RESEARCH PAPER

Heavy Metal Exposure in Humans- a review

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ABSTRACT
Human beings are exposed to heavy metals from various sources every day. The exposure to lead, mercury, arsenic, chromium, cadmium and nickel may take place through ingestion, inhalation and dermal contact. Most of these heavy metals are harmful to the body. The body comes in contact with these chemicals through household, industry, agriculture, technology and cosmetic applications. The heavy metals injure the body by various mechanisms such as enhancing oxidative stress and inflammation, interfering with the actions of proteins, enzymes and DNA, and disrupting the signalling pathways. Depending on the type, dose and duration of exposure, the clinical manifestations can be acute or chronic. The features can be mild, moderate and severe. It can involve multiple organs. An individual may be exposure to many metals at the same time, producing a complex picture of multi-organ involvement. Various diagnostic tools are available to detect the levels of heavy metals. Management involves identifying the source of poisoning, use conservative measures, improve nutritional status and introduce chelators when the level of damage is high. This review was aimed to study the sources of heavy metals, mechanisms and clinical features of toxicity, the diagnostic and management protocols to handle heavy metal toxicity.
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INTRODUCTION

Sodium, potassium, magnesium and calcium are essential metals which are required for certain functions of the body. However, certain metals are toxic in spite of being present in trace amounts. Certain essential metals such as zinc, copper, iron, and manganese can be toxic to the body when the levels exceed a certain level (1).

Chronic exposure to heavy metals such as mercury, lead, cadmium, arsenic, and nickel may produce cumulative toxic effects to human health. Heavy metals are hazardous because of certain properties. They act as systemic toxicants, and persist in the body for prolonged periods. They tend to displace essential metals from the body proteins and enzymes, thus disrupting multiple enzymatic and metabolic pathways. They can induce both acute and chronic toxicity even at very low concentrations. Human exposure has increased substantially in recent times due to widespread use in industrial, agricultural, and cosmetic applications. The toxic nature varies according to the chemical form, dose, route, and duration of exposure. Toxic metals interact with DNA and nuclear proteins, leading to oxidative stress, and site-specific cellular and genetic damages (1).

Heavy Metals:

Heavy metals are metals and metalloids, characterized by high density (>5 g/cm³) and have high atomic numbers (>20) (2). They are widespread in the Earth's crust but redistributed extensively by human activity (3). They are distributed among transition metals, basic metals, lanthanides, and actinides (4). Heavy metals include lead, mercury, cadmium, arsenic, and gold (5).

In environmental chemistry, the term "heavy metal" is used to identify metallic elements that are poisonous or toxic even at very low concentrations (3). Iron and Zinc are essential trace elements for human biology in small doses but toxic in large amounts. Lead and Mercury serve no biological function and are hazardous (3). Heavy metal ions can act as either cofactors or inhibitors in enzymatic metabolic pathways (1).

The toxicity due to the exposure to heavy metals toxicity has been widely studied due to the wide spread presence of these in the environment and also because of their tendency to accumulate in human tissues (6). While a subset of metals is indispensable in trace amounts for normal physiology, many others have no beneficial role and can cause profound harm even at very low

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concentrations (7). Long-term exposure to heavy metals tends to disrupt cellular homeostasis; thus, causing disruption of structure and function of the nervous, renal, cardiovascular, endocrine, hematologic, and reproductive systems, and few are categorised as carcinogens (8).

Categories of heavy metals:

Essential trace metals (micronutrients): includes iron, copper, zinc, manganese, and cobalt. Among the trace elements, several are essential for human health because they function as enzymatic cofactors, maintain the structural integrity of biomolecules, and participate in numerous biochemical and physiological processes. Deficiency of these essential trace elements can disrupt normal cellular functions and result in a wide range of pathological conditions. They play crucial roles in the regulation of cardiovascular health, glucose metabolism, blood coagulation, inflammatory responses, DNA synthesis, cholesterol metabolism, neuromuscular function, and cancer prevention. However, excessive accumulation of trace elements can also be deleterious, leading to the development of various diseases. Therefore, maintaining optimal concentrations of essential trace elements through a balanced and nutritious diet is critical (9).

