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**Bertrand Pourrut, Muhammad Shahid, Camille Dumat, Peter Winterton,
and Eric Pinelli**

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1 Introduction

Plants are the target of a wide range of pollutants that vary in concentration, speciation, and toxicity. Such pollutants mainly enter the plant system through the soil (Arshad et al. 2008) or via the atmosphere (Uzu et al. 2010). Among common pollutants that affect plants, lead is one of the most toxic and frequently encountered (Cecchi et al. 2008; Grover et al. 2010; Shahid et al. 2011). Lead continues to be used widely in many industrial processes and occurs as a contaminant in all environmental compartments (soils, water, the atmosphere, and living organisms). The prominence of environmental lead contamination results both from its persistence (Islam et al. 2008; Andra et al. 2009; Punamiya et al. 2010) and from its present and past numerous sources. These sources have included smelting, combustion of leaded gasoline, or applications of lead-contaminated media (sewage sludge and fertilizers) to land (Piotrowska et al. 2009; Gupta et al. 2009; Sammut et al. 2010; Grover et al. 2010). In 2009, production of recoverable lead from mining operations was 1690, 516, and 400 thousand metric tons by China, Australia, and the USA, respectively (USGS 2009).

Despite a long history of its beneficial use by humankind, lead has no known biological function in living organisms (Maestri et al. 2010) and is now recognized as a chemical of great concern in the new European REACH regulations (EC 1907/2006; Registration, Evaluation, Authorization, and Restriction of Chemicals). Moreover, lead was reported as being the second most hazardous substance, after arsenic, based on the frequency of occurrence, toxicity, and the potential for human exposure by the Agency for Toxic Substances and Disease Registry (ATSDR 2003). The transfer of lead from polluted soils to plants was therefore widely studied, especially in the context of food quality, use in phytoremediation, or in biotesting (Arshad et al. 2008; Uzu et al. 2009).

Lead is known to induce a broad range of toxic effects to living organism, including those that are morphological, physiological, and biochemical in origin. This metal impairs plant growth, root elongation, seed germination, seedling development, transpiration, chlorophyll production, lamellar organization in the chloroplast, and cell division (Sharma and Dubey 2005; Krzesłowska et al. 2009; Gupta et al. 2009, 2010; Maestri et al. 2010). However, the extent of these effects varies and depends on the lead concentration tested, the duration of exposure, the intensity of plant stress, the stage of plant development, and the particular organs studied. Plants have developed various methods for responding to toxic metal exposures. They have internal detoxification mechanisms to deal with metal toxicity that includes selective metal uptake, excretion, complexation by specific ligands, and compartmentalization (Gupta et al. 2009; Krzesłowska et al. 2010; Maestri et al. 2010; Sing et al. 2010; Jiang and Liu 2010).

The various responses of plants to lead exposure are often used as tools (bioindicators) in the context of environmental quality assessment. To develop tools that are relevant for ecotoxicological studies, it is essential to understand the mechanisms involved in plant uptake, transfer, and toxicity. This is especially true in selected research areas, such as choice of plant species, when polluted soils are under study.

(i.e., reduced transfer when studying vegetables or increased transfer when phytoextraction is desired). For example, legumes are considered more suitable to grow on contaminated soil than Umbelliferae, Liliaceae, Compositae, and Chenopodiaceae, because they take up reduced amounts of lead (Alexander et al. 2006). The reduced lead uptake by vegetables minimizes the threat of lead introduction to the food chain. In contrast, phytoextraction requires plants that can sequester excessive amounts of lead in their biomass without incurring damage to basic metabolic functions (Arshad et al. 2008; Zaier et al. 2010). *Pelargonium* (Arshad et al. 2008) and *Brassica napus* (Zaier et al. 2010) are characterized as Pb hyperaccumulators, and they can extract huge amounts of lead from contaminated soil without showing morphophytotoxicity symptoms. Indeed, these plants have efficient natural detoxification mechanism to alleviate lead toxicity. In this review, we propose to trace the relationship that exists between lead uptake, accumulation, translocation, and toxicity in plants.

