



Review Article

## Environmental Heavy Metal Pollution: Chemical Forms, Sources, Toxicological Effects, and Remediation Approaches

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### Abstract

Heavy metal pollution constitutes one of the most pressing environmental and public health challenges of the twenty-first century. Unlike organic pollutants, heavy metals are non-biodegradable, and their persistence in soil, water, and atmospheric compartments poses irreversible ecological risks. This review provides a comprehensive examination of the chemical speciation of key heavy metals, lead (Pb), cadmium (Cd), mercury (Hg), arsenic (As), chromium (Cr), and nickel (Ni). It elucidates how their physicochemical forms govern bioavailability, toxicokinetics, and environmental fate. Anthropogenic sources, including mining, industrial effluents, agricultural inputs, and combustion processes, are critically appraised. Toxicological mechanisms involving reactive oxygen species (ROS) generation, enzymatic inhibition, DNA damage, and organ-specific pathologies are discussed with reference to current molecular evidence. Physical, chemical, and biological remediation strategies are comparatively evaluated, with emphasis on phytoremediation, microbial bioremediation, and emerging nanomaterial-assisted approaches. The review underscores the need for integrated, multidisciplinary strategies that combine policy frameworks with green technologies to mitigate heavy metal pollution globally.

**Keywords:** heavy metals; chemical speciation; environmental pollution; oxidative stress; phytoremediation; bioremediation; toxicity; soil contamination

### 1. Introduction

Heavy metal pollution has escalated dramatically over the past century, driven by unprecedented industrialisation, urbanisation, and agricultural intensification. Heavy metals are conventionally defined as metallic elements with atomic densities exceeding 5 g/cm<sup>3</sup>, a classification encompassing lead (Pb), cadmium (Cd), mercury (Hg), arsenic (As), chromium (Cr), and nickel (Ni), among others [1]. Although

some trace metals such as copper (Cu) and zinc (Zn) are biologically essential at low concentrations, their excess, along with non-essential metals like Pb, Cd, and Hg, poses severe toxicological risks to human health and ecosystem integrity [2].

Unlike organic pollutants, heavy metals are chemically inert and non-biodegradable; they resist decomposition and accumulate persistently in environmental matrices, including

soil, sediment, water, and biota [3]. Their persistence is compounded by bioaccumulation and biomagnification through food chains, resulting in progressively higher concentrations in organisms at higher trophic levels. The World Health Organisation (WHO) has identified lead, cadmium, mercury, and arsenic among the top ten chemicals of major public health concern [4].

The environmental distribution and toxicity of heavy metals are not merely functions of total concentration but are critically governed by chemical speciation, the particular forms in which a metal exists in a given environment. A metal's oxidation state, ligand associations, and matrix interactions determine its solubility, mobility, bioavailability, and ultimately its toxicological potential [5]. For example, hexavalent chromium [Cr (VI)] is far more toxic and mobile than its trivalent counterpart [Cr (III)], while methylmercury exhibits substantially greater neurotoxicity than inorganic mercury forms.

The scientific literature on heavy metal pollution has grown substantially in the 2020–2025 period, with increasing focus

on integrated remediation strategies, nano-enabled clean-up technologies, and the role of climate change in altering metal speciation and mobilisation [6]. This review synthesises current knowledge on the chemical forms, environmental sources, toxicological mechanisms, and remediation approaches associated with major heavy metals, providing an evidence-based resource for researchers, clinicians, environmental scientists, and policymakers.

## 2. Chemical Forms and Speciation of Heavy Metals

Chemical speciation refers to the distribution of an element among distinct chemical forms and phases in a given medium. In environmental science, speciation determines the reactivity, mobility, and bioavailability of heavy metals and is therefore central to risk assessment and remediation planning [5,7]. Table 1 summarises the principal chemical forms, oxidation states, densities, and environmental fate of the six major heavy metals reviewed here.

