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Lead in Plants: Sources, Toxicity, and Tolerance Mechanisms

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ABSTRACT

Lead (Pb) stands out as the most prevalent heavy metal contaminant in the environment. While plants absorb Pb from their surroundings, it is not a vital element for their growth. Pb is notably abundant, especially in the soil of roadside fields due to emissions from automotive exhaust. Additionally, it is present in fields with a prolonged history of fertilization using fertilizers that contain Pb as an impurity. Numerous sources contribute to Pb contamination, including soil, water, air, batteries, toys, cans, and fertilizers. In this review, our focus is on examining the impact of Pb on plant growth and development, along with exploring the mechanisms plants employ to endure lead toxicity. Pb ranks among the most frequently encountered heavy metals in terrestrial and aquatic ecosystems, entering through various natural and human-induced sources. The accumulation of Pb in plants is dose-dependent and leads to toxicity. The uptake of Pb increases the concentration of Mn, while reducing the total concentrations of most other minerals, including K, Ca, Na, P, Mg, Zn, Fe, and Cu. Plant exposure to Pb limits the sprouting and development of young seedlings. Plants defend themselves against Pb toxicity through various mechanisms, including passive, inducible, and antioxidant enzyme-based mechanisms. In conclusion, Pb poses

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harm, but plants have evolved mechanisms to resist its adverse effects. Further research should focus on selecting and developing cultivars with superior tolerance to Pb.

Keywords: Lead, Sources of lead, Toxicity, Tolerance mechanism, uptake

Introduction:

Heavy metals, like chromium (Cr), cobalt, nickel (Ni), copper (Cu), zinc (Zn), arsenic (As), selenium (Se), silver (Ag), cadmium (Cd), antimony (Sb), mercury (Hg), thallium (TI), and lead (Pb), are environmentally hazardous metals or metalloids denser than iron [1]. These metals naturally occur in the earth and are released during weathering, with concentrations known as background concentrations. Common sources of heavy metal contamination result from human activities, including the disposal of industrial and domestic wastes, vehicular emissions, wastes from Pb acid batteries, paints, treated woods, and the use of various organic and mineral fertilizers [1]. In the 1930s-1970s, Pb was extensively used in gasoline, significantly increasing Pb levels in the aquatic environments of industrialized societies [4]. Although the use of leaded gasoline in North America largely phased out by 1996, soils near roads still have high Pb concentrations. From about 500 BC to 300 AD, lead was extensively used in Roman aqueducts. The use of lead azide or lead styphnate in firearms leads to Pb accumulation in firearms training grounds, posing a risk of Pb poisoning to the local population, especially firing range employees [5]. Heavy metals reach plants, animals, and human tissues through air inhalation, contaminated diet, and manual handling. Airborne contamination, mainly from motor vehicle emissions, is a major source, although the origin of heavy metals is not clear [6]. Leaching from consumer and industrial wastes can pollute water sources, and acid rain can worsen this process by releasing trapped heavy metals in soils [7]. These metals enter plants through water uptake, animals through contaminated plant consumption, and humans primarily through the ingestion of plant- and animal-based foods. Another potential source of heavy metal contamination is skin contact, followed by absorption through the skin [8]. Heavy metals can accumulate in organisms as they are not metabolized [9]. The paper aims to review recent literature on the effects of Pb toxicity on plant growth and enzymatic activities. Salient results are discussed with examples from recent literature, covering varietal differences in tolerance or resistance to Pb toxicity, along with the need for future research to reduce Pb toxicity in crops.

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Lead contamination from different sources

Toxic lead poses health risks based on factors like concentration, current health, exposure route (air, water, or food), and age. Up to 20% of children's lead exposure may be from water, with contaminated water being a significant risk for young children, fetuses, and infants. Lead processing plants handle both primary and secondary sources. Primary lead is mined, separated, and refined, while secondary lead comes from used objects like lead-acid batteries. Smelting, a crucial process, involves heating lead ore or recovered lead, releasing significant amounts into the environment. Lead sources include home fittings, water pipes with lead soldering, and water sitting in recently built houses with these fittings, especially those less than 5 years old. Various common products, such as toys, candies, and traditional medicines, are unexpected sources of lead. Dust and chips from old paints are common culprits in lead contamination of soil and water, leading to poisoning through the food chain.

i. **Soil:** Pb, phased out from gasoline since 1996 in the USA/India, still poses a risk from car exhaust near roads, industrial units, and flaking paint. Wind can spread Pb-contaminated dust, impacting homes [10].

ii. **Drinking Water:** Pb seldom occurs naturally; it enters water through corrosion in household plumbing. Older constructions may have Pb-contributing plumbing, and though regulations limit Pb content, older systems may still pose a risk [10].

iii. **Paint:** Pb was banned in home paints in 1978 in the USA, but homes, furniture, and toys made before then may still have Pb-based paints. When the paint chips turn to dust or mix with soil, it becomes a concern [10].

iv. **Dust:** Pb exposure often occurs through dust generated during home activities like scraping or sanding paint. Young children are at risk when they ingest Pb dust from items or surfaces [10].

v. **Air:** Outdoor Pb comes from industrial sources, soil, road dust, and past use of leaded gasoline. Indoor sources include outdoor air, dust, and certain hobbies. Motor vehicle emissions are a major source of airborne contaminants, including Pb [6].

