INTERNATIONAL JOURNAL OF
Clinical and Pharmaceutical Innovations
An International Peer Reviewed Open Access Journal

Review Article

ISSN (Online): 3142-8665
CODEN (USA): IJCPSM

2026, Volume 1, Issue 5, Page No: 187-192.

MEDICINAL PLANTS FOR THE ALLEVIATION OF CHROMIUM-INDUCED TISSUE TOXICITY: A COMPREHENSIVE REVIEW

Soumita Dey[1], Amit Nandi[1], Sandip Kumar Sinha[1], Sankar Kumar Dey*[2]

[1]Department of Human Physiology, Vidyasagar University, Midnapore-721102, West Bengal, India.
[2]Department of Physiology, SBSS Mahavidyalaya (Affiliated to Vidyasagar University), Goaltore-721133, Paschim Medinipur, West Bengal, India.

Article Info: **Received:** 24 June 2026, **Revised:** 14 July 2026, **Accepted:** 04 August 2026

***Corresponding Author: Sankar Kumar Dey**

Department of Physiology, SBSS Mahavidyalaya (Affiliated to Vidyasagar University), Goaltore-721133, Paschim Medinipur, West Bengal, India.

Citation:
Soumita Dey[1], Amit Nandi[1], Sandip Kumar Sinha[1], Sankar Kumar Dey*[2]. (2026). Medicinal Plants For The Alleviation Of Chromium-Induced Tissue Toxicity: A Comprehensive Review. International Journal of Clinical and Pharmaceutical Innovations, 1(5), 187-192.
DOI: https://doi.org/10.5281/zenodo.21827476

ABSTRACT
The accumulation of the heavy metal chromium (Cr) in different organs such as liver, kidneys, heart, and lungs can cause a negative impact on human health. Inhalation is one of the most frequent ways that people are exposed to Cr, and it is linked to harm. Much of the damage produced by Cr poisoning has been related to increased generation of reactive oxygen species (ROS). The body's antioxidant system balance has been observed to be impacted by a redox imbalance brought on by Cr-induced ROS-mediated oxidative stress. Thus, the positive impact of antioxidant nutrients through exogenous antioxidant molecule supplementation may be linked to either lowering the likelihood of Cr interacting with essential biomolecules and causing oxidative damage or enhancing the antioxidant defenses of the cell. This review addresses to discuss the advantageous effect of herbal plants in tissue damage caused by chromium.

KEYWORDS: Chromium, Tissues, Toxicity, Medicinal Plants.

INTRODUCTION

Urbanization often leads to increased pollution as cities grow, heavy metal pollution can serve health consequences for humans. Exposure to heavy metals such as lead, mercury, cadmium, chromium, arsenic etc are most abundant metal in the earth's crust and a potent environmental contaminant. In different industry used these types of toxic metal from several decades which affects living bodies as well as human. Hexavalent chromium is one of them toxic industrial pollutant that is classified as a human carcinogenic agent by several regular and non regular agencies.

CHEMISTRY OF CHROMIUM

Chromium is a hard metalloid, lustrous, ubiquitously available in the earth's environment in dual personality. Chromium has several oxidative state range from 2-6, but most active form is chromium (III) and chromium (VI).[1] Chromium forms stable coordination complexes due to its ability to accept and donate electrons from its d-orbital's. This property makes it useful in catalysis and as a component in coordination compounds.

SOURCES OF CHROMIUM

According to the Toxics Release Inventory, in 1997, the estimated releases of chromium was 1111,384 pounds to water from 3391 large processing factories which accounted for about 0.3% to total environmental releases.[2] Chromium used in several metal industries like electroplating, leather tanning, textile manufacture industries, welding of alloys or steel, wood preservatives etc. Chromium compounds are used as industrial catalysts and pigments (in bright green, yellow, red and orange colors). Chromium is second most important chromophore which helps to colored gemstone, like

rubies get their red color from chromium and emerald get their bright green color.[1]

