Research Paper E-ISSN NO : 2455-295X | VOLUME : 9 | ISSUE : 2 | FEBRUARY 2023

HEXAVALENT CHROMIUM INDUCED TOXICITY IN NATURE AND LIVING BEINGS

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ABSTRACT:

Hexavalent Chromium is widely used from chrome coated guns to chrome coated toilet seats, and from coated wooden furniture's to chrome coated automobile's, chromium coating till date is registered as one of the best superior qualities coating material for household and industrial applications. Chromium provides excellent corrosion, wear, shear, and tear resistance. But production of chrome electroplated materials of day-to-day use could lead to formation of highly toxic hexavalent chromium in nature. Chromium in hexavalent state is regarded as highly carcinogenic for living beings if ingested orally, respirational or cutaneously. Chromium hexavalent is widely known for causing cancerous mutation in cells of lungs, liver, kidney, digestive tract and even cell of skin. Presence of Chromium hexavalent in soil is known for causing alteration in functioning of enzymatic activity thus leading to cause greater disturbances in plant physiology and also results in impairment of the germination of seedlings. Interaction of human beings and marine life with hexavalent chromium contaminated water resources leads to serious implications of health on both the species. Whereas the deep reaching negative impact of inhalation of hexavalent chromium present in air leads to various cancerous problems related to nasal and sinus. Chromium hexavalent exposure to skin and eyes could lead to irritations, ulceration and even permanent damage to the eyes.

This article is an attempt to provide the gist of research studies, bulletins, news and collection of various pieces of information that are published at various platforms for Chromium hexavalent toxicity awareness, so as to communicate the deep reaching negative impact of contaminated environment due to chromium hexavalent on this living world.

KEYWORDS:

CARCINOGENIC, HEXAVALENT CHROMIUM, ENZYMATIC ACTIVITY, NASAL DISEASES, ELECTROPLATING.

INTRODUCTION

Chromium was first discovered in the mineral of crocoite (lead chromate) by a French chemist Louis Vauquelin in the year1798. It is a transition element and located in the group 6-B of the periodic table. Although Chromium exist from +2 to +6 oxidation states, only trivalent Cr(III) and hexavalent Cr(VI) species of chromium are of great relevance[1]. Chromium ions of valences Cr(II), Cr(IV), and Cr(V) have no stable water solution chemistry and primarily exist as transients intermediates between Cr(VI) and Cr(III) to acquire solid forms[2]. Every year approximately 20,000 tons of chromium is used as a metal worldwide due to its amazing non oxidizing property which maintains its surface stainless and shining even when it comes in contact with air. Chromium and its alloys are used in various industrial applications including steel, pigments, wood preservatives, electroplating, metal finishing, dyes, leather tanning, textiles and chemical industries etc[3]. Chromium is extracted from chromite ore ($FeCr_2O_4$), the majority of it is imported from South Africa and Kazakhstan. No chromite mines currently exist in the United States[4]. The process of electroplating using chromium began in the late 1920s. Electroplating with chromium provides a superior surface combined with excellent wear and corrosion resistance[5]. Chrome platings have top quality glint due to its more reflective (brighter), bluer (less pale, grayish, or yellowish), and more specular (the reflection is deeper, less distorted, more like a mirror) than other finishes. Chrome plating is an electrolytic process that can be applied on the surfaces of mild steel, stainless steel, aluminum, and many other materials of day to day use. Chrome platings are of two types: hard chrome plating and decorative chrome plating. Hard chrome plating is a heavy coating applied to those applications where materials are subjected to a very high degree of wear resistance. Hard chrome plating is usually 0.0008 to 0.005 inches thick and used for wear resistance, lubricity, oil retention, and to provide anti-galling properties. Decorative chrome plating is exceptionally thin measures in millionths of an inch i.e. 0.0002 to 0.0006 inch thick and always involves electroplating of nickel onto the object before plating the chrome, sometimes copper is electroplated onto the object before nickel is plated and then chrome plating is done[6]. Plating of chrome on metal surface takes place through number of stages. During the first step of electroplating the metal surface is thoroughly checked for any deposited foreign substances and then this metal is treated chemically, which ensures the metal is degreased and foreign material and unwanted tiny particles are cleaned from the surface and then a number of tests are performed in order to smoothen the metal surface so that it should have long lasting integrity. Afterwards this metal is then dipped in a vat consisting of