Non-essential/toxic metals: include lead, mercury, cadmium, arsenic, and chromium. They do not have any physiological requirement; on the other hand, can be dangerous even at minimal exposures (2). These metals are introduced into the environment through both natural (geogenic) and anthropogenic sources. Heavy metal pollution is severe in point-source areas such as mining operations, foundries, smelters, and other metal-based industries, where environmental concentrations are significantly elevated, posing substantial risks to ecosystems and human health (2).

Heavy metal toxicity:

Bioaccumulation and chronic burden heavy metal poisoning occurs when intake and tissue retention are much higher than the excretion levels (7). The heavy metals enter the food chain through contaminated air, water, and soil, leading to build up these metals in high concentrations in living organisms. Once absorbed, heavy metals accumulate in bones, liver, kidneys, and the brain, where they exhibit prolonged biological half-lives. The severity of toxicity depends on the type of metal, its concentration, and the duration of exposure. Although acute poisoning can produce immediate clinical manifestations, while chronic low-level exposure causes cumulative cellular damage which may gradually impair physiological functions and increase the risk of chronic diseases (10).

Sources of heavy metals:

Heavy metals are significant environmental pollutants due to their persistence, non-biodegradability, and toxic effects on living organisms. The harmful effects of the heavy metals are recognized for centuries. Mercury toxicity is associated with the consumption of contaminated food. Mercury-containing cinnabar and arsenic-containing realgar are being used in medicinal preparations (11). Rapid industrialization, urbanization, and agricultural expansion have markedly increased environmental contamination with heavy metals. These contaminants persist in the environment, enter the food chain, and accumulate in living organisms through ingestion, inhalation, and dermal exposure, posing significant risks to human health (11).

Table 1: Sources of heavy metals

Type of exposure	Processes
Natural (geogenic)	**Weathering of bedrock and soils**, volcanic emissions, forest fires, tectonic processes. Ex: contamination of groundwater with arsenic from mineral deposits (3,12)
Anthropogenic (human-derived)	**Industrial processes**: mining, ore smelting/refining; leather tanning (chromium), battery manufacturing and recycling (lead, cadmium), chlor-alkali production (mercury), electroplating, pigment and dye manufacture (2,3)
	Agriculture: metal-containing pesticides, herbicides, and phosphate fertilizers introducing arsenic and cadmium into soils and crops (3,13)
	Waste streams: discharge of industrial effluents, e-waste processing, informal recycling, and landfill leachate contaminating soil, air, and water; incineration emissions (3,14)
	Consumer and household products: lead in aging paints, ceramics with lead glazes, few toys and vintage items; mercury in older thermometers, fluorescent lamps, few skin-lightening creams and dental amalgams; aluminium in cookware, antacids, and some deodorants; chromium in leather goods (15)
	Tobacco smoke: recognized source of cadmium and chromium inhalation
	Occupational: welding, battery plants, artisanal gold mining (mercury vapor), tanning, electroplating, electronics recycling (16)
	Diet: large predatory fish (methylmercury), rice irrigated with contaminated

	water (arsenic, cadmium), leafy vegetables grown in polluted soils (17)
	Hemodialysis: Patients undergoing hemodialysis are susceptible to the accumulation and toxicity of aluminium, cadmium, and arsenic (18). Aluminium toxicity is due to elevated aluminium levels in the water supply, and defects in the dialysate distribution system within dialysis units (19). Blood lead levels tend to increase with the duration of hemodialysis. Even low-level lead exposure may contribute to the development of interstitial nephritis, hypertension, hyperuricemia, and an increased risk of cerebrovascular and cardiovascular diseases, thereby accelerating the deterioration of pre-existing renal dysfunction (20).

