IJARSCT
International Journal of Advanced Research in Science, Communication and Technology
International Open-Access, Double-Blind, Peer-Reviewed, Refereed, Multidisciplinary Online Journal
ISSN: 2581-9429 Volume 8, Issue 1, August 2020 Impact Factor: 4.819

Chromium Pollution and Its Impact on Human Health

Naina Srivastava
Department of Botany
D.A.V. P.G. College, Dehradun, Uttarkhand, India
drnainasrivastava@gmail.com

Abstract: *Chromium gets into soil, water, and air both due to geological weathering and due to a broad spectrum of industrial processes, and the health effects of contamination are critically dependent on the oxidation state in which the metal is found. Trivalent chromium is a relatively stable and scarcely absorbed form, whereas hexavalent chromium is able to cross cell membranes easily and produces reactive intermediates that can cause injury to DNA and proteins. The present review reviews the literature, environmental behaviour and toxicological pathways of chromium especially focusing on the mechanisms that lead to the hexavalent chromium causing oxidative stress, genotoxicity and malignancy. The majority of the epidemiological evidence to date associating inhaled chromium(VI) with lung cancer has been obtained in occupational cohorts who are exposed either by electroplating, tanning, or chromate production and this data has been balanced against the more recent results of ingestion exposure. Also addressed in the review are organ-specific impacts on the respiratory system, renal system, hepatic system, dermatological system, reproductive system, and immune system, and concludes with a discussion on monitoring methods, remediation strategies and regulatory systems designed to reduce human exposure. Throughout missing dose-response data of low-level environmental exposure are noted as a priority in future research.*

Keywords: Chromium Pollution and Its Impact on Human Health

I. INTRODUCTION

Chromium holds a strange niche among environmental pollutants in that it is an important trace nutrient and a known human carcinogen at the same time. In 1797, the element was discovered by a French chemist Louis Nicolas Vauquelin in a Siberian mineral and its name is based on the Greek word of colour, a reference to the bright colours of most of the chromium compounds. This chemical versatility, the ability to be both in oxidation states as negative as –2 all the way up to +6, is what has made chromium so handy in industry, and such a pain in the neck in the environment. (Zayed & Terry, 2003).

As a trivalent chromium is involved in glucose and lipid metabolism in extremely small amounts, and was formerly commonly sold as a dietary supplement, but the evidence is now doubted (Jaishankar *et al*., 2014). The same element, in its hexavalent form, is nearly a different substance: dissolves freely, moves in groundwater, and can penetrate cell membranes unlike trivalent chromium can. This duality has influenced decades of regulatory discussion, since establishing a single safety level of "chromium" without indicating which oxidation state covers that level does not help much in the real world. (Agency for Toxic Substances and Disease Registry, U.S., 2012)

The world chromium ore output has increased to more than several million tonnes each year mainly due to the use of chromium metals in stainless steel and other metals that are alloyed with chromium. Whatever the site of ore mining or refining, or the use of the ore in manufacturing, some portion is leached off into the soil and water around the mining or refining site. Contamination levels differ vastly according to the location, with diffuse and low exposure near urban roads and cement plants, to intense local pollution around derelicted tanneries and chromate manufacturing sites, some of which are dangerous decades after being closed down. This review is a synthesis of the existing knowledge of the environmental behaviour and health impacts of chromium based on toxicological, occupational, and epidemiological articles published recently.

IJARSCT
International Journal of Advanced Research in Science, Communication and Technology
ternational Open-Access, Double-Blind, Peer-Reviewed, Refereed, Multidisciplinary Online Journal
ISSN: 2581-9429 Volume 8, Issue 1, August 2020 Impact Factor: 4.819

Sources and Environmental Distribution of Chromium.

Chromium is the twenty-first most commonly distributed element in the earth crust that is found naturally in ultramafic rocks, serpentine soils, and chromite mineral. Weathering of these formations discharges chromium into groundwater and surface water in amounts that are typically low but sometimes high, in some cases such as those in areas of serpentine geology (such as parts of California, Italy and Greece). The natural background levels are seldom of toxicological concern in themselves, but can add to man-made inputs to cause the local exposure to reach very high levels relative to guideline values (Agency for Toxic Substances and Disease Registry, 2012).