UPTAKE OF CHROMIUM

Chromium (VI) which use in different factories is enters directly or indirectly into worker's body through skin, nasal, food etc. According the report of Mine Safety and Health Administration the mining industry in India employs over 5,60,000 people on a daily average, which comprises 13% of the private sector and 87% of the public sector.[3] If we see the average intake of chromium in our daily life through air it may be less than 0.2 -0.4 µg, through the water 2.0 µg and through the food it may be 60 µg. A large number of people or workers are potentially exposed to chromium almost every day. The highest potential exposure occurs in the metallurgy and tanning industries, where workers may be exposed to directly or indirectly under the high chromium-air concentrations. Their skin expose directly to the chromium during the use of chromium products such as wood treated with copper dichromate or leather tanned with chromic - small percentage of ingested chromium will enter the body through the digestive tract also.

ACCUMULATION, DISTRIBUTION AND EXCRETION OF CHROMIUM

After ingestion of chromium in our body through inhalation or through food it found ultimately in blood stream, by the help of small intestine. Once chromium arrives into the blood then spread to the different organs and tissues, including spleen, lungs, gastrointestinal tract, and adipose tissues. Experimental studies in Japanese quail proved that continue exposed to chromium can weighted some organs like liver kidney, spleen and bone tissues.[4]

The extent of distribution depends on factors like the duration and level of exposure. Some chromium particles enter by nose may be remaining long time in the upper part of the lungs and likely to be coughed up and swallowed or passes to the blood.[5] Chromium can be eliminated through the urine by the help of kidney or through the faces by the biliary excretion from liver. Minimum amount of chromium also excreted through sweat.

METABOLISM OF CHROMIUM

Chromium (VI) is rapidly taken by erythrocytes after absorption and reduced to chromium (III) inside the RBCs but binds directly to transferrin, an iron transporting protein in the plasma.[6] Cr (VI) is structurally very similar to sulphate and phosphate anions, facilitating swift uptake by the membrane non-specific anion transporters then reduced back into lower oxidative state chromium (V), chromium (IV) then ultimately chromium (III) which is a stable form of chromium. Therefore many hydroxyl ions are formed via fenton mechanism during this reduction reaction of hexavalent chromium in cells which increased ROS generation.[7]

TOXICITY OF CHROMIUM

Through the inhalation, accumulation, distribution and excretion, via this route chromium exposure occurs has been implicated in damage to the liver, kidneys, cardiovascular disorders, GI track damage and also cancer in different organs.[8] There are several theories on the mechanisms of chromium carcinogenesis or toxicity, including the generation of free radicals, the certain of Cr-DNA adducts, it should be able to double breakage, single strand breaks in DNA, and cross–links between proteins and DNA.[9] Additionally apoptosis has been reported as a result of chromium toxicity, which is mediated or regulated by reactive oxygen species (ROS) generation. Research has indicated that an increase in ROS generation is mostly responsible for the damage of liver and kidney tissues caused by chromium poisoning which is described in below -

A. Liver Toxicity

It is well known that the liver is the main organ which is involved in the xenobiotic metabolism and is a major target organ for chemicals and drugs. Hepatotoxicity is therefore an important endpoint to evaluate the effects of particular xenobiotics. Several studies are suggesting chromium as a hepatotoxin.[10] But the mechanisms of chromium induced hepatotoxicity at the molecular level are not clear. Indeed, hepatotoxic effects were shown previously in experimental animals as evidenced by large necrotic areas, destruction of the tissue general architecture as well as increased serum transaminases and lactate dehydrogenase.[11] Reduction of Cr (VI) has been shown to yield reactive species such as free radicals, superoxide anions, and hydroxyl radicals, possibly through Fenton like reactions of Cr (V) and Cr (IV) with hydrogen peroxide. Although the direct relationship between DNA reactive oxygen species (ROS) and chromium induced DNA damage is heavily debated and unclear, there have been several studies supporting the role of ROS in Cr (VI) induced genotoxicity, cytotoxicity, and oxidative.[12] Once Cr (VI) enters into the cells, it is rapidly reduced to Cr (III) by GSH and other antioxidants. Chromium is known to produce oxidative damage in the liver by enhancing the peroxidation of membrane lipids, a deleterious process slowly carried out by free radicals. Although $K_2Cr_2O_7$ itself does not generate free radicals directly, it indirectly generates various radicals like superoxide, hydroxyl, and nitric oxide, thus causing damage consistent with oxidative stress. The free radicals attack the cell membrane, thus leading to the destabilization and disintegration of the cell membrane as a result of lipid per-oxidation damage the normal function of liver.[13]