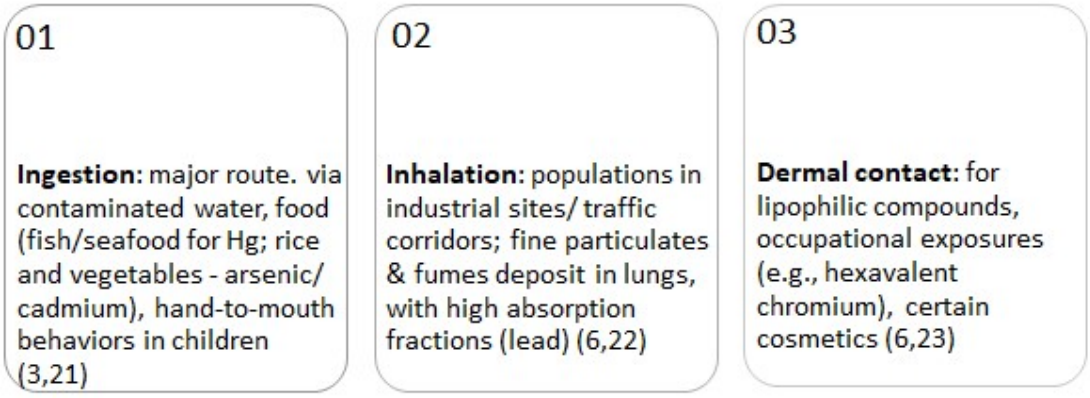

Figure 1: Human Exposure Pathways

Mechanisms of heavy metal toxicity:
Mechanisms of heavy metal toxicity can be in the forms of oxidative stress, inflammation, DNA damage, endocrine disruption, neuro-, nephron-, and hepatotoxicities, cardiovascular disorders, reproductive dysfunction, and carcinogenesis. The severity of toxicity depends on the type of metal, dose, duration of exposure, and individual susceptibility. Understanding the biochemical mechanisms is essential for developing effective preventive, diagnostic, and therapeutic strategies (11).

Oxidative stress: Chromium, cadmium, and lead impair antioxidant defences by binding to the sulfhydryl groups on certain enzymes like superoxide dismutase (SOD), catalase, glutathione peroxidase (GPx) (3,6). Redox-active metals such as iron and copper catalyze Fenton-type reactions, producing hydroxyl radicals that oxidize lipids, proteins, and nucleic acids. Metals form complexes with glutathione (GSH), a tripeptide involved in redox reactions. This lowers cellular antioxidant capacity, predisposing the cells to oxidant injury (24)

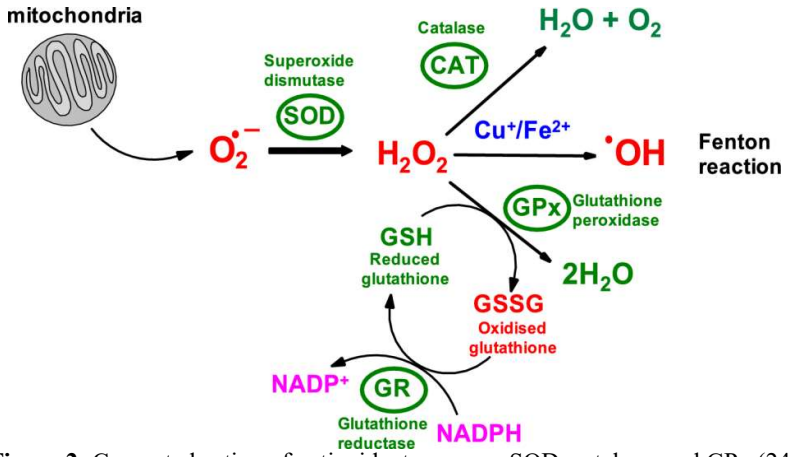

Figure 2: Concerted action of antioxidant enzymes SOD, catalase, and GPx (24)

Interferences in transport and signalling pathways: Lead competes with other divalent ions like calcium and zinc, and disrupts calcium-dependent neurotransmission, synaptic pruning, and second-messenger signalling, and can trigger neuronal apoptosis (3,25). Cadmium can replace zinc in metallothioneins and zinc-finger proteins, and impair protein function and release free cadmium which may injure renal and hepatic tissues (6,26).

Arsenate and aluminium replace phosphate, thus disrupting ATP-dependent processes (3,27).

Genotoxicity: Direct DNA damage (strand breaks, crosslinks) and interference with repair pathways. Cadmium impairs p53 function, diminishing DNA repair and facilitating carcinogenesis (3,28). Chromium enters cells, where it is reduced to reactive intermediates that form DNA adducts and crosslinks and thus interferes with DNA replication (29).

