollution at Lower Moore North Cornwall: Report of the Lower Moore incident health advisory committee. Truro, Cornwall District Health Authority, 22 pp. Duan N, Wang XL, Liu XD, Lin C, Hou J. (2010).
20. Effect of anaerobic fermentation residues on a chromium-contaminated soil-vegetable system. Procedia Environmental Sciences 2: 1585–1597.
21. Ferner DJ. Toxicity, heavy metals. eMed J 2(5): 1. Ferrara R, Mazzolai B, Lanzillotta E, Nucaro EA, Pirrone N. (2000). Temporal trends in gaseous mercury evasion from the Mediterranean seawaters. Sci Tot Environ 2001; 259(1–3): 183–190.
22. Fine JS. Iron poisoning. CurrProblPediatr 2000; 30(3): 71–90.
23. Flora SJS, Mittal M, Mehta A. (2008). Heavy metal induced oxidative stress & its possible reversal by chelation therapy. Indian J Med Res 128: 501–523. Gardner JL, Al-Hamdani SH. (1997).
24. Interactive effects of aluminum and humic substances on Salvinia. J Aquat Plant Manage 35: 30–34.
25. Gerhardsson L, Dahlin L, Knebel R, Schütz A. Blood lead concentration after a shotgun accident. Environ Health Perspect 2002; 110(1): 115–117. Ghani A. (2011).
26. Gordon JJ, Quastel GH. Effect of chromium toxicity on growth, chlorophyll and some mineral nutrients of Brassica juncea L. Egyptian Acad J Biol Sci 1948; 2(1): 9–15.
27. Effect of organic arsenicals on enzyme system. Biochem J Goyer RA. Lead toxicity: from overt to subclinical to subtle health effects. Environ Health Perspect 1990; 86: 177–181.
28. 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.
29. Gupta N, Gaurav SS, Kumar A. Molecular Basis of Aluminum Toxicity in Plants: A Review. Am J of Plant Sci 2013; 4: 21–37.
30. Gürkan R, Ulusoy HI, Akçay M. Simultaneous determination of dis solved inorganic chromium species in wastewater/natural waters by surfactant sensitized catalytic kinetic spectrophotometry. Arabian J Chem [in press]. Haley BE. Mercury toxicity: genetic susceptibility and synergistic effects. Medical Veritas 2012; 2(2): 535–42.
31. Han JX, Shang Q, Du Y. Effect of environmental cadmium pollution on human health. Health 2009; 1(3):159–166.
32. Henson MC, Chedrese PJ. Endocrine disruption by cadmium, a common environmental toxicant with paradoxical effects on reproduction. ExpBiol Med (Maywood) 2004; 229(5): 383–392.
33. Hershko C, Link G, Ioav C. (1998). Pathophysiology of iron overload. Ann N Y Acad Sci 850: 191–201.
34. Hillman RS. (2001). Chapter 54. Hematopoietic agents: growth factors, minerals, and vitamins, in Goodman & Gilman's The Pharmacological Basis of Therapeutics, 10th Edition (Hardman JG, Limbird LE, Gilman AG eds), pp. 1487 1518, New York: McGraw-Hill.
35. Hoque MA, Burgess WG, Shamsudduha M, Ahmed KM. Delineating low-arsenic groundwater environments in the Bengal Aquifer System, Ban gladesh. Appl Geochem 2011; 26(4): 614–623.
36. Hughes JP, Polissar L, Van Belle G. Evaluation and synthesis of health eff ects studies of communities surrounding arsenic producing industries. Int J Epidemiol 1988; 17: 407–413.`
37. Irfan M, Hayat S, Ahmad A, Alyemeni MN. Soil cadmium enrichment: Al location and plant physiological manifestations. Saudi J Biol Sci 2013; 20(1): 1–10.
38. Järup L. Hazards of heavy metal contamination. Br Med Bull 2003; 68(1): 167–182.
39. Jaishankar M, Mathew BB, Shah MS, Gowda KRS. (2014). Biosorption of Few Heavy Metal Ions Using Agricultural Wastes. Journal of Environment Pollution and Human Health 2014; 2(1):1–6.