2 Retention, Mobility, and Bioavailability of Lead in Soil

Lead occurs naturally in the earth's crust (Arias et al. 2010) and its natural levels remain below 50 mg kg⁻¹ (Pais and Jones 2000). But, anthropogenic activities often modify the amount and nature of lead species present in soil. In soils, lead may occur as a free metal ion, complexed with inorganic constituents (e.g., HCO₃⁻, CO₃²⁻, SO₄²⁻, and Cl⁻), or may exist as organic ligands (e.g., amino acids, fulvic acids, and humic acids); alternatively lead may be adsorbed onto particle surfaces (e.g., Fe-oxides, biological material, organic matter, and clay particles) (Uzu et al. 2009; Tabelin and Igarashi 2009; Sammut et al. 2010; Vega et al. 2010). Anthropogenic-sourced lead generally accumulates primarily in the surface layer of soil, and its concentration decreases with depth (Cecchi et al. 2008). Because of its strong binding with organic and/or colloidal materials, it is believed that only small amounts of the lead in soil are soluble, and thereby available for plant uptake (Kopittke et al. 2008; Punamiya et al. 2010).

However, lead behavior in soil, in the context of species, solubility, mobility, and bioavailability, is largely controlled by complex interactions governed by many biogeochemical factors (Punamiya et al. 2010). These factors include pH (Kopittke et al. 2008; Lawal et al. 2010; Vega et al. 2010), redox conditions (Tabelin and Igarashi 2009), cation-exchange capacity (Vega et al. 2010), soil mineralogy (Dumat et al. 2006), biological and microbial conditions (Arias et al. 2010), amount of lead present (Bi et al. 2010; Cenkci et al. 2010; Lawal et al. 2010), organic and inorganic ligand levels (Padmavathiamma and Li 2010; Sammut et al. 2010; Shahid et al. 2011), competing cation levels (Kopittke et al. 2008; Komjarova and Blust 2009), and plant species involved (Kovalchuk et al. 2005; Bi et al. 2010; Liu et al. 2010). Such factors may act individually or in combination with each other and may alter the soil behavior of the lead present, as well as the rate of uptake by plants.

Lead bioavailability is strongly influenced by its speciation and, in particular, by the concentration of free lead ions present (Dumat et al. 2006; Uzu et al. 2009; Sammut et al. 2010; Shahid et al. 2011). This is because the most significant plant uptake route for many cationic metals (and especially for the free metal ion) is via the soil solution in dissolved form (Punamiya et al. 2010). Moreover, the free lead ion concentration in soils depends on the adsorption/desorption processes in which it participates (Vega et al. 2010).

3 Lead Behavior in Plants

3.1 Lead Uptake by Plants

With the exception of the special conditions that exist for plants cultivated near metal recycling industries (Uzu et al. 2010), the main pathway by which plants accumulate metals is through root uptake from soils (Sharma and Dubey 2005; Uzu et al. 2009). Part of the lead present in the soil solution is adsorbed onto the roots, and then becomes bound to carboxyl groups of mucilage uronic acid, or directly to the polysaccharides of the rhizoderm cell surface (Seregin and Ivanov 2001). Lead adsorption onto roots has been documented to occur in several plant species: *Vigna unguiculata* (Kopittke et al. 2007), *Festuca rubra* (Ginn et al. 2008), *Brassica juncea* (Meyers et al. 2008), *Lactuca sativa* (Uzu et al. 2009), and *Funaria hygrometrica* (Krzeslowska et al. 2009, 2010). Once adsorbed onto the rhizoderm roots surface, lead may enter the roots passively and follow translocating water streams. However, lead absorption is not uniform along plant roots as a lead concentration gradient from root apex can be observed (Tung and Temple 1996; Seregin et al. 2004). Indeed, the highest lead concentrations can be found in root apices, where root cells are young and have thin cell walls (with the exception of root cap cells) that facilitate root uptake (Tung and Temple 1996; Seregin et al. 2004). Moreover, the apical area is the area where rhizodermic pH is the lowest, which increases solubility of lead in the soil solution.