B. Kidney Toxicity

According to IARC chromium (VI) is considered as a class I human carcinogen.[14] When chromium (VI) enters in our body, it can be converted to chromium (III) which is relatively less toxic. However, this conversion process leading to cellular damage through oxidative stress.[15] There are several studies already proved that chromium

is a leading cause of kidney damage. There is reason to suspect that chromium contributes to the development of chronic renal failure of its instability in biological materials. This adverse effect of chromium depending upon how much chromium enters in our body for how long time. Study on ratsrevealed that kidney tissue exposed to chromium had changes its outer part of the nuclear membrane and mitochondria membrane. Endothelial cells and the pedicles were also seen to be damaged upon chromium exposure. Another study on rats has shown that exposure of kidney tissue to chromium compounds caused a shrinking of the nucleus and chromatin migration. Loss of cristae and variability in mitochondrial size was seen as well. Some research has also shown that chromium exposure in the form of potassium dichromate caused fragmentation of the mitochondria, irregular shape of the mitochondria and a reduction in the size of it the kidney.[16] According to Berndt (1976) both Cr (III) and Cr (VI) are uptake by the peroximal tubules, but hexavalent chromium can produce tubular necrosis and acute renal failure because chromium (III) is less toxic then Cr (VI).[17]

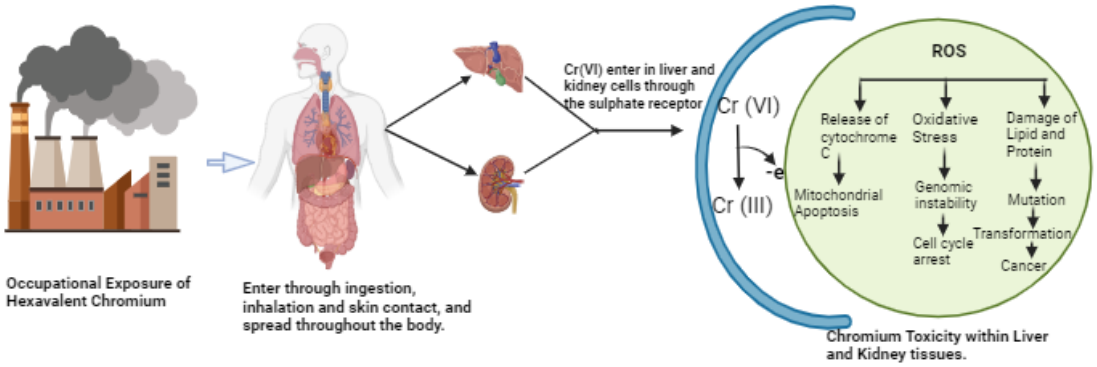


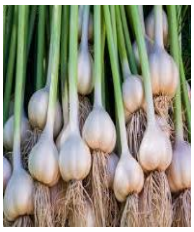
Figure: Mechanism of liver and kidney toxicity in response to chromium.

PROTECTIVE ROLE OF MEDICINAL PLANTS AGAINST CHROMIUM TOXICITY

Herbal medicines are rich in antioxidants, flavonoids, and polyphenols, which can mitigate chromium-induced oxidative stress and cellular damage. Researchers have recently become interested in medicinal herbs as possible remedies for heavy metal poisoning. The bioavailability and gastrointestinal absorption of heavy metals are decreased by chelating compounds found in certain plants. By promoting gastrointestinal motions that speed up the elimination of toxicants through the stool, herbal remedies might reduce the bioavailability of harmful compounds. If ingestion is the route of exposure, this might be seen as an auxiliary strategy to lessen the consequences of heavy metal poisoning. Various previous researches suggest that consuming herbal items on a daily basis can greatly minimize heavy metal absorption.[18] Several herbal medicines have shown protective effects against Cr VI toxicity by enhancing antioxidant defenses, reducing inflammation, and scavenging free radicals which are described in a table.