The vast majority of releases of chromium of concern in the view of the public health perspective are industrial activity (Kimbrough *et al*., 1999). Tanning of leathers is still among the greatest sources, as primitively tanning chromium sulfate is employed to fix collagen fibres in hides and tanning effluent has typically been released into waters with little treatment in most regions of the world, resulting in river sediments adjacent to tanning areas being highly enriched with chromium. The direct sources of hexavalent chromium waste streams are electroplating and metal finishing processes, since chromic acid baths are employed to apply ornamental and protective layers of chromium. Other contributes to the industrial burden, include stainless steel making, the processing of chromite ores, producing pigments (chrome yellow and chrome green pigments were once widespread), and even some textile dyeing.

Upon release, chromium is partitioned differently among environmental compartments both with respect to its oxidation state and the geochemistry of the surroundings. In soils with high organic matter and oxides of the iron or manganese, hexavalent chromium is likely to be reduced to the trivalent state, which is tightly bound to clay particles and made relatively immobile. This decrease is reduced under alkaline, oxidizing conditions, and the hexavalent chromium may be retained and people may travel long distances in ground water, a trend which has been recorded at polluted locations in New Jersey, Glasgow, and multiple industrial belts in China. Airborne chromium is primarily due to burning of fossil fuels, cement manufacture, and welding fumes, and although the content in the air is usually smaller than that in water or soil, the inhalation route exerts a disproportionate toxicological burden since it transfers hexavalent chromium directly to the lung tissue. (Bagchi *et al*., 2002).

Contamination of food chains Chromium-contaminated irrigation water or soil is absorbed by the crops and research of vegetables grown in the tanneries in India and Bangladesh has shown chromium levels several times higher than those grown in uncontaminated zones. Chromium has also been shown to be bioaccumulated by aquatic organisms although biomagnification along the food chain seems less effective than with metals like mercury, because chromium is often stored in gills and exoskeletons and not in muscle tissue. (Barceloux, 1999).

Chemistry and Shapes of Chromium.

To comprehend the toxicology of chromium, it is important to draw a line between the two oxidation states of chromium that are of importance to the environment. Cr(III) (trivalent chromium) is octahedral-shaped and stably complexes with oxygen and nitrogen donor ligands and is insoluble in water over most physiological pHs (Barceloux, 1999). It is the form of most rocks and soils and is generally considered to be the biologically poised, and relatively harmless, oxidation state. Cr(III) has a sluggish surface uptake owing to its inability to enter the cell by the transport channels to which other ionic species are able to enter and to which the little that does enter the cell is inefficiently retained.

The action of hexavalent chromium, Cr(VI), is very different. It occurs mainly in the form of the chromate (CrO_4^{2-}) or dichromate (CrO_7^{2-}) anion, which is very soluble and structurally related to sulfate and phosphate. This structural mimicry enables the Cr(VI) to gain access to cells via the anion transporters used by these vital nutrients, which trivalent chromium just does not have at its disposal (Dayan & Paine, 2001). Cr(VI) is further reduced in the cell, via a cascade of intermediate forms, Cr(V) and Cr(IV), into Cr(III) and it is the intracellular reduction, and not hexavalent chromium, that produces most of the reactive oxygen species that cause cellular damage. (Beaumont *et al*., 2008).

The difference is crucial to risk assessment. Simply declared regulatory limits of total chromium may be misleading, as a water sample with a large proportion of Cr(III) does not present the same degree of risk as one with the same total

IJARSCT
International Journal of Advanced Research in Science, Communication and Technology
ternational Open-Access, Double-Blind, Peer-Reviewed, Refereed, Multidisciplinary Online Journal
ISSN: 2581-9429 Volume 8, Issue 1, August 2020 Impact Factor: 4.819

concentration of Cr(VI). This is one motivation that the World Health Organization and the United States Environmental Protection Agency have shifted to speciation-specific guidance (World Health Organization, 2003; United States Environmental Protection Agency, 1998) in which it is possible using analytical methods, but in many monitoring programs, total chromium is more frequently reported than speciation analysis, which is costlier and more technically strenuous. (Costa & Klein, 2006).