**Table 1. Chemical Forms, Oxidation States, and Environmental Fate of Major Heavy Metals [1,2,5,7]**

Heavy Metal	Chemical Forms	Oxidation States	Density (g/cm <sup>3</sup> )	Environmental Fate
Lead (Pb)	PbSO <sub>4</sub> , PbCO <sub>3</sub> , PbS, Pb <sup>2+</sup>	+2, +4	11.34	Adsorbs to soil particles; persists; enters the food chain via plant uptake
Cadmium (Cd)	CdS, CdCO <sub>3</sub> , Cd <sup>2+</sup> , CdCl <sub>2</sub>	+2	8.65	Highly mobile in acidic soils; strongly bioaccumulates in plants
Mercury (Hg)	Hg <sup>0</sup> , Hg <sup>2+</sup> , CH <sub>3</sub> Hg <sup>+</sup> (methylmercury)	0, +1, +2	13.53	Methylation in aquatic sediments; biomagnification in food webs
Arsenic (As)	As(III), As(V), arsenite, arsenate	-3, 0, +3, +5	5.73	Adsorption to Fe/Al oxides; groundwater contamination via reductive dissolution
Chromium (Cr)	Cr(III), Cr(VI), CrO <sub>4</sub> <sup>2-</sup> , Cr <sub>2</sub> O <sub>3</sub>	+2, +3, +6	7.19	Cr(VI) is highly mobile and toxic; Cr(III) precipitates as hydroxide at neutral pH
Nickel (Ni)	NiS, Ni <sup>2+</sup> , Ni(OH) <sub>2</sub> , NiCO <sub>3</sub>	+2, +3	8.91	Moderate soil mobility; affected by organic matter and pH

Lead exists predominantly as Pb<sup>2+</sup> in oxidised environments, often associated with carbonates, sulphates, and organic matter in soils. In reducing conditions, galena (PbS) is the

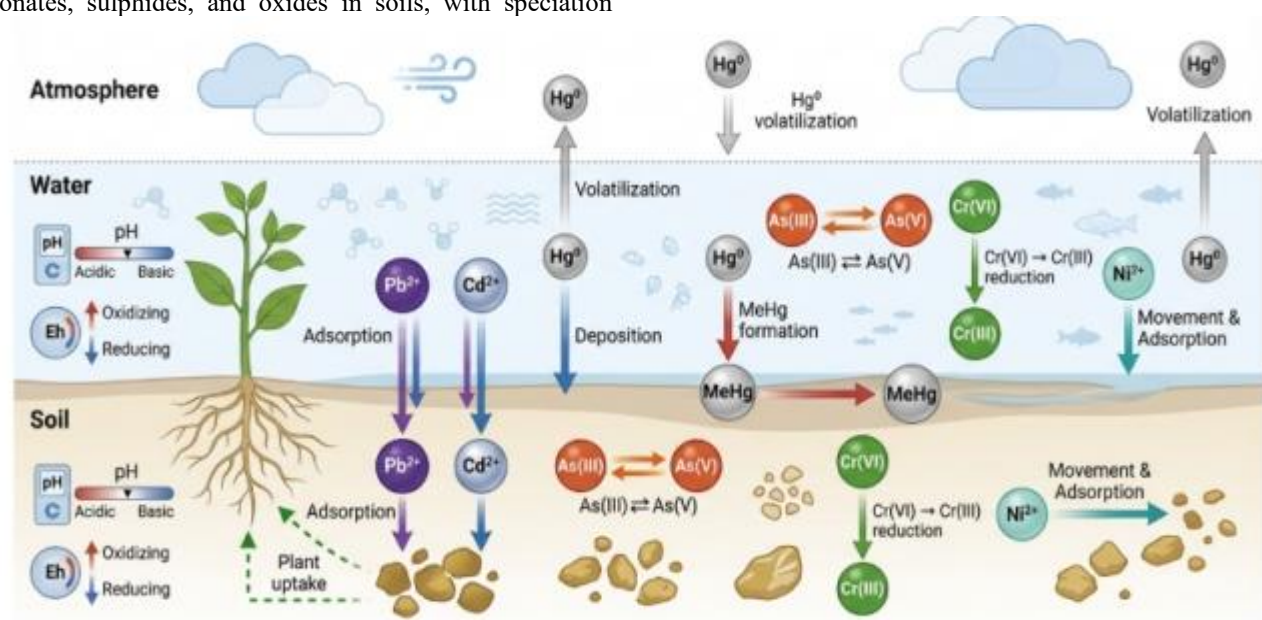
stable form. Its low solubility at neutral pH moderates bioavailability, but acidification markedly increases Pb<sup>2+</sup> release [8]. Cadmium predominantly occurs as Cd<sup>2+</sup>, exhibiting high mobility in acidic soils and a strong affinity for organic matter and clay particles. Its geochemical behaviour closely mimics zinc and calcium, facilitating uptake by plant roots [9].

