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Biomedical Impact of Heavy Metal Ions on Human Health

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Editorial

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Abbreviations: TDI: Tolerable Daily Intake; ADI: Acceptable Daily Intake; PTWI: Provisional Tolerable Weekly Intakes; IARC: International Agency for Research on Cancer; EPA: Environmental Protection Agency.

Editorial

Nearly thirty chemical elements are frequently institute in bio-organisms, involving in basic physio-biochemical and functions, and recognized as necessary elements for life [1]. Larger fraction of the identified metals and metalloids are extremely toxic to bio-organisms and even those considered as vital can be toxic if present in excess [2,3] as a result of human activities. They can upset important biochemical processes, constituting a key threat for the health of plants and animal systems including human beings. After entering the body, the metal deposited in nasopharyngeal, tracheobronchial, or pulmonary compartments may be transported by mucociliary action to the gastrointestinal tract. Macrophages phagocyted the wandering metals. Food is the most important route for entering essential and toxic elements. Some elements like mercury (Hg) are biologically magnified at higher trophic level. The dietary involvement for toxic metal intake has been comprehensively studied by a research group of toxicologists [4]. If the body is deficient in minerals and trace elements, it will absorb heavy metals in their place. Every cell membrane breaks down and rebuilds every two weeks but does not release the heavy metals if essential fats are not ingested or if bad fats are ingested. The liver, which performs detoxification 100% of the time, cannot perform this important task without all the essential nutrients [1-4].

Chemical elements present as free ions and those readily ionized are more or less absolutely absorbed by the body. Transition metals actively form stable covalent complexes and frequently interact as parts of macromolecules (enzymes, proteins, hormones, etc.) according to their chemical properties together with oxidation state [2,5]. These metals are conjugated with amino acids (glutathione (GSH), cysteine, and histidine) and proteins (metallothioneins, transferrin, ferritin, lactoferrin, hemosiderin, ceruloplasmin, and melanotransferrin). Health damage triggered by toxic metals may be less (irritation) or acute (teratogenic, mutagenic, and carcinogenic). These reactive elements of food present as complexes with fiber have a low solubility within the intestinal lumen and are poorly absorbed. Absorption of these minerals promotes by low concentration of fiber and absence of phytates, oxalates in the diet [6]. Micronutrients can interact with toxic metals at several stages in the body like absorption, transport, binding to target proteins, metabolism, sequestration, excretion of toxic metals and finally, in secondary symptoms of toxicity such as oxidative stress [7]. Therefore, a diet poor in micronutrients can have an important influence on the toxicity. In biological fluids and tissues, most metals and metalloids are not present as free cations. In blood, they are usually bound to red cells or to plasma proteins. Lead (Pb) and cadmium (Cd) are almost completely bound to red blood cells. The chemical elements bound to plasma proteins constitute the fraction available for transport into and out of the tissues [7].

Albumin, a plasma protein, has a prominent ability to bind a number of metals. In order to avoid undesirable health effects as resulted from “excessive” intake of toxicants (including toxic metals), FAO/WHO, FDA, European Union, etc. have used the safety guidelines for establishing tolerable or acceptable intakes of substances, which show signs of threshold toxicity. The tolerable daily intake (TDI) or acceptable daily intake (ADI) provisional tolerable weekly intakes (PTWI) are in practice to describe “safe” levels of intake for numerous toxicants including toxic metals [8]. Exposure larger than the TDI value for short period should not have lethal effects upon health. Nevertheless, acute

effects may take place if the TDI is considerably exceeded even for short periods of time. Additionally, contaminants possessing very long half-lives can be accumulated in the body and chronic effects are most often observed when critical concentrations are reached in target tissues, ultimately resulting in cancer that is a foremost reason of morbidity, mortality, and premature death worldwide [8].

This continual and emergent burden of cancer in the world's populations' warrants finely tuned public health awareness. Prevention, early detection, and therapy have all established parameters in checking certain types of cancer and in thus dropping down the burden of premature death and advanced disease [9,10]. The event and mortality as a consequence of multifactorial polygenic diseases such as varieties of cancer deviate depending upon genetic vulnerability and environmental precursors since they have definite mendelian subsets. Speedy alterations in diet and lifestyle may affect heritability of the variant phenotypes, which are dependent on the nutraceutical supplementation for their expression [9]. It is likely to differentiate the interaction of specific nutraceuticals, with the genetic code obsessed by all nucleated cells [9,10]. In several countries, however, these well-distinguished approaches to cancer check have not been practical to their complete potential and in various countries are not applied at all. Besides, enormous disparities still exist in cancer check in reference to gender, race, ethnicity, and socioeconomic status.