Table: Plants with Protective Properties Against Chromium Toxicity.

Plants	Plants part used in Phytoremediation	Bioactive compounds	Protective mechanism against chromium toxicity	References	Plant Species
Withania somnifera	Roots	➢ Alkaloids ➢ Steroidal flavonoids ➢ Tannins ➢ Saponins	● W. somnifera is rich in antioxidant phytochemicals, likely reducing DNA damage by Scavenging free radicals. ● Enhancing cellular antioxidant- defenses, possibly inhibiting chromosomal breakage or mitotic disruption.	[19,20]	

Azadirachta indica	Leaves	➢ Azadirachtin, ➢ Nimbin ➢ Quercetin,	● Functional groups on neem leaves (hydroxyl, carboxyl, etc.) actively bind and reduce Cr(VI) toxicity. ● Immunomodulatory and detoxifying properties against chromium toxicity. ● Protects against genotoxicity in aquatic models immunoprotective effects.	[21,22]	
Mangifera indica	Leaves	➢ Mangiferin ➢ Phenolic acids	● Mango wastes are rich in phenolic and gallotannin compounds able to chelate and reduce Cr (VI). ● Mango leaves demonstrated effective absorption of chromium (VI) from both laboratory solutions and actual industrial effluent.	[23]	
Allium sativum	Bulbs	➢ Allyl cysteine ➢ Allicin ➢ Diallyl disulfide (DADS) & Diallyl trisulfide (DATS) ➢ Ajoene ➢ Phenolic compound	● Sulfur compounds chelate chromium. ● Organosulfur compounds bind heavy metals Enhances glutathione synthesis Allicin traps free radicals and prevent cytotoxic effect. ● Protective effect of Aqueous Garlic extract attributed to the presence of bioactive compounds in garlic, such as organosulfur compounds (e.g., allicin, S-allylcysteine), known for their antioxidant and membrane-stabilizing properties.	[24,25]	
Aloe barbadensis miller	Leaves gel	➢ Aloin and Aloe-emodin (anthraquinones) ➢ Polysaccharides (notably acemannan) ➢ Vitamins (A,C,E) ➢ Phenolic compounds (flavonoids, tannins)	● Antioxidants that scavenge reactive oxygen species (ROS) generated by chromium toxicity. ● Bioactive compounds suppress inflammatory responses.	[26]	
Phyllanthus emblica	Fruits	➢ Ascorbic acid (Vitamin C) ➢ Gallic acid ➢ Ellagic acid ➢ Tannins ➢ Flavonoids	● Amla scavenge reactive oxygen species (ROS). ● Amla's antioxidants inhibit lipid peroxidation, protecting membrane integrity. ● Amla treatment modulates and normalizes these enzyme activities	[27]	

Panax ginseng C.A. Meyer	Roots	➢ Ginsenosides ➢ Polysaccharides ➢ Peptides ➢ Polyacetylenes ➢ Phenolic compounds	● Reduction of free radical generation: Prevents lipid peroxidation ● Enhancement of endogenous antioxidative enzymes: SOD, CAT, GPx etc. ● Helps reduce tissue inflammation caused by chromium toxicity.	[28]	
Brassica oleracea var. *italica*	Flower heads	➢ Sulforaphane (SFN) Glucosinolates ➢ Flavonoids ➢ Phenolic acids	● Akt activation: Promotes cell survival and inhibits apoptosis. ● Inhibition of GSK-3β: Reduces oxidative stress and prevents mitochondrial damage. ● Regulation of Fyn kinase: Helps stabilize cellular redox balance.	[29]	
Andrographis paniculata Nees	Leaves	➢ Andrographolide (primary and most studied compound) ➢ Neoandrographolide ➢ Deoxyandrographolide ➢ Flavonoids ➢ Diterpenoids	● Scavenges free radicals (ROS) generate ● Prevents lipid peroxidation in cell membranes. ● Enhancement of endogenous antioxidant enzymes: CAT, SOD, GPx. ● Reduces oxidative membrane damage, preserving structural integrity of cells.	[30]	
Ocimum sanctum	Leaves	➢ Eugenol – a major phenolic compound found. ➢ Ursolic acid ➢ Linalool ➢ Rosmarinic acid ➢ Ocimene	● Chlorophyll degradation: Interferes with pigment biosynthesis, reducing photosynthesis. ● Proline Accumulation: Acts as an osmoprotectant and ROS scavenger. ● Eugenol Biosynthesis: May be disrupted due to interference in secondary metabolism. ● Defense Activation: Antioxidant enzymes (SOD, CAT, GPX) increase to neutralize ROS.	[31]	

