

Environmental Risks of Heavy Metal Accumulation in Farmland and Its Influence on Crop Performance

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Abstract: -

Heavy metals and metalloids (HMs) such as cadmium, lead, arsenic, mercury, and chromium are major environmental contaminants. When these biodegradable elements build up to harmful levels in agricultural soils, they negatively affect plant growth and crop productivity. The degree of HM toxicity in crops is influenced by several factors, including the crop species, growth conditions, developmental stage, specific toxic properties of each element, soil physical and chemical characteristics, the presence and bioavailability of HM ions in the soil solution, and rhizosphere interactions. HMs can damage cellular structures, disrupt normal cellular functions, and interfere with key metabolic and developmental processes.

Introduction:

including those considered Metals potentially toxic are inorganic elements with RE NT Transition metals: chromium (Cr), atomic densities several times greater than that of water (1 g cm⁻³). They are broadly categorized into heavy metals, light metals, and semi-metals. Based on their physical, chemical, and physiological characteristics, metals can be grouped into several classes,

- manganese (Mn), iron (Fe), cobalt (Co), nickel (Ni), copper (Cu), and molybdenum (Mo);
- Post-transition metals: aluminum (Al), zinc (Zn), cadmium (Cd), mercury (Hg), and lead (Pb);

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- Alkaline earth metals: calcium (Ca), magnesium (Mg), beryllium (Be), and barium (Ba);
- Alkali metals: lithium (Li), sodium (Na), potassium (K), and cesium (Cs);
- Metalloids (semi-metals): elements exhibiting both metallic and nonmetallic traits, such as boron (B), silicon (Si), arsenic (As), and antimony (Sb).

Heavy metals and metalloids (HMs) are significant environmental pollutants and major contaminants of agricultural soils. When present in high concentrations, these elements can severely impair crop growth and yield. Because HMs are not easily degraded, they tend to persist in soils for long periods, accumulating if they are not absorbed by plants or removed through leaching. Common include Cd, Pb, Cr, As, Hg, Ni, Cu, and Zn, with Cd, Pb, As, Hg, and Cr being particularly harmful even at very low levels. Many metals, such as Cu, Zn, Fe, Mn, Mo, Ni, Mg, Ca, and B, are essential nutrients required for plant growth and metabolic activities including ion balance, pigment formation, photosynthesis, respiration, enzyme function, gene expression, carbohydrate metabolism, and nitrogen fixation. However, these beneficial elements become toxic when present above optimal levels and can negatively influence plant

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growth. development, and reproduction. Likewise, if their concentrations drop below critical thresholds, plants show deficiency symptoms. Heavy metal contamination of agricultural land is a worldwide concern. While certain natural and climatic factors contribute, rapid urbanization and increased industrial, municipal, agricultural, domestic, medical, and technological activities have intensified HM pollution. The issue is particularly severe in many developing nations, often due to limited awareness of the harmful effects of these pollutants on both human and crop health. This review focuses specifically on how heavy metals adversely impact crop health.

Mechanisms of Plant Growth Inhibition by **HMs**

Plants take up heavy metals (HMs) toxic metals found in contaminated farmland R from the soil solution through their roots, primarily as ionic forms, and move them into different subcellular compartments using a range of ion channels and transporter proteins such as HM-ATPases, ATP-binding cassette transporters, and cation diffusion facilitators. When HMs interfere with root development, they disrupt the plant's water status as well as the uptake and movement of essential mineral nutrients, ultimately reducing overall growth, biomass production, and yield. Moreover, once HM concentrations surpass a critical threshold within plant tissues, they disturb ionic



homeostasis across cellular membranes and impair the structure and functionality of key including organelles chloroplasts, mitochondria, the nucleus, and vacuoles as well as major macromolecules such as carbohydrates, lipids, proteins, and nucleic acids. High HM levels have been reported to disrupt chloroplast ultrastructure, chlorophyll a/b ratios, inhibit the synthesis of photosynthetic components, modify pigment organization in grana and stroma membranes, and reduce the activities of several catalytic and non-catalytic proteins involved in diverse metabolic and developmental pathways.