Epigenetic alterations: Changes in DNA methylation and histone modifications alter gene expressions without changing gene sequence. Mercury exposure is associated with methylation changes in renal matrix metalloproteinase genes, leading to cytoskeletal injury in renal cells and renal dysfunction (30). Chromium decreases S-adenosylmethionine leading to hypomethylation of DNA. Cadmium inhibits histone deacetylases leading to increased histone acetylation and altered chromatin structure, which affected the gene transcription. Mercury causes altered methylations in brain leading to tumor formation. Chronic lead exposure resulted in elevated histone acetylation levels in hippocampal tissues (11).

Immunotoxicity and endocrine disruption: Dysregulated cytokine signaling, altered immune surveillance, and potential interference with hormone receptors and synthesis. Heavy metals are well-recognized endocrine-disrupting agents, with lead, cadmium, arsenic, mercury, copper, nickel, zinc, and manganese being among the most extensively studied. These metals can disrupt endocrine function by interacting with hormone receptors and modifying hormonal signaling pathways, thereby producing adverse health effects through either synergistic or antagonistic actions on endogenous hormones (3). Many heavy metals act as hormone mimics by serving as ligands that bind to specific hormone receptors. The receptor–metal complexes are activated to interact with hormone response elements in target genes of hormones, thus causing altered gene expression and undesirable downstream effects. The heavy metals can interfere with the metabolism of endogenous hormones, resulting in impaired hormone homeostasis. All these lead to deranged metabolic, reproductive, and developmental functions (31).

Clinical Features by Heavy Metal Poisoning:

Lead: acute clinical features include abdominal colic, headache, fatigue, irritability, and acute hemolytic anemia (23,32). Chronic exposure to lead leads to neurologic features such as mental retardation, attention and behavioral problems, and learning disabilities in children (3). Peripheral neuropathy and encephalopathy are seen in adults (1). It causes tubulointerstitial nephropathy, hyperuricemia and gout, which can progression to CKD (20). Hematological features include impaired heme synthesis, and anemia with basophilic stippling. Skeletal manifestations are due to long-term storage in bone (t½: 20–30 years). Cardiovascular complications are due to hypertension and vascular dysfunction (20). Neuroinflammation involves microglia and astrocytes with the production of inflammatory cytokines, increased reactive oxygen species (ROS) generation, diminished antioxidant capacity, neuronal loss, and injury to the central nervous system (3).

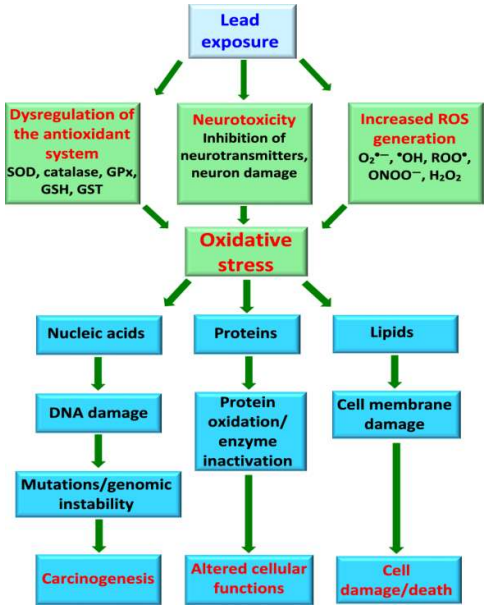

Figure 3: Mechanisms of lead toxicity (3)

Mercury: can enter the body by inhalation or ingestion. Acute manifestations include necrotizing bronchitis/pneumonitis, severe gastrointestinal injury with corrosive ulcers (33). Chronic features include Minamata disease with ataxia, paraesthesia /numbness, constricted visual fields, dysarthria, and hearing loss; fetal exposure causes neurodevelopmental deficits (Methylmercury) (34); acrodynia (pink disease) in children causing rash,

irritability, sweating, tachycardia, and tremor (35). Renal manifestations include proximal tubular injury leading to proteinuria and membranous nephropathy (1). Neuroinflammation leads to the development, and progression of multiple sclerosis, Alzheimer's disease, Parkinson's disease, Huntington's disease, and other disorders (3). Mercury binds to thiol groups, inactivates enzymes, and promotes reactive oxygen species (ROS) production. Mercury chloride inhibits tyrosinase activity (23).