40. Khlifi R, Hamza-Chaff ai A. Head and neck cancer due to heavy metal exposure via tobacco smoking and professional exposure: A review. Toxicol Appl Pharmacol 2010; 248: 71–88.
41. 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.
42. Koutras GA, Schneider AS, Hattori M, Valentine WN. Studies on chro mated erythrocytes. Mechanisms of chromate inhibition of glutathione re ductase. Br J Haematol 1965; 11(3): 360–369.
43. Krewski D, Yokel RA, Nieboer E, Borchelt D, Cohen J, Harry J, Rondeau V. Human health risk assessment for aluminum, aluminum oxide, and aluminium hydroxide. JJ Toxicol Environ Health B Crit Rev. 2007; 10(S1): 1–269.
44. Lambert M, Leven BA, Green RM. New methods of cleaning up heavy metal in soils and water. Environmental science and technology briefs for citizens. Kansas State University, Manhattan, KS. Lamhamdi M, El Galiou O, Bakrim A, Nóvoa-Muñoz JC, Arias-Estévez M, Aarab A, Lafont R. Effect of lead stress on mineral content and growth of wheat (Triticumae stivum and spinach (Spinaciaoleracea) seedlings. Saudi J Biol Sci2013; 20(1): 29–36.
45. Markowitz, M. Lead Poisoning. Pediatr Rev 2000; 21(10): 327–335.
46. Martin S, Griswold W. Human health effects of heavy metals. Environ mental Science and Technology Briefs for Citizens 2009; (15): 1–6
47. Mathew BB, Tiwari A, Jatawa SK. Free radicals and antioxidants: A re view. Journal of Pharmacy Research 20114(12): 4340–4343.
48. Matschullat J. Arsenic in the geosphere – a review. Sci Total Environ 2011; 249(1–3): 297–312.
49. Matsumoto ST, Mantovani MS, Malaguttii MIA, Dias AL, Fonseca IC, Marin Morales MA.Genotoxicity and mutagenicity of water contaminated with tannery effl uents, as evaluated by the micronucleus test and comet assay using the fish Oreochromis niloticus and chromosome aberrations in onion root-tips. Genet Mol Biol 2006; 29(1): 148–158.
50. Mazumder G. Chronic arsenic toxicity & human health. Indian J Med Res 2008; 128(4): 436–447.
51. McCord JM. Iron, free radicals, and oxidative injury. Semin Hematol 1998; 35: 5–12.
52. Mohanty M, Kumar Patra H. Effect of ionic and chelate assisted hexavalent chromium onmung bean seedlings (Vigna Radiata l. Wilczek. Var k-851) during seedling growth. JSPB 2013; 9(2):232–241.
53. O'Brien T, Xu J, Patierno SR. (2001). Eff ects of glutathione on chromium-induced DNA crosslinking and DNA polymerase arrest. In Molecular Mechanisms of Metal Toxicity and Carcinogenesis (pp. 173–182).
54. Springer US. Olaniran AO, Balgobind A, Pillay B. Bioavailability of heavy metals in soil: impact on microbial biodegradation of organic compounds and possible improvement strategies. Int J Mol Sci 2013; 14(5): 10197–10228.

55. Osweiler GD, Carson TL, Buck WB, Van Gelder GA. Clinical and diagnos tic veterinary toxicology. Kendall/Hunt Publishing Company. Papanikolaou NC, Hatzidaki EG, Belivanis S, Tzanakakis GN, Tsatsakis AM. Lead toxicity update. A brief review. Med Sci Monitor 1985; 11(10): RA329.
56. Patrick L. Mercury toxicity and antioxidants: Part 1: role of glutathione and alpha-lipoic acid in the treatment of mercury toxicity. Altern Med Rev 2003; 7(6): 456–471. Patrick L.
57. Toxic metals and antioxidants: Part II. The role of antioxidants in arsenic and cadmium toxicity. Altern Med Rev 8(2): 106–128.
58. Phippen B, Horvath C, Nordin R, Nagpal N. (2008). Ambient water quality guide lines for iron: overview. Ministry of Environment Province of British Columbia. 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, 2005 in Berlin.