American International Journal of Research in Science, Technology, Engineering & Mathematics

Available online at http://www.iasir.net

ISSN (Print): 2328-3491, ISSN (Online): 2328-3580, ISSN (CD-ROM): 2328-3629

AIJRSTEM is a refereed, indexed, peer-reviewed, multidisciplinary and open access journal published by International Association of Scientific Innovation and Research (IASIR), USA

(An Association Unifying the Sciences, Engineering, and Applied Research)

Toxicology Effects of Arsenic, Cadmium, Mercury and Beryllium Ions on Mankind

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Abstract: *Toxic chemicals pose the problem of health hazards and put threat on lives of man animals. Many metals listed as environmental hazards are essential dietary trace elements required for normal growth and development of man and animals. Toxic chemicals attack the active sites of enzymes inhibiting essential enzyme function. The actual toxic action of each metal is different but most of them bind to the metabolically active groups such as carbonyl, phosphoryl, amino, imino, phenolic, sulphydryl or imidazole group. Metal ions As^{3+}, Cd^{2+}, Hg^{2+} have strong affinity for sulphur containing ligands like –SH and $–SCH_3$ in cysteine and methionine amino acids which are part of the enzymes, and act as enzyme inhibitors. As^{3+} is a general protoplasmic poison and it afects all the systems in man. Zn^{2+} in some metalloenzymes is substituted by Cd^{2+} and lead to cadmium toxicity. Cd^{2+} being potentially carcinogenic can inhibit activity of several enzymes. Hg^{2+} ion attacks mainly liver, kidneys, and can combine with carboxyl, sulphydryl, phosphoryl, amide and amino groups. Acute Beryllium poisoning induces chemical pneumonia resulting from the toxic effect of beryllium in its elemental form or in various chemical compounds, and is distinct from berylliosis. Signs and symptoms arising in man due to toxicological effects of these metal ions have been discussed, and treatment and remedial measures have been prescribed.*

Keywords: *Toxicology, Hazards, Essential and Trace Metals, Biochemical Effects, Enzymes.*

I. INTRODUCTION

About 81 trace elements play a vital role in plant and animal nutrition. Out of 40 naturally occurring elements, about 25 of them are highly essential. These include, metals : Na, K, Ca, Mg, Cr, Mn, Fe, Co, Mo, Zn, Sn, V and Cu, non-metals : H, C, N, O, S, P, Se, F, Cl, Br, I and Si then come less essential elements such as Ba, Sr, Cr, Br, Sn and Ni, non essential elements include Al, Pb, Cd, Bi and Hg and elements toxic at higher level are As, Cd, Hg, Sb and Se etc.

Chemical toxicology deals with the study of toxic chemicals and their mode of action in living organisms. Metals like Cu, Zn, Mn, Cr etc found in water essential at low levels serving as nutrients for animals and plants, but toxic in recessive amounts. Schwartz[1] used the term concentration window to draw the arbitrary lines of demarcation between essential limits and toxic limits of elements.

Toxic chemicals attack the active sites of enzymes inhibiting essential enzyme function. Heavy metal ions, in particular Pb^{2+}, Hg^{2+}, Cd^{2+}, As^{3+}, Sn^{4+} act as effective enzyme inhibitors. They have strong affinity for sulphur containing ligands like –SH and SCH_3 in cysteine and methionine amino acids which are part of the enzymes.

Metalloenzymes contain metals in their structures. Their function in inhibited when one metal ion of metalloenzyme is replaced by another metal ion of similar size and charge. For example, Zn^{2+} in some metalloenzymes is substituted by Cd^{2+} and lead to cadmium toxicity. Cd^{2+} being potentially carcinogenic can inhibit activity of several enzymes.

BIOCHEMICAL EFFETS OF ARSENIC[2-6] : Arsenic occurs in earth crust (2 ppm), sea water (5 ppb), soils (1 to 40 ppm), in human body tissues (18 mg) and blood (25 mg).

***Toxicological Effects*:** Arsenic undergoes sequential methylation by some bacteria and fungi leading to the release of dimethyl and trimethyl arsine. Arsenites being soluble in water are more phytotoxic than arsenates.Toxicology of Arsenic may be understood as it is a general protoplasmic poison and it affects all the systems in man. The order of toxicity of arsenic compound is: Arsines [As(III)] > arsenite [As(III)] > arsenate [As(V)] > arsenic organic acids [As(V)]. As(III) exerts its toxic action by attacking SH group of an enzyme thereby inhibiting enzyme action.

***Symptom and Treatment of Arsenic Poisoning*:** Acute arsenic poisoning is diagnosed by headache, vertigo, stupor, coma or by gastrointenstinal symptoms such as intense pain, projectile vomiting, diarrhea followed by shock and hypoxic convulsions etc. Antidote for Arsenic poisoning are chemicals having –SH groups capable of bonding to As(III), *e.g*., 2, 3-dimercaptopropanol (British Antilewisite, BAL).