Cadmium: inhalation causes fever, chills, myalgias, chemical pneumonitis, while severe exposure causes pulmonary edema. Chronic renal involvement leads to proximal tubular dysfunction which may progress to CKD, hypercalciuria and nephrolithiasis (3). Itai-itai disease-osteomalacia seen in Japan, bone pain, and fractures (36). It can precipitate hypertension thus increasing cardiovascular risk (1). Cadmium is a highly toxic metal, and classified as a carcinogen. It binds to metallothioneins and other metal transporters and decreases the absorption of zinc, calcium and copper. It causes hepatotoxicity, nephrotoxicity, and painful degenerative bone disease (23).

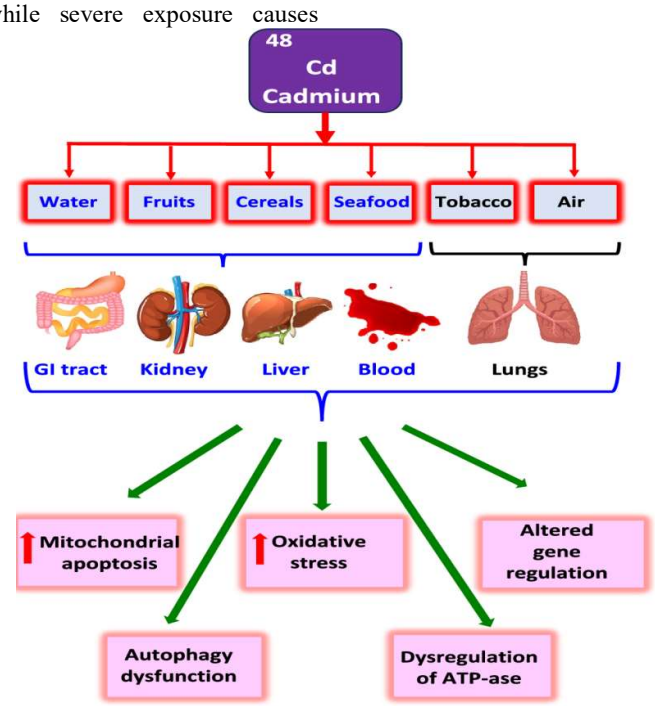

Figure 4: Sources and toxic effects of cadmium (3)

Arsenic: acute poisoning causes garlic odour in breath, vomiting, watery diarrhea- rice water stools, hypotension, shock, QT prolongation by electrocardiogram (ECG), arrhythmias and neuropathy (37). Chronic exposure leads to hyperpigmentation- "raindrop", palmar/plantar keratoses, Mees' lines on nails, and Blackfoot disease - severe peripheral vascular disease with gangrene (38-40). Also increases the risk of cancer of skin, lung, bladder, and liver and sensorimotor neuropathy (1). Arsenic induces epigenetic alterations and DNA damage, thereby increasing carcinogenicity (23).

Chromium: especially hexavalent form causes allergic contact dermatitis and "chrome holes or sores"- deep, and penetrating ulcers (41). Respiratory signs include nasal septum perforation, asthma, and increased risk of lung cancer with inhalational exposure (3). Chromium is required in trace amounts for lipid and protein metabolism. The toxicity and carcinogenicity are due to genomic instability, DNA damage, and oxidative stress (23).

Aluminium: It accumulates in the body following repeated exposure and it is released from the body very slowly. It causes osteomalacia and Alzheimer's disease and other neurodegenerative disorders. It also affects breast tissue morphology (23). Toxic effects are related to its pro-oxidant effects, which in turn cause oxidative stress-induced damage to proteins and membrane lipids (3). It increases the levels of macrophage inflammatory protein-1α (MIP-1α), interleukin-1 (IL-1) and tumor necrosis factor (TNF-α). In renal failure or high parenteral/medication exposure causes encephalopathy ("dialysis dementia"), speech abnormalities, proximal myopathy, osteomalacia and microcytic anemia (3,19).

Nickel: it binds to keratin, and accumulates in the stratum corneum (23). Skin dermatitis caused by nickel present in jewellery and cosmetics (1). It is also classified as a carcinogenic and causes neurotoxicity (23).