CONCLUSION

Chromium contamination is a major environmental concern as it is toxic. This contamination has adverse effects on ecosystems, including harmful impacts on soil, air, water which can reduce enzymatic activities that interference with growth and development. Phytoremediation presents a promising approach to remediate chromium-contaminated environments. While phytoremediation has its limitations, it is a valuable tool in environmental remediation techniques, and further research and development can enhance its effectiveness and expand its applications.

REFERENCES

1. Kawanishi, S.; Hiraku, Y.; Murata, M.; Oikawa, S. 2002. The role of metals in site-specific DNA damage with reference to carcinogenesis. Free Radic Biol Med., 32(9): 822-832.

2. ATSDR. 2000. "Toxicological Profile for Chromium." U.S. Department of health and human services.
3. Dhatrak, S.; Nandi, S. S. 2009. Risk assessment of chronic poisoning among Indian metallic workers. Indian J Occup Environ Med., 13(2): 60.
4. Patlolla, A. K.; Barnes, C.; Yedjou, C.; Velma, V.R.; Tchounwou, P.B. 2009. Oxidative stress, DNA damage, and antioxidant enzyme activity induced by hexavalent chromium in Sprague-Dawley rats. Environ Toxicol., 24(1): 66-73.
5. Donaldson, R. M.; Barreras, R. F. 1966. Intestinal absorption of trace quantities of chromium. J Lab Clin Med., *68*(3): 484-493.
6. Anderson, R. A. 1997. Nutritional factors influencing the glucose/insulin system: chromium. J Am Coll Nutr., *16*(5): 404-410.
7. Valko, M.; Morris, H.; Cronin, M. T. D. 2005. Metals, toxicity and oxidative stress. Curr Med Chem., 12(10): 1161-1208.
8. Nayak, R. S.; Khanna, B.; Pasha, A.; Vinay, K.; Narayan, A.; Chaitra, K. 2015. Evaluation of nickel and chromium ion release during fixed orthodontic treatment using inductively coupled plasma-mass spectrometer: An in vivo study. J Int Oral Health., *7*(8): 14-20.
9. Clarkson, T.W.; Friberg, L.; Nordberg, G.G.; Sager, P.R. 1988. Biological monitoring of toxic metals. Springer. Doi: 10.1007/978-1-4613-0961-1
10. Langård, S.; Vigander, T. 1983. Occurrence of lung cancer in workers producing chromium pigments. Br J Ind Med., 40(1): 71-74.
11. Zhitkovich, A.; Song, Y.; Quievryn, G.; Voitkun, V. 2001. Non-oxidative mechanisms are responsible for the induction of mutagenesis by reduction of Cr (VI) with cysteine: role of ternary DNA adducts in Cr (III)-dependent mutagenesis. Biochem., 40(2): 549-560.
12. Mathur, A. K.; Chandra, S. V.; Tandon, S. K. 1977. Comparative toxicity of trivalent and hexavalent chromium to rabbits II. Morphological changes in some organs. Toxicol., 8(1): 53-61.
13. Dayan, A. D.; Paine, A. J. 2001. Mechanisms of chromium toxicity, carcinogenicity and allergenicity: review of the literature from 1985 to 2000. Hum Expl Toxicol., 20(9): 439-451.
14. IARC. 1990. International Agency for Research on Cancer. Monographs on the evaluation of the carcinogenic risk to humans, IARC, Chromium, Nickel and welding, Vol-49, 677, ISBN; 92 832 1249 5.
15. Zhang, X. H., et al. 2011. Chronic occupational exposure to hexavalent chromium causes DNA damage in electroplating workers. BMC Public Health, 11(1): 224.