In context to carcinogens ranking, heavy metals have been classified by the International Agency for Research on Cancer (IARC) and Environmental Protection Agency (EPA) as the first group, except for selenium that has been listed within group 3 (not carcinogen to humans) of the IARC classification [9]. The focus on comparison of the epigenetic effects caused by various heavy metals in cancer-concerned genes in biological systems, including human has been highlighted as: (i) Chronic Pb intoxication in adults resulted in anemia, some types of cancer, reproductive harm in males while in young children hormonal imbalance of metabolite of vitamin D, namely 1,25-dihydroxy-vitamin D, and drop in IQ [8,11]; (ii) Cd exposure, during human pregnancy, led to reduced birth weights and premature birth [9,11]. Besides, evidence of a sex difference in the association between maternal Cd exposure and birth size was noticeable only in girls. Outcomes add support for the need to reduce Cd pollution to improve public health; (iii) The certain specific compounds of Al have been used in wide range of applications in different industries including cosmetics and food additives [12]. The epigenetic effects of Al take place through the binding of trivalent (Al^{3+}) to the phosphate groups of double-stranded DNA under physiologic pH, thus changing DNA topology from B to Z in (CCG) [12] repeat regions [9]; (iv) Arsenic is generally known as an epigenetic carcinogen metalloid when in the form of an inorganic compound. In the environment, arsenic is usually found combined with other elements as inorganic and organic forms. Inorganic arsenic is more poisonous than organic one. In humans, arsenic toxicity has been occured due to ingestion of As-containing powders or solutions accidentally, for suicide, homicide, or consumption of contaminated food or drinking water. Arsenic has been associated with hypertension and has serious effects on the cardiovascular system, and at high doses it causes hepatic damage [2,9]. It has a suppressive effect on spermatogenesis and gonadotrophin and testosterone release in rats [9,12]; (v) Cr exposure at very high concentrations activates all subclasses of MAPK through phosphorylation; thus, Cr acts as a MAPK kinase and enhances survival/proliferation in a dose-dependent manner. This function is connected with its capability in ROS generation [2]; (vi) Nickel has been observed to bind to DNA in different positions. It binds to phosphate backbone of DNA in place of Mg and promotes the conversion of suppressor genes to the heterochromatin [2]. Besides, its binding to histone H4 leads to the inhibition of lysine acetylation, and subsequently DNA hypermethylation, ultimately playing a significant role in silencing of tumor suppressor genes and the other genes, involved in carcinogenesis pathways; (vii) Elemental Hg can be oxidized to Hg^{2+}, accumulating preferentially in the kidneys. The augmented excretion of low molecular weight proteins is demonstrated at low-level exposure and related to damage to the renal tubes. It is a potent neurotoxin to human due to their ability to cross the blood–brain barrier. It is absorbed in the gastrointestinal track, immediately entering the bloodstream [6]; (viii) Selenium is an essential trace element with a narrow range between toxic and therapeutic doses; its activity for that reason is highly dose-dependent. Enzymes containing selenium such as glutathione peroxidase, like other antioxidant elements, can protect body from oxidative damage and reduce the risk of cancer incidence and mortality through several pathways such as apoptosis and alteration of some collagen types [5,9]; (ix) Hg and its compounds are highly toxic, wide dispersion through the atmosphere. It is biomagnified through the food chain [13].

Conclusion and Future Perspectives

Convincingly, the heavy metals play a pivotal role in the production of ROS and NF-kB, as well human genetic demarcation through polymorphisms in GST, metallothioneins and heavy metal methyltransferase genes induce carcinogenesis. Noticeably, heavy metals are epigenetic carcinogen, solely responsible for tumors expression and progression. Taken together, the documentation made by Mishra, et al. [14] and the ongoing research provides new insights and biochemical and molecular mechanisms concerning the expansion of pathological conditions in human system.

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