Diagnostics and Monitoring of Heavy Metal Exposure

Exposure assessment (body burden): The diagnosis of heavy metal toxicity begins with a detailed history of environmental, occupational, dietary, or accidental exposure, followed by laboratory tests selected according

to the suspected metal and the clinical objective, such as detecting recent exposure, estimating body burden, or assessing organ damage (6).

Laboratory Evaluation (6)
No single biological specimen is ideal for all heavy metals. The biological sample to be used is based on the suspected metal poisoning (6).

- Whole blood: recent exposure to lead and mercury
- Urine: for cadmium, chromium and inorganic arsenic metabolites
- Hair and nail analysis: can estimate long-term exposure to methylmercury and arsenic; interpret cautiously due to external contamination risk.
- X-rays: metaphyseal "lead lines" in pediatric long bones.
- Bone lead measurement (K-XRF): research/advanced clinical settings for cumulative lead.

Organ injury biomarkers:
- Free erythrocyte protoporphyrin or zinc protoporphyrin in lead exposure
- Urine beta-2 microglobulin and N-acetyl-β-D-glucosaminidase- early tubular markers in cadmium poisoning.
- Kidney function: serum creatinine, BUN, and urine albumin/creatinine ratio.
- Oxidative stress panels

Specialized tests:
- Chelation mobilization test for lead body burden.
- Speciation testing: differentiates organic vs inorganic mercury/ arsenic metabolites (6)

Monitoring and Follow-up: based on the biological half-life of each metal (6)

- Baseline investigations- complete blood count, renal and liver function tests, electrolytes, and urinalysis.
- Cadmium exposure: monitored over months to years, urinary biomarkers- β2-microglobulin or retinol binding protein
- Mercury and lead: monitored over weeks to months
- Arsenic: monitored over days to weeks.

Management of Heavy Metal Poisoning
According to the WHO/Europe, the tolerable weekly intake (TWI) for mercury, lead, cadmium, and arsenic is 0.0016, 0.025, 0.007, and 0.015 mg/kg body weight, respectively. In spite of taking critical efforts, many regions still report concentrations exceeding the recommended WHO limits in agricultural soils and wastewater. As a result, there is growing interest in sustainable approaches to mitigate heavy metal pollution (1).

Generally, removing the source of exposure and providing appropriate supportive care are important and safer than pharmacological treatment alone. The main principles of management include confirming the type and extent of exposure, preventing further exposure, correcting contributing factors such as nutritional deficiencies, underlying medical conditions, and promptly treating acute complications (6). An individual may by exposed to more than one metal and multiple organs may be involved. Hence multidisciplinary approach is required at the individual, household, workplace, and public health levels to reduce exposure and minimize long-term health effects (6).

Supportive Care and Prevention
The crucial step in managing heavy metal exposure is controlling at the source. At home, measures include repairing deteriorating lead-based paint, removing contaminated dust with High-Efficiency Particulate Air (HEPA) vacuum cleaners, replacing lead-containing plumbing, using certified water filters, and avoiding contaminated ceramics, spices, cosmetics, and traditional medicines. Smoking cessation is strongly recommended because tobacco is a major source of cadmium (6). In occupational settings, engineering controls, such as local exhaust ventilation and enclosed processes, combined with administrative measures, personal protective equipment, and workplace hygiene practices to prevent both occupational and take-home exposure (6).

Acute Management (6)
Patients with acute heavy metal poisoning require immediate stabilization of airway, breathing, and circulation, followed by decontamination. Early consultation with a poison control centre or medical toxicologist is mandatory.

- **Mercury:** removal from the contaminated area and supportive respiratory care.
- **Arsenic:** acute poisoning requires aggressive fluid and electrolyte replacement, cardiac monitoring, and early consideration for chelation therapy.
- **Lead:** encephalopathy is a medical emergency requiring seizure control, management of raised intracranial pressure, and prompt chelation therapy.
- **Mixed-metals:** in those involved in battery recycling or electronic waste handling
- **Tobacco:** cadmium and chromium intake.