Mercury presents a uniquely complex speciation profile. Elemental mercury (Hg<sup>0</sup>) volatilizes into the atmosphere, where it undergoes long-range transport and eventual deposition. In anoxic aquatic sediments, sulfate-reducing bacteria catalyse methylation to methylmercury (CH<sub>3</sub>Hg<sup>+</sup>), the most toxic and bioaccumulable form, responsible for Minamata disease and widespread aquatic ecosystem disruption [10]. Arsenic speciation is pH and redox-dependent; arsenite [As(III)] predominates in reducing conditions, while arsenate [As(V)] prevails in oxidised environments. Both forms have distinct toxicological

profiles, with As (III) exhibiting greater acute toxicity through thiol-group binding [11].

Chromium speciation is toxicologically critical: Cr(VI) exists as highly water-soluble oxyanions (chromate, dichromate), is readily absorbed by organisms, and is classified as a Group 1 human carcinogen. Cr(III) forms insoluble hydroxides at neutral pH, exhibiting far lower toxicity and mobility [7]. Nickel primarily exists as  $Ni^{2+}$  in aquatic systems and forms carbonates, sulphides, and oxides in soils, with speciation

profoundly influenced by pH and organic matter content. Figure 1 presents the Schematic representation of the environmental speciation and transformation pathways of lead (Pb), cadmium (Cd), mercury (Hg), arsenic (As), chromium (Cr), and nickel (Ni) across soil, water, and atmospheric compartments, showing redox transitions and bioavailability drivers.



**Figure 1. Schematic representation of the environmental speciation and transformation pathways of lead (Pb), cadmium (Cd), mercury (Hg), arsenic (As), chromium (Cr), and nickel (Ni) across soil, water, and atmospheric compartments, showing redox transitions and bioavailability drivers.**

*Caption: A detailed scientific infographic illustrating the environmental speciation and transformation pathways of six heavy metals (Pb, Cd, Hg, As, Cr, Ni). The diagram shows three horizontal compartment layers: atmosphere (blue sky), water (light blue), and soil (brown). A colour-coded arrow is used to indicate transformations:  $Hg^0$  volatilizing to the atmosphere, then depositing; methylmercury formation in sediment;  $As(III)/As(V)$  redox cycling;  $Cr(VI)$  to  $Cr(III)$  conversion; Pb and Cd adsorption to soil particles with plant uptake arrows.*

**Sources: Adapted from references [5,7]**

### 3. Sources of Environmental Heavy Metal Pollution

#### 3.1 Natural Sources

Geogenic processes constitute baseline sources of heavy metals in the environment. Weathering of metal-bearing parent rocks, volcanic emissions, hydrothermal activity, and natural erosion continuously release heavy metals into soil, groundwater, and the atmosphere [1,5]. For example, areas underlain by ultramafic and mafic rocks, rich in nickel, chromium, and cobalt, inherently exhibit elevated background concentrations of these metals. Geogenic arsenic

release in deltaic and alluvial aquifers, as observed in Bangladesh, West Bengal, and the Indo-Gangetic Plain, represents a significant natural source of groundwater contamination affecting millions of people [11].

#### 3.2 Anthropogenic Sources

Human activities dramatically accelerate the release of heavy metals into the environment, far exceeding natural fluxes. Table 2 presents major anthropogenic and natural sources for each metal reviewed.