Inhibition of **Photosynthetic Phosphorylation**

During the light-dependent phase of photosynthesis, many heavy metals (HMs) disrupt primary photochemical processes, electron flow and photophosphorylation. These inhibitory effects have been observed in isolated chloroplasts, thylakoid membranes, and photosystem II (PSII) sub-membrane fractions [128,147,148]. Evidence indicates that PSII-driven electron transport is generally sensitive HM toxicity more to photosystem I (PSI). Although the precise mechanism by which HMs impair PSII remains incompletely understood, the oxygenevolving complex (OEC) has been proposed as a major target site. Studies measuring variable

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PSII chlorophyll fluorescence of intact membranes exposed to Pb^{2+} and other additives suggest that HMs primarily act on the water-oxidizing side (WOS) of PSII. Increasing Pb2+ concentrations were found to progressively suppress the rise in variable fluorescence, indicating that electron transfer at the WOS of PSII is inhibited under Pb²⁺ stress. These findings were further supported by immunoblot analyses using antibodies against the three extrinsic polypeptides of the oxygen-evolving complex (OEC), with molecular masses of 16, 23, and 33 kDa. The results demonstrated that Pb2+ and Zn2+ selectively removed these polypeptides from the OEC [150]. These proteins normally function as protective components, shielding the OEC from external reductants in PSII submembrane preparations. Since the loss of these leading to the inhibition of photosynthetic Rextrinsic polypeptides whether caused by heavy metals or by detergent treatments is known to deactivate PSII, it follows that the production of ATP and NADPH through noncyclic photophosphorylation (required for CO2 reduction to carbohydrates) would also be impaired. Nevertheless, even when non-cyclic photophosphorylation is compromised, plants may still rely on PSI-driven cyclic electron flow to generate a limited amount of ATP, allowing CO₂ fixation to continue, albeit at a reduced efficiency.

Generation of Oxidative Stress



harmful When exposed concentrations of heavy metals, plants produce excessive amounts of reactive oxygen species (ROS) including superoxide (O₂⁻), hydrogen peroxide (H₂O₂), hydroxyl radicals (OH·), and singlet oxygen (1O2) at multiple cellular locations such as mitochondria, chloroplasts, peroxisomes, and the outer surface of the plasma membrane. Both redox-active metals (e.g., Cr, Cu, Mn, Fe) and non-redox-active metals (e.g., Cd, Ni, Hg, Zn, Al) generate ROS through distinct pathways, but the resulting oxidative stress damages essential macromolecules lipids, proteins, and nucleic acids. This oxidative injury manifests as lipid peroxidation; protein oxidation, misfolding, and aggregation; and DNA double-strand breaks. Although ROS produced under various stresses including heavy metal exposure can sometimes enhance resistance to certain fungal R NADP+-glyceraldehyde-3-phosphate pathogens, plants primarily rely on an antioxidant defense network to detoxify ROS. This system includes enzymes such as superoxide dismutase, catalase, peroxidases, and glutathione reductase, which help mitigate oxidative damage. However, certain heavy metals can impair the activities of these protective enzymes. Altogether, existing evidence indicates that excessive heavy metal buildup in agricultural soils and subsequent accumulation in plants harms crop health and productivity not only by triggering oxidative

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stress but also by compromising antioxidant defense machinery. Nevertheless, more research is required to elucidate the precise interactions between heavy metal stress and antioxidant responses in plants.

Inactivation of Plant Enzyme Activities

Heavy metals disrupt vital metabolic and developmental processes in plants by binding to enzyme active sites and interacting with functional groups such as carboxyl, amino, carbonyl, and sulfhydryl groups, leading to enzyme and protein inactivation. For example, several heavy metals suppress key enzymes associated with carbohydrate and phosphorus metabolism such as rubisco (ribulose-1,5-bisphosphate carboxylase), phosphoenolpyruvate carboxylase, phosphoribulokinase, aldolase. fructose-6phosphate kinase, fructose-1,6-bisphosphatase, dehydrogenase, carbonic anhydrase, and various phosphatases by binding to their

Because many heavy metals have strong affinity for sulfhydryl (-SH) groups, they can also disrupt disulfide bonds, thereby impairing the structure and activity of photosynthetic proteins and water channel proteins. In some cases, heavy metals interfere with the proper folding of nascent proteins, leading to their aggregation. Nickel and

functional side chains and altering their

conformations.



cadmium, for instance, can cause protein unfolding, rendering them non-functional, although the plant chaperone system may attempt to refold them. Zinc ions can deactivate rubisco by replacing magnesium at its active site. Likewise, Pb and Zn can inactivate the water-oxidizing enzyme of PSII by removing Mn from the tetra-Mn cluster, along with the 33-kDa extrinsic polypeptide.