Routes of Human Exposure.

The paths that people are exposed to chromium take a few overlapping routes, and the proportion of importance of each varies significantly depending on the occupation, geography, and diet. A high-profile route of exposure in contaminated aquifers is drinking water, and the town of Hinkley, California became a household name when hexavalent chromium leeching out of the cooling towers of a natural gas compressor station was discovered to have polluted the groundwater supply in the town over many decades. In some aspects of Italy, Mexico, and China, similar water pollution by drinking water has been reported, usually due to the emission of industrial effluent or poorly controlled waste ponds. (WHO, 2003).

Depending on the source of chromium, dietary intake has a smaller but non-trivial portion of the total exposure of most individuals, both through naturally occurring chromium in food and by contamination of food processing machinery or irrigation water. Since the amount of ingested hexavalent chromium is largely reduced to the trivalent form by gastric acid and saliva until it could be absorbed, a low dose of Cr(VI) taken orally, by comparison with the same dose taken inhaled, is significantly less toxic than a high dose of the same, although the reductive power in low doses can be surpassed at a very high dose of Cr(VI), which was the focal point of the drinking water litigation near Hinkley (Sedman *et al*., 2006).

The most evident and the most harmful health effects are the occupational inhalation route of exposure. Chromate producers, chrome platers, workers in stainless steel welding and chromium pigment manufacturing are capable of inhaling the aerosols of hexavalent chromium directly into the lower respiratory tract bypassing the gastrointestinal system protective reduction. The epidemiological basis of the evidence on chromium-induced lung cancer that is discussed later in this review is based on historical occupational cohorts in chromate plants in the United States, the United Kingdom and Germany that followed them over more than fifty years. (Dayan and Paine, 2001).

Another route, which is occupationally and in the general population, is dermal contact. Chromium compounds have been identified as a common cause of allergic contact dermatitis, and in the past, chromium-tanned leather products, cement and some cleaning products have been suspected to cause sensitization. Intact skin absorption is minimized, whereas broken skin or abraded skin permits much higher penetration, and this is especially important with construction workers who have to deal with wet cement.

Mechanisms of Toxicity of Chromium.

The toxicity of hexavalent chromium is most effectively considered as the result of intracellular reduction to a form that is toxic rather than an inherent property of the Cr(VI) ion. The passages that occur as the chromate is deprotonated in small steps to Cr(III) within the cell undergo unstable intermediate oxidation reactions that react easily with hydrogen peroxide and other reductants in the cell, resulting in the formation of hydroxyl radicals and other reactive peroxy species by Fenton-like chemistry (Bagchi *et al*., 2002). A widely cited review of the toxicology of chromium in humans by Costa and Klein (2006) identified this reductive activation as the central link in the chain between exposure and downstream cellular effects, a structure that has since been supported by mechanistic studies.

Lipids, proteins and nucleic acids are damaged indiscriminately by the oxidative stress produced in such a sequence of reductions. There are various types of DNA damage, such as oxidized bases, DNA-protein crosslink, and Cr-DNA adducts when the trivalent chromium generated at the end of the reduction cascade reacts directly with DNA. Since the Cr(III) affinity towards the phosphate and carboxylate functional groups is strong, the adducts are stable in chemistry and may last long enough to disrupt DNA replication and transcription, altering the double helix in a manner that