**Table 2. Principal Sources of Heavy Metal Pollution and Environmental Concentration Thresholds [1,3,5,9,12]**

Metal	Primary Anthropogenic Sources	Natural Sources	Typical Soil Threshold (mg/kg)
Lead (Pb)	Battery manufacturing, smelting, leaded paint, vehicle emissions, mining	Galena (PbS), crustal weathering	≤300 (WHO guidance)
Cadmium (Cd)	Phosphate fertilizers, electroplating, nickel-Cd batteries, paint pigments	Volcanic emissions, weathering of Cd-rich rocks	≤3 (EU limit)
Mercury (Hg)	Coal combustion, chlor-alkali industry, artisanal gold mining, crematoria	Volcanic outgassing, geothermal vents	≤1 (USEPA screening)
Arsenic (As)	Pesticides, smelting, coal combustion, wood preservatives, semiconductor industry	Arsenopyrite weathering, geogenic groundwater	≤20 (USEPA)
Chromium (Cr)	Leather tanning, steel production, electroplating, dye manufacturing	Chromite ore, ultrabasic rock weathering	≤100 (guideline)
Nickel (Ni)	Stainless steel, alloy manufacturing, oil refining, mining	Ultramafic rocks, dust from soils	≤75 (EU guideline)

Mining and smelting operations represent the most concentrated point sources of heavy metal pollution, releasing Pb, Zn, Cd, As, and Hg through acid mine drainage, tailings, and atmospheric deposition over vast surrounding areas [3]. The combustion of fossil fuels, particularly coal, constitutes a major diffuse source of mercury, arsenic, and selenium, emitting these elements into the atmosphere from where they deposit globally [5]. Agricultural practices contribute substantially through the repeated application of phosphate fertilisers (which contain Cd impurities), sewage sludge rich in multiple metals, and pesticides (historically containing Pb, As, and Hg) [9].

Industrial effluents from leather tanning (Cr), electroplating (Ni, Cr, Cd), battery manufacturing (Pb, Cd), and the chlor-alkali industry (Hg) discharge directly into aquatic ecosystems, causing severe localised contamination [1,3]. Artisanal and small-scale gold mining (ASGM), widely practised across sub-Saharan Africa, South America, and Southeast Asia, employs mercury amalgamation techniques and constitutes the largest anthropogenic source of mercury pollution globally [12]. Electronic waste (e-waste) processing, common in developing nations, releases toxic metals including Pb, Cd, Hg, and Cr through informal recycling activities [6].

## 4. Toxicological Effects of Heavy Metals

### 4.1 General Mechanisms of Toxicity

Heavy metals exert toxicity through shared and metal-specific molecular mechanisms. Common pathways include the generation of reactive oxygen species (ROS), depletion of cellular antioxidants, enzyme inhibition through displacement of essential metal cofactors, and binding to sulfhydryl (-SH) groups of proteins [13]. The resulting oxidative stress disrupts the balance between pro-oxidant and antioxidant systems, leading to lipid peroxidation, protein oxidation, DNA strand breaks, and activation of apoptotic cascades [14].

Redox-active metals such as chromium and iron generate ROS directly through Fenton-type reactions. Redox-inactive metals, including Pb, Cd, and Hg, deplete critical antioxidants, particularly glutathione (GSH) and selenocysteine-containing enzymes, thereby indirectly facilitating oxidative injury [13]. The NLRP3 inflammasome pathway and NF-κB signalling are activated by several heavy metals, amplifying pro-inflammatory cytokine release (TNF-α, IL-6) and perpetuating tissue damage [15].

#### 4.2 Organ-Specific and Metal-Specific Toxicology

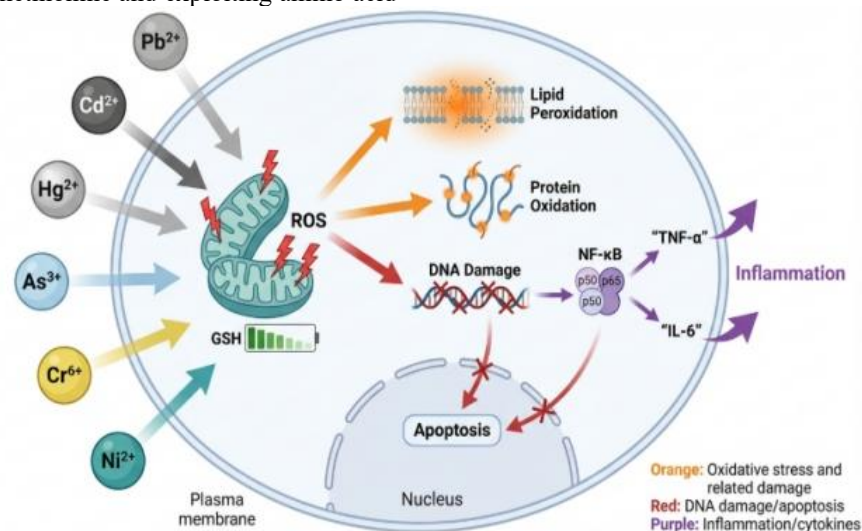
Lead (Pb) is a potent neurotoxin, particularly in children, where blood lead levels as low as 5 µg/dL are associated with reduced cognitive function, behavioural deficits, and irreversible intellectual impairment. Mechanistically, Pb inhibits δ-aminolevulinic acid dehydratase (ALAD) and ferrochelatase, disrupting heme biosynthesis and causing hemolytic anaemia [16]. Pb also substitutes for calcium in cellular signalling, disrupting protein kinase C activity and long-term potentiation in hippocampal neurons, with consequences for learning and memory [13].