a solution which helps in gradual warming of the metal. The metal is warmed to an optimum temperature so that chrome spreads evenly on the surface. The actual plating is performed in this stage, the vat is filled with chromium components. These components are allowed to engrave the surface of the metal and depending on the required thickness of the chromium plating, the metal is kept in the vat[7].Chromium in pure metal form does not impose any environmental or health risks. However, the chemistry involved in present technology leads to produce hexavalent chromium ions as potentially carcinogenic deposits. Chromium electroplating is a categorically regulated industry, and as per EPA mixture and derived from rule the waste products from this industry even if the particular substance is so dilute that it is actually harmless cannot be mixed with other waste products or used to derive another product from it. The chrome plating industries are under the strict regulations of law and the wastes are always pretreated before disposal and have safe working facilities of exhaust scrubbing and fume suppressants and are monitored every day, along with regular medical health surveillance of the workers[8].

HEXAVALENT CHROMIUM INDUCED TOXICITY IN SOIL AND PLANT PHYSIOLOGY

When Hexavalent chromium gets released naturally or artificially into environment either in dry state or in soluble form, it is a highly toxic soil and groundwater contaminant. In many contaminated sites throughout the world hexavalent chromium poses a serious health risk to humans, animals and plants[9].Chromium present in trivalent oxidation state in the environment is less toxic because of its limited water solubility, resistance to weathering, diagenesis, and low-grade metamorphic reactions[10]. Studies show that trivalent chromium present in chromite ore which is generally considered as geochemically inert in nature easily undergoes oxidation when associated with common manganese mineral birnessite to form hazardous hexavalent chromium, this indicates that minerals of manganese that pervasively form as surface coating of soil minerals may serves as potential oxidants for converting Cr(III) to Cr(VI)[11]. The effect of chromium compounds on physiochemical and biological properties of soil depends mainly on soil pH, granulometric composition, redox potential and content of humus [12]. Chromium in hexavalent state is highly soluble in soils of acidic or alkaline nature and undergoes reduction to form trivalent chromium which is weakly soluble in acid and alkaline soils and are not readily available as mineral for plants growth. Hexavalent chromium exhibits harmful impact on biological activity of soil microorganisms[13] also and influence the enzymatic activity and composition of microflora of soil by modifying the environment in which microorganisms of soil live[14]. Enzymatic studies of dehydrogenases, urease, acid phosphatase and alkaline phosphatase showed great level of dependency on the rate of soil contamination caused by hexavalent chromium. Stronger inhibitory influence by hexavalent chromium were observed in presence of inoculated *bradyrhizobium* bacteria which are also known as *Nitrazine*. Several studies reveal that among all the enzymes dehydrogenases are least tolerant even to the lesser concentrations of even 10mg. kg^{-1} of Cr(VI), whereas concentrations of 10 to 40 mg. kg^{-1} were found to stimulate activity of urease to an extent, but further increase in concentration of hexavalent chromium lead to decreased activity of enzymes like urease, acid phosphatase and alkaline phosphatase. Minimal concentration of 10mg. $kg^{-1}$of Cr(VI) was found to cause symptoms of toxicity involving disturbed water balance, chlorosis of new leaves and damage of the growing point like roots and shoots. Concentration of 150 mg. kg^{-1} of Cr(VI) present into the soil lead to even inhibition in the emergence of plants. The symbiotic bacteria stimulated toxicity on the growth and development of lupine plants[15] were also observed. The process of uptake, translocation and accumulation of hexavalent chromium in plants causes detrimental effects to their growth and development. Hexavalent chromium is a nonessential element to plants and does not possess specific mechanisms for its uptake, therefore, it primarily depends on carriers involving essential anions and elements such as $SO4^{2-}$, Fe, S and P which are essential for plant metabolism[16]. The maximum accumulation of hexavalent chromium is reported in roots of the plants and a minimum in the vegetative organs. Natural toxicity response of the plant was observed by immobilization of hexavalent chromium in the vacuoles of the root cells[17]. Shankar. A et al[18] suggested that since hexavalent chromium has to cross the endodermis via symplast, there are chances of its reduction to trivalent state in cell itself thus causing lesser toxicity in the root cortex cells of plants. In many cases high levels of hexavalent chromium lead to depressive effect on the amylase activity and transport of sugars to the embryo axes and leads to increased protease activity and contributed in the reduction of germination of seeds. Hexavalent chromium also affects the process of CO_2 fixation, electron transport, and photophosphorylation during photosynthesis. Hexavalent chromium produces great impact on Photo system I activity by diverting the electrons from the electron donating sites of PS I and leading to decrease in photo synthesis rate as compared to Photo system II in an isolated chloroplasts of peas plants, But both the photo systems were found to be affected. Inhibition in the process of photosynthesis by the presence of hexavalent chromium was also observed between 40% -95% during the life span of 52-89 days of plant with the minimum quantity of 0.1 mM Cr(VI)[19,20]. Hexavalent chromium has also been known to interfere with uptake of several minerals and nutritients such as K, Mg, P, Mo, Fe and Mn in roots of soybean. The uptake of Fe in plants is also affected by hexavalent chromium by inhibiting reduction of Fe(III) to Fe(II). Hexavalent chromium also competes with Fe(II) at the site of absorption which leads to Fe-deficient conditions and thus results in chlorosis.