IJARSCT
International Journal of Advanced Research in Science, Communication and Technology
ternational Open-Access, Double-Blind, Peer-Reviewed, Refereed, Multidisciplinary Online Journal
ISSN: 2581-9429 Volume 8, Issue 1, August 2020 Impact Factor: 4.819

promotes mutagenesis. Work on chromium in drinking water exposure and metabolism has characterised these adducts as having a functional similarity to the well-established chemical carcinogen-induced DNA lesions (Zhitkovich, 2011).
When such genotoxic damage remains unproperly mended, it can cause chromosomal aberration, exchange of the sister chromosomes and even the formation of micronucleus, which have been reported to occur at high levels in workers exposed to chromium (Wise & Wise, 2012). The injury is inflammatory: the exposure of chromium to air leads to the nuclear factor kappa B and other pathways, which prompts the presence of the pro-inflammatory cytokines that causes the infiltration of the immune cells into the location of exposure, which is usually lung tissue in the workplace. Self-perpetuating cycles This type of chronic inflammation produces a self-perpetuating loop of immune cells producing more reactive oxygen species, and thus exacerbating the initial insult.
Cells adapt to accumulating damage by functioning through apoptotic signalling pathways designed to kill damaged cells before they can undergo abnormal proliferation, and chromium exposure does trigger apoptosis of a wide variety of cells in both mitochondrial and death-receptor mediated ways. This protective response is carcinogenic in that it becomes ineffective due to either excessive accumulation of damage quicker than it can be fixed or removed or due to interference with the same repair/apoptotic machinery that protects against malignant transformation. Various in vitro experiments have revealed that chromium had the ability to block nucleotide excision repair, essentially weakening one of the primary protective mechanisms of the cell against precisely the type of bulky DNA lesions that chromium itself is known to cause, a self-perpetuating process that helps account for chromium being termed a genotoxic carcinogen and not one that acts solely via non-genotoxic secondary pathways.

The Health Effects of Chromium Exposure.
The organ-specific effects of chromium exposure depend on route, dose and duration of exposure and the oxidation state to which it is exposed. The following is briefing the major systems that are involved which is based on both occupational cohort and experimental toxicology.

Respiratory Effects
Chromium affects the lung, the organ system most frequently and severely impacted by the metal, in this regard the lung is the most important in the occupational inhalation exposure. Acute high level exposure may result in nasal mucosa irritation and long-term exposure of chromate workers has been linked to nasal septal perforation which is such a common lesion that it was previously almost diagnostic of chromate plant employment. Exposed cohorts have been reported to have chronic bronchitis, impaired pulmonary function, and asthma-like symptoms, although possibly most effectively, lung cancer. (IARC, WHO, 1990).
In its first monograph on chromium compounds, the International Agency for Research on Cancer (1990) classified some hexavalent chromium compounds as Group 1 human carcinogens, and with repeated reviews, risk increased with cumulative exposure. Subsequent epidemiological research, such as chromate plant studies in Baltimore and stainless steel welder studies in multiple countries, has generally supported such a dose-response relationship, but it has been hard to determine exact exposure-response curves due to incomplete exposure records of the past. (Jaishankar *et al*., 2014).

Dermatologic, Hepatic, and Renal Effects.
The two clinical patterns are produced when a person comes into contact with chromium compounds by means of the skin. Irritant contact dermatitis is caused by direct chemical damage, and may arise after a single exposure at high concentrations, whereas allergic contact dermatitis arises during sensitisation in individuals who have been exposed to chromium and may last years after the agent is stopped, as chromium allergy once acquired is thought to be permanent. Another occupational finding is chromium ulcers also known as chrome holes, which are painless deep lesions that form at locations where skin breaks occur in employees who handle chromic acid.

IJARSCT
International Journal of Advanced Research in Science, Communication and Technology
International Open-Access, Double-Blind, Peer-Reviewed, Refereed, Multidisciplinary Online Journal
ISSN: 2581-9429 Volume 8, Issue 1, August 2020 Impact Factor: 4.819

Postabsorption systemic chromium toxicity has an overburdened burden on the liver and kidneys, the major organs of xenobiotic metabolism and excretion. Studies in animals demonstrate a consistent hepatocellular damage, increase in liver enzymes, and histopathological alterations following the administration of chromium, and case reports of accidental or intentional ingestion of hexavalent chromium in humans report acute liver failure and renal injury. It seems that the kidney is especially susceptible since it is the primary route of chromium excretion, concentrating the compound in renal tubular cells during filtration; acute tubular necrosis has been reported following exposure to high doses, and chronic exposure to low doses has been associated in many studies with less obvious indications of renal dysfunction including high urinary beta–2–microglobulin. (Kimbrough *et al*., 1999).