Cadmium (Cd) exhibits high nephrotoxicity, accumulating in proximal renal tubular cells where it disrupts calcium reabsorption, leading to Itai-Itai disease (painful osteomalacia and renal tubular dysfunction) as documented historically in Japan. Cd inhibits hexokinase activity, affecting glucose metabolism, and activates NADPH oxidase, generating ROS [17]. Long-term Cd exposure is classified as a Group 1 carcinogen (IARC), with strong associations with lung and kidney cancer [14].

Mercury (Hg), in its methylated form, demonstrates extreme neurotoxicity, crossing both the blood-brain barrier and placenta. Methylmercury binds to cysteine residues on amino acids, masquerading as methionine and exploiting amino acid

transporters to enter the central nervous system. It inhibits glutathione peroxidase by binding to selenol groups, severely compromising cellular antioxidant defences [16]. Chronic low-level exposure is associated with cognitive decline, sensory disturbances, and, in prenatal exposures, severe neurodevelopmental disorders [17].

Arsenic (As) carcinogenicity is mediated via multiple pathways, including ROS generation, inhibition of DNA repair enzymes, epigenetic modifications, and induction of genomic instability. Inorganic arsenic is metabolised to monomethylarsonate (MMA) and dimethylarsinate (DMA); trivalent metabolites (MMA[III], DMA[III]) are particularly genotoxic [11,18]. Chronic arsenicosis manifests as peripheral neuropathy, Mees' lines, hyperkeratosis, and elevated risks of bladder, lung, and skin cancers [18]. Hexavalent chromium [Cr(VI)] is a well-characterised genotoxic carcinogen; its reduction to Cr(III) within cells generates Cr-DNA adducts and reactive intermediates that cause DNA double-strand breaks [13,14]. Nickel compounds are classified as Group 1 carcinogens, promoting lung and nasal cancer through epigenetic silencing of tumour suppressor genes [14]. Figure 2 shows the Molecular mechanisms of heavy metal toxicity: convergent pathways of ROS generation, antioxidant depletion, DNA damage, and apoptotic activation induced by Pb, Cd, Hg, As, Cr, and Ni.



**Figure 2. Molecular mechanisms of heavy metal toxicity: convergent pathways of ROS generation, antioxidant depletion, DNA damage, and apoptotic activation induced by Pb, Cd, Hg, As, Cr, and Ni.**

**Caption:** A scientific mechanistic diagram showing cellular heavy metal toxicity pathways. Central cell (cross-section view) showing: heavy metal ions ( $Pb^{2+}$ ,  $Cd^{2+}$ ,  $Hg^{2+}$ ,  $As^{3+}$ ,  $Cr^{6+}$ ,  $Ni^{2+}$ ) entering from extracellular space. Inside cell: mitochondria releasing ROS (red lightning bolts), GSH depletion shown with decreasing bar, NF- $\kappa$ B activation leading to cytokine release (TNF- $\alpha$ , IL-6), DNA double helix with damage marks (X), nucleus with apoptosis label. Arrows show cascade from metal entry to ROS to lipid peroxidation, protein oxidation, DNA breaks, and apoptosis.