16. Avila-Rojas, S. H., et al. 2020. Alterations in mitochondrial homeostasis in a potassium dichromate model of acute kidney injury and their mitigation by curcumin. Food Chem Toxicol., 145: 111774.
17. Berndt, W. O. 1976. Renal chromium accumulation and its relationship to chromium-induced nephrotoxicity. J Toxicol Environ Health, 1(3): 449-459.
18. De Smet PAGM, Keller K, Hansel R, Frank Chandler R. 1997. Adverse Effects of Herbal Drugs. Germany: Springer-Verleg, 1-13. DOI: https://doi.org/10.1007/978-3-642-60367-9
19. Trivedi, S.P.; Prasad, R.; Khan, A.A. 2020. Amelioration potential of Withania somnifera root extract on hexavalent chromium induced micronucleus in Channa punctatus (Bloch, 1793). J Environ Biol., 41: 677–684.
20. Mansour, H.H; Hafez, H.F. 2012. Protective effect of Withania somnifera against chromium-induced hepatotoxicity in experimental rats. Ecotoxicol Environ Saf., 80: 14-19.
21. Gupta, S.C.; Prasad, S.; Tyagi, A.K.; Kunnumakkara, A.B.; Aggarwal, B.B. 2016. Neem (*Azadirachta indica*): An indian traditional panacea with modern molecular basis. Phytomed., 34: 14-20.
22. Sarah, R.; Tabassum, B.; Idrees, N.; Hussain, M.K. 2019. Bioactive compounds isolated from neem tree and their applications. In book: Natural Bio-active Compounds Sub Volume 1 Production and application, Publisher: Springer. DOI:10.1007/978-981-13-7154-7_17
23. Kitbumrungsap, P.; Suntornwat, O.; Rayanil, K. (2011). Mangiferin and antioxidant capacity from *Mangifera indica* leaves extracts. *Health & Environ Res.*, *44*(5): 62–66.
24. Biswas, S.; Talukder, G.; Sharma, A. 1999. Prevention of cytotoxic effects of chromium by *Allium sativum* L. extract. Cytobios., 99(394): 13-21.
25. Dey, S.; Dey, S.K. 2021. Ameliorative role of garlic (*Allium sativum*) on chromium (VI)-induced membrane damage in male albino rats. Asian Journal of Pharmaceutical and Clinical Research, 14(9): 69-72.
26. Koul, A.; Mallick, T.; Chugh, N. A. 2024. *Aloe vera (L.) Burm.f. renders protection against chromium-induced damage in mice: A preliminary study on renal and testicular tissues.* Ind J Nat Prod Resources, 15(3): 449-461.
27. Pandey, A. K.; Gautam, A.; Pandey, P.; Dubey, R. S. 2018. "Alleviation of chromium toxicity in rice seedling using *Phyllanthus emblica* aqueous extract in relation to metal uptake and modulation of antioxidative defense. South African J Bot., 121: 306-316.
28. Mosaad, A.; Wahhab, A.; Ahmed, H.H. 2004. Protective Effect of Korean Panax ginseng against Chromium VI Toxicity and Free Radicals Generation in Rats. J Ginseng Res., 28(1): 11-17.
29. Jiang, Y. Lv, H.; Li, S.; Han, B.; Liu, Y.; Yang, D.; Li, J.; Yang, Q.; Wu, P.; Zhang Z. 2020. Sulforaphane prevents chromium-induced lung injury in rats via activation of the Akt/GSK-3β/Fyn pathway. *Chemico-Biol Interac*t, 317: 108960.

30. Dolai, D.P.; Roy, S.; Dey, S.K. 2020. Study the protective efficacy of mixed solvent extract of Andrographis paniculata Neesin different proportions against chromium (VI)-induced toxicity. European J Pharma Med Res., 7(4): 457-464.
31. Babu, K.; Maheswari, K.C.U. 2006. In vivo studies on the effect of Ocimum sanctum L. leaf extract in modifying the genotoxicity induced by chromium and mercury in Allium root meristems. J Environ Biol,. 27(1): 93-95.