Overall, heavy-metal-induced enzyme inactivation and protein denaturation result in widespread physiological disturbances in plants, ultimately impairing growth and reducing crop productivity.

Genetic Modifications: Effects on DNA Metabolism

Heavy metals are considered genotoxic but not necessarily mutagenic, as there is no evidence indicating that they directly induce gene mutations in plants, although Crottisupper Management in plants i known to be mutagenic in mammalian cells. It is important to distinguish that while all mutagens are genotoxic, not all genotoxic agents are mutagenic. As described earlier, heavy metals can damage DNA in both plants and animals through the excessive production of reactive oxygen species (ROS). They may also hinder DNA replication and repair by inactivating key enzymes involved in these processes. For example, arsenic inhibits Poly (ADP-ribose) polymerase-1 in humans, an enzyme essential for repairing oxidative DNA

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strand breaks. Metals such as Cd, Hg, and Pb exert strong genotoxic effects in plants, resulting in various forms of DNA lesions. High concentrations of Cd and Pb have been shown to cause extensive double-strand breaks and genomic instability in fava bean (Vicia faba). Similarly, soils contaminated with Hg, Pb, Cu, Cd, and Zn increase chromosomal abnormalities such as bridges, laggards, stickiness, and fragmentations in chickpea (Cicer arietinum). Some heavy metals can also interact directly with DNA by binding to nucleobases or the phosphate backbone, leading to strand cleavage. Mercury, in particular, can form covalent bonds with DNA, resulting in sister chromatid exchanges, reduced mitotic index, and higher frequencies of chromosomal aberrations.

Conclusions and Perspectives

degradable elements. making their accumulation in agricultural soils a serious for sustainable global challenge crop production. Toxic effects become evident when HM levels exceed permissible limits in soils or surpass plant tolerance thresholds. Their availability for plant uptake generally increases under acidic soil conditions, low organic matter, and reduced cation exchange capacity (CEC). The decline in plant growth and yield under HM stress results from multiple direct impacts such as oxidative



stress, disrupted ion balance and water regulation, reduced nutrient uptake and assimilation, inhibited photosynthesis enzyme function, and hormonal imbalance as well as indirect effects, particularly the suppression of beneficial soil microbes. Plants employ three primary defense strategies against heavy metal toxicity. The first involves limiting metal entry by forming extracellular complexes with organic acids (e.g., oxalic, citric, malic, tartaric acids), amino acids, secondary metabolites (phenolics, flavonoids, alkaloids, S- and N-containing compounds), or microbial metabolites in the rhizosphere. The second line of defense includes metal chelation by cell wall components (cellulose, hemicellulose, pectin) or proteins, followed by sequestration in vacuoles or_ osmotic adjustment through soluble sugars and proteins. The detoxifying ROS via the plant's antioxidant systems. Failure of these barriers or excessive metal accumulation that overwhelms them leads to severe impairment of plant growth and crop productivity.

To reduce the risk of HM-induced crop injury, farmers should maintain detailed records of pesticide, fertilizer, manure, compost, biosolid applications, and irrigation practices. They should adopt appropriate tillage and crop rotation methods, preserve adequate soil organic matter, and use chemical

inputs judiciously. Because microbial populations are sensitive indicators of metal testing through stress, soil accredited microbiological laboratories can help assess microbial biomass and diversity. Chemical analyses of soil pH, organic matter, and CEC can further determine metal solubility and plant-available fractions. Before investing in extensive laboratory testing, a simple soil bioassay using HM-sensitive cultivars can provide preliminary evidence of toxicity. If contamination is confirmed, proper remediation strategies should be implemented. For researchers in molecular agriculture, developing HM-tolerant crop varieties through genetic enhancement of metal-binding and antioxidant defense pathways is essential. Equally important is the creation of microbial biosensors for rapid detection of third defense Grelies on R contamination. ESustained funding will be critical to support these research efforts and advance sustainable soil management and crop productivity.

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