Nutritional Support
Good nutrition helps reduce heavy metal absorption and toxicity. Correcting iron deficiency decreases lead absorption, while adequate calcium and zinc intake provides additional protection. Selenium reduces mercury toxicity through antioxidant effects. Antioxidants such as alpha-lipoic acid (ALA) and GSH may support cellular antioxidant defences. Choosing low-mercury fish and using safer rice preparation methods, such as washing and

cooking rice in excess water, can reduce mercury and arsenic exposure (6).

Special Populations

During pregnancy, the primary goal is prevention and exposure reduction, with chelation reserved for severe maternal poisoning under specialist supervision. Children are vulnerable to heavy metal toxicity and require careful dosing, close monitoring, and home environmental assessment. Patients with CKD may need dose adjustments. Workers with occupational exposure are required to undergo regular biological monitoring, workplace risk assessment, and appropriate protective measures (6).

Detoxification of Heavy Metals by Chelation

Chelation therapy is one of the primary treatments for heavy metal poisoning. It involves the administration of chelating agents that bind toxic metals in the body and promote their elimination through urine or bile. Prompt medical intervention is essential in cases of acute heavy metal poisoning (3). Chelating agents are molecules that form stable complexes with metal ions such as lead, cadmium, and mercury. Chelators bind metal ions through electron-donating atoms such as nitrogen, oxygen, and sulfur. The stability of these metal–chelator complexes depend on factors such as the type of metal ion, its chemical properties, and the nature of the chemical bond formed (6).

Chelating agents mobilize the heavy metals from tissues into the bloodstream and facilitate their excretion by the kidneys in urine or by the liver through bile (6). Commonly used chelating agents include ethylenediaminetetraacetic acid (EDTA), dimercaprol (British Anti-Lewisite, BAL), 2,3-dimercaptosuccinic acid (DMSA), 2,3-dimercapto-1-propanesulfonic acid (DMPS), and D-penicillamine. The choice of chelator depends on the specific metal involved, the severity of poisoning, and the patient's clinical condition (6).

Table 2: Common Chelating Agents Used in Heavy Metal Poisoning (3,37)

Chelators	Mechanism
EDTA	Approved by the U.S. Food and Drug Administration (FDA) for the treatment of lead poisoning in both adults and children. It can also bind cadmium, mercury, zinc, and iron. Once bound, the metal–EDTA complexes are excreted through the urine. As EDTA does not readily enter cells, it removes metals from the bloodstream and extracellular fluid. Side effects: kidney toxicity, which may lead to proteinuria and, in severe cases, renal failure.
BAL	Used mainly for acute poisoning by arsenic, mercury, lead, and gold. Side effects: fever, nausea, vomiting, and hypertension. Approved for treating Wilson's disease.
DMSA	Enhances the urinary excretion of lead, cadmium, arsenic, and methyl including the brain.
DMPS	Effective against cadmium, arsenic, and mercury. It is converted into disulfide forms and eliminated through urine and bile.
D-Penicillamine	Used to remove excess copper in Wilson's disease. It can also chelate lead, cadmium, arsenic, and mercury. However, it is generally less effective than DMSA and DMPS, in removing methylmercury and eliminating metals from brain tissue.

Overall, the choice of chelating agent depends on the type of metal involved, the severity of poisoning, the route of exposure, and the patient's clinical condition (3).

Emerging and Future Therapies

Current research on heavy metal toxicity aims to overcome the limitations of existing chelation therapy, particularly its lack of specificity, unwanted removal of essential minerals, and inability to account for individual differences in genetics, metal metabolism, and the gut microbiome. An integrated approach of biochemistry, soil science, pharmacology, and personalized medicine may improve the strategy of handling of heavy metal poisoning (6).

Next-Generation Chelators

New chelating agents can selectively bind toxic metals, while decreasing the uncontrolled wastage of metals like zinc and copper. Improved techniques such as nanoparticles, polymer-based carriers, liposomes, and dendrimers are being tried to deliver chelators to target organs such as liver or kidneys, thereby improving efficacy of chelators. Prodrugs are being developed which get converted to active forms at the sites of tissue injury (6).

Biological Approaches

These techniques use the body's natural metal-handling mechanisms. Newly discovered proteins with high affinity for mercury, lead, and cadmium are being investigated to capture toxic metals. Other studies have researched on enzymes, which may convert toxic metal species into less harmful forms. In addition, modulation of the gut microbiome may decrease intestinal metabolism of heavy metals, thereby promoting their excretion (methylmercury) (6).