HEXAVALENT CHROMIUM INDUCED TOXICITY IN DRINKING WATER AND MARINE LIFE

Hexavalent chromium contaminated water is considered as one of the major sources of exposures to its hazardous impacts on both human beings and marine life. Hexavalent chromium in its oxides are known to an excellent water-soluble compound with the solubility of 1680 g. L^{-1} which is considered as very toxic and listed[21] as water hazard class 3. Hexavalent chromium detected in ground water samples in some parts of California and other states has led to serious public concerns about the safety of their drinking water sources. Studies were conducted for determining the toxicity and carcinogenic effect of hexavalent chromium by different government and non-government California based agencies such as The California Congressional Delegation, California Environmental Protection Agency, and the California Department of Health Services. Since the U.S government does not have separate policy for quantifying the presence of hexavalent chromium in water, the Environmental Protection Agency (EPA) has set a maximum contaminant level to be 100 µg.L^{-1} for total chromium present i.e. Cr(III) and Cr(VI) combined together in drinking water. Numerous states of America including California have established limits of 50 µg. L^{-1} of total presence of chromium in drinking water[22]. Burge. S et al[23] used an automated "universal system" capable of performing automated calibration, sampling and analysis without the requirement of any resident operator. This process encompasses monitoring the temporal changes of hexavalent chromium in the concentration range of 30 to 120 ppb with internal calibration for both shallow or deep down ground water. Saxena et al have studied the toxicological effects of hexavalent chromium on a freshwater fish named "*channa punctatus",* the toxic effect of hexavalent chromium was found to cause elevated levels of cholesterol and severe decrease in total protein and total lipid content[24] in this variety of fishes. Mishra. A and Mohanty.B[25] investigated cytotoxic effect and systemic toxicity of vital organs of "*channa punctatus fishes"* caused by hexavalent chromium induced toxicity. Histopathological effects due to acute and chronic exposures of hexavalent chromium were found to cause alteration in ovarian histology of "*channa punctatus"* thus leading to increase in percentages of atretic oocytes and decrease in percentage of vitellogenin oocytes in fishes with with acute and chronic exposures. Hexavalent chromium induced toxicity was also found to cause hepatocellular vacuolization and atrophy along with pyknotic nuclei. Oshida and Wright[26] used species of brittle star (*ophiothrix spiculata*) and sea urchin, (*strongylocentrotus purpuratus*) to study the sensitivity of these marine animals towards exposure to hexavalent chromium toxicity. Brittle stars subjected to the toxicant concentrations of 1.05 mg. L^{-1} to 2.10 mg. L^{-1} showed that the level of hexavalent chromium that causes a 50 percent reduction (LC_{50}) was 1.7 mg. L^{-1}. Whereas the LC_{50} in case of embryos of sea urchin during a 7 day study was between 2.9 and 29 mg. L^{-1} of hexavalent chromium. The LC50 value for chromium in sea fish lies between 7 to 400 ppm, for daphnia at 0.01-0.26 ppm, and for algae at 0.032-6.4 ppm. Recent animal studies conducted by National Toxicology Program suggested that oral ingestion of Cr (VI) in drinking water can cause cancer[27].