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vi. **Folk Medicines, Ayurvedic Medicines, and Cosmetics:** Some folk medicines, especially from Southeast Asia, may contain Pb contaminants. Ayurvedic medicines, practiced mainly in India and Eastern Asian countries, may contain Pb. Some cosmetics, like Surma and Kohl, have been reported to contain Pb.

vii. **Lead Acid Batteries:** Residents in Uznova reported Pb exposure, leading to testing and awareness initiatives by EDEN Center in collaboration with government agencies and the media [2].

Effects and Mechanisms of Lead Toxicity on Plants

Pb, a prevalent heavy metal in terrestrial and aquatic ecosystems, enters through various natural and human-made sources [11-13]. Plants absorb Pb from the soil solution, primarily retained in roots in a precipitated form [14]. Pb accumulation varies among plant species, impacting seedling development [15-17]. Low Pb concentrations hinder aerial and root growth, while higher concentrations strongly affect root growth, resulting in stubby, short, bent, and swollen roots with more secondary roots per unit length [18]. Pb accumulation leads to reduced plant growth and mineral uptake, affecting concentrations of Na, K, Ca, P, Mg, Fe, Cu, and Zn, while increasing Mn concentration. Mineral nutrient deficiency results in a more pronounced decrease in proline, chlorophylls a and b, soluble proteins, and various biochemical and physiological dysfunctions, affecting seed germination, nitrate assimilation, water status, and plant growth [13,20,21]. However, Pb transport from root to shoot has limitations [22]. Pb negatively impacts carbon dioxide assimilation, photosynthetic rate, carotenoid contents, and chlorophyll, significantly reducing the photosynthetic rate in plants [23]. After Pb exposure, a reduction in Ca, Fe, and Zn levels in root tips is observed, signifying inhibition of mineral ion uptake. Increased provision of specific inorganic salts can partially alleviate Pb effects [25]. Pb toxicity triggers the generation of free radicals, inducing oxidative stress and the production of reactive oxygen species (ROS) in plants [26]. Pb exposure induces morphological changes in plant cells, including mitochondrial swelling, vacuolization of the endoplasmic reticulum, injured plasma membrane, dictyosomes, and deep-colored nuclei after 48-72 hours [27]. Lead interacts with proteins in the cytoplasm, and a higher concentration of Pb may reduce the protein pool [28-31]. The quantitative decrease in total protein occurs with Pb addition due to modifications in gene expression, increased

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ribonuclease activity [17], acute oxidative stress of reactive oxygen species (ROS) [31-33], protein utilization by plants for Pb detoxification, and a decrease in free amino acid content [32], correlated with disturbances in nitrogen metabolism [28]. Certain amino acids, like proline, increase under Pb stress, playing a crucial role in Pb tolerance by the plant. Conversely, low concentrations of Pb increase total protein content [29].

Lead Tolerance mechanisms

Plants counteract Pb's harmful effects through various responses. They absorb selective metals, bind them to the root surface, and induce antioxidants like proline, NP-SH, glutathione, cysteine, ascorbic acid, and enzymes such as GPX, SOD, CAT, APX, and GR [33].

i. **Passive Mechanisms:** Pb interacts with cellular components, increasing cell wall thickness even with a small amount. Plant cell walls with pectin complex with Pb through carboxyl groups, a crucial interaction for resisting Pb toxicity [25]. In F. *hygrometrica protonemata*, Pb binding to JIM5-P acts as a physical barrier, limiting Pb access to the plasma membrane. Subsequent studies reveal that Pb bound to JIM5-P can be taken up or remobilized within the cell through endocytosis along with pectin epitope [35].

ii. **Inducible Mechanisms:** Transporter proteins among plant cells play a vital role in metal detoxification, enabling metal ion excretion into extracellular spaces [36-38]. The human DMT1 expressed in yeast transports Pb via a pH-dependent process [39] in plants. ATP-binding cassette carriers like AtATM3 or AtADPR12 at ATP-binding sites in Arabidopsis contribute to Pb resistance [40,41]. Although suspected to act against Pb, this detoxification mechanism hasn't been definitively established. Transcriptome analysis shows that gene expression of these carriers is stimulated by Pb [11].

iii. **Antioxidant Enzymes:** Plants have an antioxidant enzyme system to counteract oxidative damage and manage increased ROS production in different cell compartments [32]. The synthesis of these enzymes may be induced or inhibited by Pb-induced toxicity. Pb-induced induction or inhibition of antioxidant enzymes depends on metal, plant species, specific form of the metal, and the duration/intensity of the treatment [19,32]. Generally, Pb inhibits plant enzymatic activities, with the Ki ranging between 10^{-5} and 2×10^{-4} M, resulting in 50%

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inhibition in enzymatic activities within this concentration range [21]. Pb's affinity for enzyme -SH groups suggest enzyme inhibition [13,32]. These findings, validated on more than 100 enzymes, including nitrate reductase and RuBisCO, indicate altered tertiary protein structures on catalytic sites or elsewhere, leading to inactivation. Pb can bind with the protein-COOH group, producing a similar effect, and interact with metalloid enzymes. Indeed, Pb can disrupt some essential parts of these enzymes involved in plant absorption of minerals, including Mn, Zn, and Fe. Pb and other divalent cations can also substitute for these metals, leading to enzyme inactivation, as observed with ALAD [32,42,43]. The impact of Pb on ROS constitutes another mechanism through which Pb exposure influences protein behavior [32].

Conclusion:

Lead (Pb) is a harmful metal for crops, and it's found extensively in the environment. It contaminates the entire ecosystem through the soil, air, water, and food materials. Plants possess different mechanisms to resist or tolerate lead. This enables us to pinpoint cultivars with higher tolerance to lead. We can use biotechnological tools to create cultivars that resist lead toxicity by identifying the responsible gene(s).

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