**Sources:** Adapted from references [13,14,15,17]

## 5. Remediation Approaches

The remediation of heavy metal-contaminated environments is a multidisciplinary challenge requiring strategies tailored to the type of metal, its speciation, the contaminated matrix (soil, water, or sediment), and site-specific conditions. Remediation approaches are broadly categorised into

physical, chemical, and biological methods, each with distinct advantages and limitations [19]. Table 3 presents a comparative overview of these approaches. Table 3 gives the Comparative Summary of Heavy Metal Remediation Strategies: Mechanisms, Advantages, and Limitations

**Table 3. Comparative Summary of Heavy Metal Remediation Strategies: Mechanisms, Advantages, and Limitations [19,20,21,24,25]**

Approach	Method	Mechanism	Advantages	Limitations
Physical	Soil washing / excavation	Removal of contaminated matrix; surfactant-enhanced mobilization of metals	Rapid, effective for point sources	Costly, generates secondary waste
Chemical	Precipitation/solidification	pH adjustment causes metal precipitation as hydroxides/carbonates; cement-based immobilisation	Fast, well-established	Produces sludge; not permanent
Chemical	Ion exchange/adsorption	Selective metal binding to zeolites, activated carbon, biochar, and nano-sorbents	High selectivity, regenerable	High cost, membrane fouling
Biological – Phytoremediation	Phytoextraction (e.g., <i>Thlaspi caerulescens</i> )	Hyperaccumulator plants translocate metals to shoots via ZIP/HMA transporters	Eco-friendly, cost-effective, solar-powered	Slow, climate-dependent, metal speciation issues
Biological – Bioremediation	Microbial biosorption / biotransformation	Bacteria/fungi sequester metals via cell wall binding, enzymatic reduction (e.g., $\text{Cr(VI)} \rightarrow \text{Cr(III)}$ )	Sustainable, minimal disruption	Variable efficiency, scalability challenges
Electrokinetic	Electromigration through soil	Applied DC field mobilizes ionic metals toward electrodes for extraction	Effective in low-permeability soils	High energy use, electrode corrosion

metals toward electrodes for extraction, particularly effective in fine-grained, low-permeability soils [19].

### 5.1 Physical and Chemical Remediation

Physical methods such as soil excavation, capping, and containment remain effective for severely contaminated point-source sites but are costly, disruptive, and do not address the fundamental problem of metal persistence [19]. Soil washing using chelating agents (e.g., EDTA, DTPA) or acidic solutions can mobilise metals for extraction, but risks leaching metals into groundwater if not carefully controlled [6]. Chemical precipitation involves adjusting pH to precipitate metals as insoluble hydroxides, carbonates, or sulfides, a widely employed technology in industrial wastewater treatment [3]. Electrokinetic remediation applies a direct current across contaminated soil, migrating ionic

Advanced chemical approaches include the application of nano-scale zerovalent iron (nZVI), biochar-based sorbents, and metal-organic framework (MOF) materials, which offer high surface areas, tunable surface chemistry, and excellent adsorption capacities for multiple heavy metal species simultaneously [6,20]. Biochar derived from agricultural wastes has shown particular promise for immobilising Pb, Cd, and As in contaminated agricultural soils, reducing their bioavailability through surface complexation and co-precipitation mechanisms [20].

### 5.2 Biological Remediation

Phytoremediation employs living plants to extract, stabilise, or volatilise heavy metals from contaminated environments. Phytoextraction, the translocation of metals from soil to