HEXAVALENT CHROMIUM INDUCED TOXICITY IN AIR

Hexavalent chromium is carcinogen and known to cause mutagenic effects in bacterial and mammalian cell systems, along with the ability to alter the DNA base sequence. Airborne emissions from chemical plants, incinerators, cement plants, tobacco smokes and combustion of fossil fuels are some of the major sources leading to dangerous levels of hexavalent chromium in air. The Occupational Safety and Health Administration (OSHA) states that Cr (VI) exposure leads to an increased risk of lung cancer and regular occupational exposure may also lead to asthma and damage of nasal lining[28]. In humans beings exposure to hexavalent chromium through inhalation has been known to cause cancers of lungs, nasal systems, respiratory irritation, severe nasal and skin ulcerations, lesions, perforation in the nasal septum, liver and kidney failure and also birth defects in new born[29]. Hexavalent chromium when comes in direct contact with skin may produce irritating and ulcerating effects and elicit an allergic response, characterized by eczema and dermatitis in sensitive individuals[30]. Xinggang. W investigated the correlation between incidence of chromium induced nasal diseases and working environment among the group of five chrome plating workers two of the workers, were found to have perforation in the nasal septum and the third worker had sever chromium-induced nasal disease indicating the incidence rate of 60%.

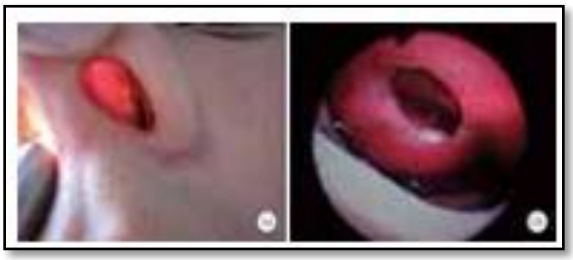

FIGURE 1: CHROMIUM-INDUCED NASAL DISEASE (XINGGANG. W. JR. OF CHIN. CLIN. MED., 4 (6), 2009).

The concentration of hexavalent chromium present in air recorded at the work place of these workers was 0.198 ± 0.186mg.m^{-3}, which largely exceeds the permissible concentration of 0.05mg.m^{-3}, thus clearly violating the standards set by National Sanitary Standard of China[31]. Yoon. C et al studied the effect of hexavalent chromium present in the fume generated during stainless steel welding using flux-cored arc with CO_2 gas. In this study the amount of total fume generated and the presence of hexavalent chromium in it was significantly correlated to the input power. As a function of input power, the increase in the concentration of total chromium from 1.57 to 8.13% in the fumes contained 0.15 to 1.08% of hexavalent

chromium[32].

EFFECT OF HEXAVALENT CHROMIUM INDUCED TOXICITY ON RODENTS AND GUNIEAPIGS

Wang X. et al, studied hexavalent chromium induced apoptosis in the liver of male swiss albino mice by administering 0, 25, 50, 100 mg. kg^{-1} body weight doses of potassium dichromate orally. Significant decrease in Bcl-2 protein level and an increasing tendency of Bax protein level in mice liver was observed. A consistent rate of hexavalent chromium induced apoptosis in mice liver was evident from dose-dependent increase in Bax/Bcl-2 protein ratio and change of p53 protein (a tumor suppressor protein) level.