Nervous, Cardiovascular, Reproductive, and Immune Effects.

There is somewhat limited, though not no evidence that chromium has effects on the nervous system. Occupational investigations have reported neuro-behavioral alterations such as memory impairment and decreased nerve conduction velocity, as well as animal studies have demonstrated that chromium has the ability to pass through the blood-brain barrier and accumulate in the brain tissue, causing oxidative damages to neural cells. Such results are not as well replicated as the respiratory and dermatological sources.

Increasing focus has been directed towards cardiovascular effects. Populations exposed to chromium have been found to be associated with disturbed lipid profiles, and increased signs of vascular inflammation in some studies, which have yet to be conclusively converted into clinically significant changes in the incidence of cardiovascular disease, but which are likely to depend on the intensity and duration of exposure.

Reproductive toxicity has been most effectively illustrated on animal models, which are impaired in spermatogenesis, a decrease in sperm motility and, at high doses, result in developmental defects in the offspring. Human data are more sparse and complex due to co-exposure to other industrial contaminants, and a number of studies of male workers in chromium related industry have shown less semen quality and some data exists of maternal chromium exposures leading to worse pregnancy outcomes, an area that still needs much more research. Immune effects involve sensitization (underlying the allergic dermatitis above) and at higher exposure levels, inhibition of some immune functions, with in vitro and animal studies demonstrating the changed lymphocyte proliferation and cytokine production after exposure to chromium (Shrivastava *et al*., 2002).

Vulnerable Populations

Some groups have a disproportionate burden of risk with chromium. The workers in the industries are the most extensively investigated and highly exposed population, and even with the improved occupational hygiene standards since the 1990s chromate manufacture and chrome plating still presents a quantifiable risk in the facilities where there is less strict adherence to the regulations. Children are less resistant to the effects of much of chromium than adults, being proportionately more susceptible to a given dose of chromium depending on body weight and passing through a faster neurological and immune developmental process, which can be derailed more easily. Chronic exposure at low-levels that occurs in communities near the contaminated industrial sites, usually in low-income regions with limited access to other water sources, is harder to epidemiologically study as compared to higher better-characterized doses in occupational settings but nonetheless a large burden to the overall population due to the number of people affected. Another at risk group is pregnant women due to the previously mentioned reproductive and developmental outcomes, but the human data in this group are still in its early stages.

Environmental and Public Health Concerns.

Outside the human toxicity, there exist larger environmental issues that are caused by chromium contamination and which feed back into human health. Polluted soils contain chromium that may suppress microbial activity and plant growth, change nutrient cycling and decrease agricultural productivity in contaminated regions, which has been extensively documented in tannery clusters near Kanpur, India, one of the most chromium-affected areas that have been

IJARSCT
International Journal of Advanced Research in Science, Communication and Technology
International Open-Access, Double-Blind, Peer-Reviewed, Refereed, Multidisciplinary Online Journal
ISSN: 2581-9429 Volume 8, Issue 1, August 2020 Impact Factor: 4.819

studied in the environmental literature. Polluted river and estuary sediments may be a source of chromium release decades after the source has been halted, because chromium attached to particulate matter may be freely released again by changes in sediment chemistry, such as during flooding, dredging, and so forth. (US Environmental Protection Agency, 1998).

Chromium pollution has an unequal effect on the public health. In regions where regulatory infrastructure and industrial wastewater treatment are developed such as most of Western Europe and North America since the 1970s, there has long been rare occurrence of large-scale chromium contamination events, although legacy contamination by old industrial facilities remains a long-term burden. In places where tanning, electroplating and chromite mining are still practiced, with little environmental regulation, the level of contamination has recorded in recent decades have in certain instances been equal or even higher than those that were recorded in Western sites long since remediated during the period of their most active industrial use. This imbalance has led to growing demands by scholars of public health that there should be internationally coordinated monitoring standards, but implementation is inconsistent. (Wise & Wise, 2012).