Precision Toxicology

Advances in various omics pave the way for precision toxicology. The metabolism of heavy metals is influenced by genetic variations of the individual. Hence, the type and extent of toxicity also vary across the individuals. Identifying genetic profile of an individual may help in identifying individuals who are at high-risk, and decide predict chelation and other therapies (6).

Device-Based Therapies

Techniques such as hemodialysis and hemoperfusion, may help in eliminating metals like mercury and arsenic especially in patients with renal failure. Metal-specific adsorption cartridges may effectively remove toxic metals and metal-chelator complexes (6).

Future Perspectives

Despite advances in managements, several challenges still exist. Future treatments should improve patient outcomes, ensure long-term safety, cost-effective, and accessible to all populations (6).

Analytical Methods used in detection of Heavy Metals

Analytical techniques such as atomic absorption spectrometry (AAS), inductively coupled plasma–optical emission spectroscopy (ICP-OES), and inductively coupled plasma–mass spectrometry (ICP-MS). They are used to estimate heavy metals in biological, environmental, food, and industrial samples (23). For mercury analysis, Cold Vapor Atomic Absorption Spectrometry (CV-AAS) is the preferred method. Among modern analytical techniques, ICP-OES offers rapid multi-element analysis with good accuracy, while ICP-MS provides the highest sensitivity (23).

Emerging Analytical Techniques for Heavy Metal Detection

X-ray fluorescence (XRF) is a rapid, non-destructive technique used to identify and measure heavy metals in cosmetic products. Portable XRF instruments have been successfully used to measure zinc and titanium in sunscreens, producing results that closely agree with those obtained by ICP-MS (23)

Fluorescence resonance energy transfer (FRET) works by transferring energy between two fluorescent molecules. FRET-based assays are highly sensitive and can simultaneously detect multiple heavy metals in complex samples. They are developed using small organic molecules, nanomaterials, or fluorescent proteins (23). FRET-based chemosensors have been widely used for rapid and selective detection of heavy metals. For example, a rhodamine-based chemosensor has been used to detect lead in lipstick. Similar optical chemosensors have also been developed for detecting cadmium, cobalt, and mercury in cosmetic products. These sensors offer excellent sensitivity, selectivity, and relatively simple analysis (23).

To provide inexpensive and user-friendly alternatives, paper-based microfluidic devices, particularly distance-based paper analytical devices (DμPADs), have been developed. They are used to assess the levels of heavy metals in native medicines and cosmetics (23). They represent a promising, low-cost, portable, and environmentally friendly approach for detecting heavy metals in cosmetic products, making them suitable for rapid on-site analysis (23).

Bioremediation: an eco-friendly approach that uses living organisms to remove or detoxify environmental pollutants. In in situ bioremediation, naturally occurring microorganisms treat contaminated soil or water at the polluted site. In ex situ bioremediation, contaminated soil is excavated and treated in a controlled environment above ground (1).

Phytoremediation: uses plants and their metal-binding proteins, such as phytochelatins and metallothioneins, to absorb, accumulate, or detoxify heavy metals. Since heavy metals inhibit plant growth and productivity, beneficial endophytic microorganisms, including species of *Pantoea*, *Pseudomonas*, and *Flavobacterium*, can enhance plant tolerance by reducing metal toxicity. In addition, cyanobacteria have shown considerable potential for removing heavy metals from contaminated soils and wastewater (1). These biological approaches represent effective and environmentally sustainable methods for remediating heavy metal-contaminated soil, water, and sediments (1).

CONCLUSION

Heavy metal poisoning occurs due to various causes involving environment, industries, and heterogeneous biological systems. The mechanisms behind the toxicity includes oxidative stress, ionic similarity, genetic and epigenetics. The toxicity features can be acute or chronic involving multiple organs, which can result in failure, degenerative disorders or malignancy. Efforts must be taken to diagnose heavy metal toxicity based on history and identify the source of toxicity. Management should focus on laboratory diagnosis involving various biomarkers. Treatment involves removal of exposure, use of appropriate chelators in addition to creating public awareness among the public.

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