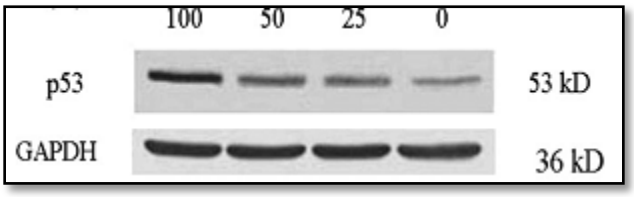

FIGURE 2: AUTORADIOGRAPH OF WESTERN BLOT SHOWING CHANGE IN P53 PROTEIN LEVEL IN MICE LIVER WHEN EXPOSED TO 0, 25, 50, 100 MG. KG^{-1} OF CR(VI) (ENVIR. TOXIC. 25, (1), 2009 BY XIAO-FENG WANG).

This increase of Bax and Bcl-2 protein ratio causes mitochondrial dysfunction including alteration of mitochondrial membrane potential, opening of the permeability transition pore, and the release of inter membrane space protein leading to significant increase in the levels of cytochrome c protein in hepatocyte's cytoplasm[33]. Agostini. F et al administered 0.25 mg. kg^{-1} body weight of sodium dichromate intra-tracheally in the "*Sprague Dawley"* rats to study induction of apoptosis in the lung by hexavalent chromium. Increase of the apoptotic index in both bronchial epithelium and lung parenchyma in hexavalent chromium treated rats was observed by TUNEL analyses. In the study cDNA array analysis showed significant enhancement in expression of apoptosis-related genes including c-Jun N-terminal kinases 1, 2 and 3, bcl-x, bcl-2-associated death promoter and bcl-2-related ovarian killer protein, caspases 1, 3 and 6, DNase I precursor, DNA topoisomerases I and II alpha, and poly(ADP-ribose) polymerase present in rats lung. Apoptosis in the respiratory tract due to hexavalent chromium leads to genotoxic effects and oxidative DNA damage in the lung tissues[34]. Studies conducted by Levy et al, reveled the development of only 1 bronchial carcinoma in 100 rats as a result of intrabronchial implantation of soluble sodium chromate whereas; intrabronchial implantation of two different dosage of strontium chromate induced 43 and 62 bronchial carcinomas in 99 rats[35]. Studies conducted by injecting lead chromate subcutaneously or intramuscularly induced significant sarcomas at the injection site indicating that lead chromate is also a carcinogen[36]. Nasu .T et al, studied inhibitory effects of hexavalent chromium ions on the contraction in ileal longitudinal smooth muscles of guinea-pigs[37]. Chorvatovicova. D studied simultaneous pretreatment effects of vitamins C and E on the toxicity and mutagenicity[38] caused by hexavalent chromium in rats and guinea pigs**,** simultaneous administration of both the vitamins C and E in Cr(VI)-intoxicated rats produced synergistic preventive effect which was confirmed by the production of lipoperoxides and also prevented the decrease in vitamin C levels in guinea pigs even though this decrease is provoked by the oxidative effects of hexavalent chromium. The micronucleus test in bone marrow showed that vitamin C produced an antimutagenic effect against bichromate, in both rats and guinea pigs and vitamin E resulted only an increase of the ratio of NCE to PCE, i.e., in a decrease of the cytotoxic but not the mutagenic effects of hexavalent chromium.