II. PREVENTION, CONTROL AND MANAGEMENT STRATEGIES

The nature of the intervention needed to reduce chromium exposure involves intervening at multiple points in the pathway between industrial source and human contact. Process additions that can decrease the use of hexavalent chromium have been demonstrated at the source; there are chromium-free tanning agents and trivalent chromium plating baths, which can replace the old hexavalent chromium processes, but their uptake has been slow due to the consideration of cost and performance in some applications.

The most easily control point that can be applied immediately to the current industrial operations is wastewater treatment. A long-established and relatively low-cost treatment, which involves the reduction of Cr(VI) to Cr(III) with reducing agents, e.g. ferrous sulfate or sodium metabisulfite, followed by precipitation and filtration, is a well-established and readily available treatment method, and more recent studies have sought to investigate biological reduction using chromium-tolerant bacteria and fungi, and adsorption onto low-cost materials including agricultural waste products and activated carbon, as more sustainable alternatives applicable to resource-limited environments (Zayed & Terry, 2003).

Regulatory control has significantly helped to minimize the exposure in areas where it has been implemented regularly. In 2006, after a reassessment of the carcinogenicity evidence, the allowed exposure limit of hexavalent chromium was considerably reduced by the United States Occupational Safety and Health Administration, and an equivalent tightening has been done in the European Union and other jurisdictions. Standards of drinking water on total chromium, established by the World Health Organization and national bodies offer additional protection, but as has been noted above, standards not based on the oxidation state can fail to reflect risk in a region dominated by Cr(VI).

In those places where the use of hexavalent chromium cannot be done away with completely, occupational safety measures, including engineering controls like local exhaust ventilation, respiratory protective equipment, and periodic biological monitoring of exposed workers, are still required. Biomonitoring methods, mainly urinary chromium analysis at the conclusion of a work shift, enable exposure to be monitored over time and offer an early warning against insufficient controls, however the interpretation must be done cautiously since urinary chromium is a reflection of current exposure rather than cumulative body burden. Response strategies to contaminated communities have involved provision of alternative drinking water supply, soil remediation by excavation or in situ chemical treatment and health surveillance programs but the resources to fully undertake the remediation efforts usually surpass the resources found in the lower-income areas where contamination has often been the most intense.

III. FUTURE PERSPECTIVES

There are still a number of unresolved spheres of chromium toxicology that could be addressed in further research. The dose-response relation of the low dose environmental exposure, especially by consumption of drinking water as opposed to the occupational exposure to these toxins, is not as well characterized as the high dose occupational data

IJARSCT
International Journal of Advanced Research in Science, Communication and Technology
International Open-Access, Double-Blind, Peer-Reviewed, Refereed, Multidisciplinary Online Journal
ISSN: 2581-9429 Volume 8, Issue 1, August 2020 Impact Factor: 4.819

that has formed the basis of most regulatory limits, and the attempts at extrapolation of the high dose occupational data to the environmental exposures are fraught with uncertainty (Beaumont *et al*., 2008). Greater improvements in routine speciation methods of chromium in environmental and biological samples would enable risk assessment to cease to be based on the total chromium measurements and commence based on assessments that capture the real risk of toxicology.

Reproductive and developmental impacts of chromium on humans also require further research as the animal evidence is almost at an advanced stage compared to the human one. Innovative forms of remediation technology, especially bioremediation with chromium-reducing microorganisms and phytoremediation with hyperaccumulator plant species provide potentially more sustainable and cost-effective solutions to contaminated sites, although most of this has been at the pilot or laboratory scale and needs to be validated in the real field conditions before any broader use can be suggested. Lastly, bridging the disparity between regulatory controls in the industrialized and developing world, as well as ensuring the wastewater treatment facilities remain up to date with the ongoing increase in chromium-intensive production will probably decide whether the number of people suffering chromium-related illness will decrease or will remain concentrated in those regions with less environmental regulation.