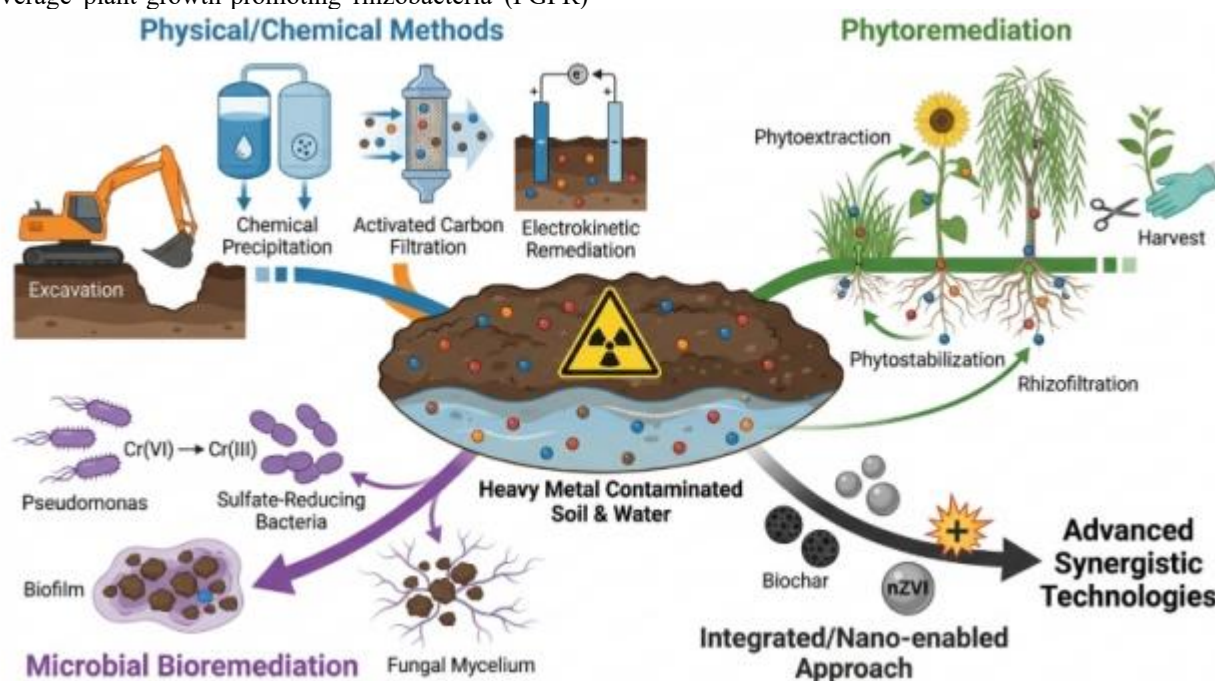
above-ground biomass, is mediated by membrane transporters of the ZIP, HMA, and NRAMP families, which facilitate metal uptake, root-to-shoot transfer, and vacuolar sequestration [21]. Hyperaccumulator species such as *Thlaspi caerulescens* (zinc and cadmium), *Pteris vittata* (arsenic), and *Arabidopsis halleri* exhibit extraordinary metal accumulation capacities, achieving shoot concentrations that exceed soil concentrations by factors of 100–1,000 [21,22]. Recent advances in plant genetic engineering using CRISPR-Cas9 and transgenic approaches aim to enhance metal uptake efficiency, stress tolerance, and biomass production in phytoremediator species [22].

Microbial bioremediation exploits the metabolic versatility of bacteria, fungi, and algae to biosorb, biotransform, or bioprecipitate heavy metals. Mechanisms include biosorption (passive binding to cell wall functional groups), bioaccumulation (active intracellular uptake), enzymatic reduction (e.g., Cr(VI) to Cr(III) by *Pseudomonas putida*), sulfate reduction to form insoluble metal sulfides, and siderophore-mediated metal complexation [23]. Bioaugmentation strategies involving engineered microorganisms with enhanced metal sequestration capacities have shown promise in controlled settings [23]. Combined microbe-plant remediation approaches (rhizoremediation) that leverage plant-growth-promoting rhizobacteria (PGPR)

to enhance phytoextraction efficiency represent a particularly promising integrated strategy [24].

### 5.3 Emerging and Integrated Approaches

Integrated remediation strategies that combine multiple approaches are increasingly recognised as most effective for complex contaminated sites. For example, combining biochar amendment (immobilising metals chemically) with phytoextraction plants (progressively removing metals biologically) has demonstrated synergistic effects in field trials [20,25]. Nano-enabled bioremediation, using nanoparticles to enhance microbial activity or plant uptake, represents a frontier area, though concerns regarding nanoparticle ecotoxicity necessitate careful evaluation [6]. The integration of multi-omics approaches (genomics, proteomics, metabolomics) into bioremediation research is accelerating the discovery of novel metal-resistance and sequestration mechanisms in microorganisms and plants [24]. Figure 3 gives the Overview of integrated heavy metal remediation strategies for contaminated soil and water, illustrating physical, chemical, and biological (phytoremediation and microbial bioremediation) approaches, their mechanisms, and the emerging combined/nano-enabled strategies.