EFFECT OF HEXAVALENT CHROMIUM ON HUMAN LIFE

Danadevi. K et al, performed genotoxic evaluation by monitoring DNA damage in blood leucocytes among 102 occupationally exposed welders to chromium emitted during wielding. In this study comet assay method which depends on measurement of comet tail length to measure the basal DNA damage in the occupationally exposed welders was performed. The exposer among these occupationally exposed welders was found to be very high in levels for Cr content upto 151.65 $mg.L^{-1}$ in their blood plasma. Whereas, the micro nucleus test on buccal epithelial cells showed a significant increase in micro nucleated cells. The absorption of Cr in whole blood samples of welders was quantified and found to be 8 to 9 fold higher as compared to the control population[39]. Studies also reveals significant correlation between alkaline filter elution rate of DNA from lymphocytes and the frequency of DNA single-strand breaks with presence of high concentration of Cr in urine[40] as a induced toxicity due to hexavalent chromium. Holmes. A et al, studied and expressed a unique mechanism of particulate chromate induced genomic instability of the cell, which further leads to lung cancer. Hexavalent chromium in particulate form is capable of acquiring both anionic and cationic characteristics in dissolved state outside the cell. The cationic and intact particles of chromate enter into the cell through phagocytosis but do not impart any effect on the cell. The anionic chromate particles which are genotoxic in property enters the cell and gets rapidly reduced due to biological activity to Cr(III) and also produces Cr(V), although the Cr(III) is nontoxic in nature, but Cr in pentavalent state is very unstable and known for its capability of causing cancerous mutation of any living cells subjected to it. The highly oxidative natured Cr(VI) and reactive species of oxygen causes to form a ternary Cr-DNA adducts, which further leads to the formation of stalled DNA replication forks. The process of crosslink repair of Cr-DNA adducts which involves proficient nucleotide excision, results in a DNA double strand breaks. Another process of mismatch repair which includes series of futile repair cycles of Cr-DNA adducts ultimately fails

and collapses DNA replication forks and this also leads to DNA double strand break, this breaking of DNA double strand in chronic stages causes G2 arrest inducing both centrosome amplification and spindle assembly which further leads to numerical chromosome instability and ultimately leading to neoplastic transformation which turns into cancer[41]. Studies conducted by Reynolds. M et al, showed that in the presence of cellular vitamin C with lower doses of hexavalent chromium causes 10–15 times more chromosomal breakage in primary human bronchial epithelial cells. The co-localization of γH2AX and 53BP1 foci in cyclin B1-expressing cells evidenced DNA double-strand breaks in G2 phase, as a result of aberrant mismatch repair of damage caused by Cr in replicated DNA as DNA polymerase inhibitor aphidicolin and silencing of MSH2 or MLH1 by shRNA suppressed induction of gH2AX and micronuclei. The presence of ascorbate also results in the formation of centromere-negative micronuclei, indicating inefficiently repaired double-strand breaks. The genotoxic potential of Cr(VI) increases with increasing concentrations of ascorbate leading to increased double-strand breaks and genetic mutations, thus indicating that hexavalent chromium is more mutagenic in cells containing ascorbate[42]. Myers. C et al, performed studies based on NADPH-dependent rate of human microsomal reduction of hexavalent chromium in presence of iron and time-dependent oxidation of cytosolic (Trx1) and mitochondrial (Trx2) thioredoxin system, which plays key role in the maintenance of cellular thiol redox balance essential for cell survival in human bronchial epithelial cells [43,44].

EFFICIENT REMOVAL OF HEXAVALENT CHROMIUM

Morales. D et al,[45] illustrated the ability of streptomyces sp to adapt and resist in adverse conditions of elevated hexavalent chromium concentrations despite of its negative effect on growth and development. Streptomyces sp efficiently removed hexavalent chromium by promoting its reduction to trivalent state. Demirbasa. E et al, used low-cost agricultural waste such as cornelian cherry, apricot stone and almond shell as activated carbon adsorbents for removal of hexavalent chromium from aqueous solution. The almond shells[46] in acidic solution of pH 1 was effective enough for removal of hexavalent chromium upto 99.99 % at 25ºC.

Brigatti. M et al, studied interaction of hexavalent chromium interaction with Fe(II) bearing solid surfaces in aqueous solutions resulting in reduction to its trivalent state[47].Studies conducted by Gupta.V et al, indicated that the biomass of spirogyra species is suitable as an efficient biosorbent for the removal and recovery of Cr(VI) from wastewater[48]. Li Sun et al, developed, Chitosan modified Fe^0 nanowires in porous anodic alumina and used it as good adsorbents for the removal of Cr^{6+} ions from water by transforming the toxic Cr^{6+} ions transform into nontoxic Cr^{3+} ions after adsorption[49].

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