BIOCHEMICAL EFFETS OF CADMIUM[7,8] : it is a toxic metal, occurs in nature in association with zinc minerals (1 : 200 in ZnS). Maximum permissible concentration of Cd in air and water is 0.3µg/m^3 and 5 ng/g. Normal Cd levels are less than 1% µg in blood. Its tolerable intake is 57 to 72µg per day. Normal Cd content in rice is about 29 ppb.

***Toxicological Effects*:** The major portion of Cd ingested into our body is trapped in the kidneys and eliminated. A small fraction is bound by the body proteins, metallothionein, present in kidneys, while the rest is stored in the body and gradually accumulates with age. Its absorption increases when there is deficiency of Ca in the diet. When excessive amounts of Cd^{2+} are ingested, it replaces Zn^{2+} at key enzymatic sites, including metabolic disorders.

BIOCHEMICAL EFFETS OF MERCURY[9-11]: Natural abundance of Hg in soil is 0.1 ppm. Fossil fuels, coal and lignite contain 100 ppb of Hg. The ambient air concentration of mercury prevailing in some industrial areas is found to be 0.7 µg/m^3. The USEPA limits of Hg in drinking water is 2 µgL^{-1}. Natural addition of Hg to the oceans is about 5000 tonnes per annum and a further 5000 tonnes is added via human activities. Sewage effluents may contain up to 10 times the level of (Hg in natural water (0.001 ppm).

Uses and Pollution Sources: The largest consumer of Hg is chloralkali industry which manufactures Cl_2 and NaOH by using Hg electrodes. Production of electrical apparatus (bulbs, switches, batteries) and the use of Hg in agricultural industry (*e.g.*, fungicides for seed dressings) constitute the second and third consumer of Hg. Pharmaceuticals, ointments, dental amalgams and finger print powders also disperse Hg in air. Once mercury is absorbed on sediments of water bodies, it is slowly released into the water and constitutes a reservoir which is likely to cause chronic pollution long after the original source of Hg is removed.

***Toxicology of Mercury*:** Minamata Epidemic of Japan after eating shell fish contaminated with mercury containing effluent from a nearby plastic factgory and tragic event of mercury poisoning of Iraq after eating wheat dusted with a mercury containing pesticide, boosted the awareness of Hg as a toxic pollutant. The vapours of elemental Hg are highly toxic when inhaled. Hg, being lipid soluble can be absorbed through the intact skin. It attacks mainly liver, kidneys and can combine with carboxyl, sulphydryl, posphoryl, amide and amino groups.

Remedial Measures: All chlor-alkali plants must not use Hg electrodes and switch to new technology, All alkyl mercury pesticides must be banned, Other mercurial pesticides must be restricted to some selected areas and Decontaminating sediments by covering the bottom sediments with fresh finely divided materials having high absorption capabilities and alternatively, by burying the sediments under inorganic inert materials.

***Treatment of Mercury Poisoning*:** Sodium formaldehyde sulphxide provides excellent local antidote, Using metal chelating agents such as N-acetyl-D, L-penicillamine etc. At the same time intramuscular injection of dimercaprol should be given to chelate the mercury and to accelerate its excretion and also by introducing a source of sulphydryl-rich protein (milk or raw eggs) and then performing copious lavage. Infusing isotonic NaCl solution (10 L per day) to produce copious diuresis or using supportive therapy.

BIOCHEMICAL EFFECTS OF BERYLLIUM[12-20] : Acute beryllium poisoning is an occupational disease. Relevant occupations are those where beryllium in mined. Processed or converted into metal alloys, or where machining of metals containing beryllium or recycling of scrap alloys occurs.

***Signs and Symptoms*:** It generally associated with exposure to beryllium levels at or above 100 µg/m^3. It produces severe cough, sore nose and throat, weight loss, labored breathing, anorexia, and increased fatigue. In addition to beryllium's toxicity when inhaled, when brought into contact with skin at relatively low doses, beryllium can cause local irritation and contact dermatitis, and contact with skin that has been scraped or cut may cause rashes or ulcers. Beryllium dust or powder can irritate the eyes. Therapy is supportive and includes removal from further beryllium exposure. For very severe cases mechanical ventilation may be required. The signs and symptoms or acute beryllium pneumonitis usually resolve over several weeks to months, but may be fatal in 10 percent of cases, and about 15-20% of cases may progress to CBD. Acute beryllium poisoning approximately doubles the risk of getting lung cancer. The mechanism by which beryllium is carcinogenic is unclear, but may be due to ionic beryllium binding to nucleic acids; it is not mutagenic. Acute beryllium disease was first reported in Europe in 1933 and in the United States in 1943.

Acknowledgment: The first two authors

(R.K. Pd. & Bina Rani) are thankful to UGC (ERO) for the award of a Minor Research Project during the period of research.

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