IV. CONCLUSION

Chromium pollution is the example of how a single element can pose radically different dangers depending upon the chemical form, a fact that has direct implications on the way contamination is now monitored and controlled. The toxicological and epidemiological history of hexavalent chromium, especially its ability to induce lung cancer by inhalation, is significant and remains consistent throughout a series of independent cohorts, but the extent of risk due to lower-level environmental exposure via water and food is still uncertain. The effective response to the problem of chromium contamination will require further development of exposure science, increased use of less harmful industrial methods, and increased uniformity of protective norms throughout the areas where industries that are dependent on chromium further grow.

REFERENCES

1. Agency for Toxic Substances and Disease Registry. (2012). Toxicological profile for chromium. U.S. Department of Health and Human Services, Public Health Service.
2. Bagchi, D., Stohs, S. J., Downs, B. W., Bagchi, M., & Preuss, H. G. (2002). Cytotoxicity and oxidative mechanisms of different forms of chromium. Toxicology, 180(1), 5–22.
3. Barceloux, D. G. (1999). Chromium. Journal of Toxicology: Clinical Toxicology, 37(2), 173–194.
4. Beaumont, J. J., Sedman, R. M., Reynolds, S. D., Sherman, C. D., Li, L. H., Howd, R. A., Sandy, M. S., Zeise, L., & Alexeeff, G. V. (2008). Cancer mortality in a Chinese population exposed to hexavalent chromium in drinking water. Epidemiology, 19(1), 12–23.
5. Costa, M., & Klein, C. B. (2006). Toxicity and carcinogenicity of chromium compounds in humans. Critical Reviews in Toxicology, 36(2), 155–163.
6. Dayan, A. D., & Paine, A. J. (2001). Mechanisms of chromium toxicity, carcinogenicity and allergenicity: Review of the literature from 1985 to 2000. Human & Experimental Toxicology, 20(9), 439–451.
7. International Agency for Research on Cancer. (1990). Chromium, nickel and welding (IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Vol. 49). World Health Organization.
8. Jaishankar, M., Tseten, T., Anbalagan, N., Mathew, B. B., & Beeregowda, K. N. (2014). Toxicity, mechanism and health effects of some heavy metals. Interdisciplinary Toxicology, 7(2), 60–72.
9. Kimbrough, D. E., Cohen, Y., Winer, A. M., Creelman, L., & Mabuni, C. (1999). A critical assessment of chromium in the environment. Critical Reviews in Environmental Science and Technology, 29(1), 1–46.

IJARSCT
International Journal of Advanced Research in Science, Communication and Technology
International Open-Access, Double-Blind, Peer-Reviewed, Refereed, Multidisciplinary Online Journal
ISSN: 2581-9429 Volume 8, Issue 1, August 2020 Impact Factor: 4.819

10. Sedman, R. M., Beaumont, J., McDonald, T. A., Reynolds, S., Krowech, G., & Howd, R. (2006). Review of the evidence regarding the carcinogenicity of hexavalent chromium in drinking water. Journal of Environmental Science and Health, Part C, 24(1), 155–182.
11. Shrivastava, R., Upreti, R. K., Seth, P. K., & Chaturvedi, U. C. (2002). Effects of chromium on the immune system. FEMS Immunology & Medical Microbiology, 34(1), 1–7.
12. United States Environmental Protection Agency. (1998). Toxicological review of hexavalent chromium (CAS No. 18540-29-9), in support of summary information on the Integrated Risk Information System (IRIS). U.S. EPA.
13. Wise, S. S., & Wise, J. P., Sr. (2012). Chromium and genomic stability. Mutation Research/Fundamental and Molecular Mechanisms of Mutagenesis, 733(1–2), 78–82.
14. World Health Organization. (2003). Chromium in drinking-water: Background document for development of WHO guidelines for drinking-water quality. WHO Press.
15. Zayed, A. M., & Terry, N. (2003). Chromium in the environment: Factors affecting biological remediation. Plant and Soil, 249(1), 139–156.
16. Zhitkovich, A. (2011). Chromium in drinking water: Sources, metabolism, and cancer risks. Chemical Research in Toxicology, 24(10), 1617–1629.