**Figure 3. Overview of integrated heavy metal remediation strategies for contaminated soil and water, illustrating physical, chemical, and biological (phytoremediation and microbial bioremediation) approaches, their mechanisms, and the emerging combined/nano-enabled strategies.**

*Caption: A comprehensive scientific illustration showing integrated heavy metal remediation strategies for contaminated soil and water. Landscape-style diagram with contaminated land in the centre (dark brown soil with*

*chemical hazard symbol). Three main branches with icons: (1) Physical/Chemical methods - showing excavation machinery, precipitation tanks, activated carbon filters, electrodes in soil; (2) Phytoremediation - showing green plants with roots absorbing metals (labelled phytoextraction,*

phytostabilization, rhizofiltration), with arrow showing metal uptake to shoots and leaf harvest; (3) Microbial bioremediation - showing bacteria icons (*Pseudomonas*, sulfate-reducing bacteria) reducing Cr(VI) to Cr(III), biofilm on soil particles, fungal mycelium. Arrow emerging from the combination zone showing 'Integrated/Nano-enabled approach' with biochar particles and nZVI. Colour scheme: green for phytoremediation, blue for chemical, orange for physical, purple for microbial.

**Sources:** Adapted from references [19,20,21,24]

## 6. Policy Frameworks and Public Health Implications

Global regulatory frameworks for heavy metal pollution control have evolved substantially, yet significant gaps remain, particularly in low- and middle-income countries (LMICs). The Minamata Convention on Mercury (2013, entered into force 2017) represents the most comprehensive international treaty specifically targeting a heavy metal, mandating reductions in mercury supply, trade, and

anthropogenic emissions [12]. The European Union's Water Framework Directive and Soil Thematic Strategy establish science-based environmental quality standards for multiple heavy metals, while the US Environmental Protection Agency's IRIS (Integrated Risk Information System) provides toxicological reference values guiding regulatory decisions [3].

In LMICs, enforcement capacity, technological access, and financial resources constrain effective implementation of heavy metal standards. The health burden is disproportionately concentrated in communities adjacent to artisanal mining sites, informal e-waste processing zones, and heavily industrialised areas, with children facing the greatest risks due to higher exposure per body weight, hand-to-mouth behaviour, and greater sensitivity of developing organ systems [16]. Biomonitoring programs tracking blood lead levels, urinary cadmium, and hair mercury in exposed populations are essential public health tools, providing empirical data to drive targeted interventions [4].

## 7. Conclusions and Future Directions

Environmental heavy metal pollution remains a formidable global challenge that intersects toxicology, environmental science, public health, and materials science. This review has demonstrated that chemical speciation is paramount in determining the bioavailability, environmental mobility, and toxicological impact of heavy metals. The convergence of industrial, agricultural, and artisanal mining pressures continues to introduce substantial metal burdens into ecosystems at scales that outpace natural attenuation processes.

Toxicologically, ROS generation, antioxidant depletion, enzyme inhibition, and genotoxicity constitute the central

mechanisms underpinning heavy metal pathology, with organ-specific manifestations dependent on metal-specific toxicokinetics and exposure pathways. From a remediation perspective, no single approach is universally adequate; optimal outcomes require integrated strategies that are ecologically sensitive, economically viable, and scalable to contaminated site characteristics.

Future research priorities should include: (i) advanced real-time speciation monitoring using synchrotron-based and electrochemical sensing technologies; (ii) development of high-efficiency, multi-metal hyperaccumulator plants through precision genetic engineering; (iii) harnessing the power of microbiome engineering and synthetic biology for bioremediation applications; (iv) understanding the interactive effects of climate change on metal speciation and mobility; and (v) global harmonization of regulatory standards with particular attention to LMIC contexts. The convergence of nanotechnology, biotechnology, and environmental policy will be critical in achieving sustainable, long-term mitigation of heavy metal pollution.

## Author Contributions

The author has significantly contributed to the study's conception, design, data collection, analysis, and interpretation. All authors were involved in writing the manuscript or critically reviewing it for its intellectual value. They have reviewed and approved the final version for submission and publication and accept full responsibility for the content and integrity of the work.

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## Conflicts of Interest

The authors state that there are no financial, personal, or professional conflicts of interest associated with this research.

## Ethical Approvals

This research did not involve the use of human participants or animal subjects and therefore did not require ethical clearance

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