At the molecular level, the mechanism by which lead enters roots is still unknown. Lead may enter the roots through several pathways, and a particular pathway is through ionic channels. Although, lead uptake is a non-selective phenomenon, it nonetheless depends on the functioning of an H⁺/ATPase pump to maintain a strong negative membrane potential in rhizoderm cells (Hirsch et al. 1998; Wang et al. 2007). Inhibition of lead absorption by calcium is well-known (Garland and Wilkins 1981; Kim et al. 2002) and is associated with competition between these two cations for calcium channels (Huang and Cunningham 1996). Several authors have demonstrated that Ca²⁺-permeable channels are the main pathway by which lead enters roots (Wang et al. 2007; Pourrut et al. 2008). The use of transgenic plants has shown that lead can penetrate into roots through alternative non-selective pathways, such as cyclic nucleotide-gated ion channels (Arazi et al. 1999; Kohler et al. 1999) or via low-affinity cation transporters (Wojas et al. 2007).

Reduced uptake and translocation of lead to aerial plant parts of vegetables is considered to be beneficial in preventing lead from entering the food chain. However, reduced uptake and translocation of lead to aerial plant parts, when plants are used to remediate polluted soils, is a major problem. Indeed, soil remediation requires plants (hyperaccumulators) that can take high lead levels up and translocate it to aerial plant parts with no or minimal toxicity. The amount of lead that moves from soil to penetrate into plants can be measured by what is called “the transfer factor”; this factor is defined as the ratio that exists between the concentration of lead in the plant vs. the concentration of lead in the soil (Arshad et al. 2008; Bi et al. 2010; Liu et al. 2010). This transfer factor will be different for different plant species and will change as soil physical and chemical properties are altered (Arshad et al. 2008; Bi et al. 2010; Liu et al. 2010). Generally, plants having a transfer factor greater than 1 are categorized as hyperaccumulators, whereas those with transfer factor less than 1 are termed as non-accumulators of lead (Arshad et al. 2008).

3.2 Lead Accumulation in Plants

Once lead has penetrated into the root system, it may accumulate there or may be translocated to aerial plant parts. For most plant species, the majority of absorbed lead (approximately 95% or more) is accumulated in the roots, and only a small fraction is translocated to aerial plant parts, as has been reported in *Vicia faba*, *Pisum sativum*, and *Phaseolus vulgaris* (Piechalak et al. 2002; Małecka et al. 2008; Shahid et al. 2011), *V. unguiculata* (Kopittke et al. 2007), *Nicotiana tabacum*, (Gichner et al. 2008), *Lathyrus sativus* (Brunet et al. 2009), *Zea mays* (Gupta et al. 2009), *Avicennia marina* (Yan et al. 2010), non-accumulating *Sedum alfredii* (Gupta et al. 2010), and *Allium sativum* (Jiang and Liu 2010). Although many metals display the translocation restriction phenomenon mentioned above, this phenomenon is not common to all heavy metals. Notwithstanding, this phenomenon in plants is both specific and very intense for lead.

When entering the root, lead mainly moves by apoplast and follows water streams until it reaches the endodermis (Tanton and Crowd 1971; Lane and Martin 1977). There are several reasons why the transport of lead from roots to aerial plant parts is limited. These reasons include immobilization by negatively charged pectins within the cell wall (Islam et al. 2007; Kopittke et al. 2007; Arias et al. 2010), precipitation of insoluble lead salts in intercellular spaces (Kopittke et al. 2007; Islam et al. 2007; Meyers et al. 2008; Małecka et al. 2008), accumulation in plasma membranes (Seregin et al. 2004; Islam et al. 2007; Jiang and Liu 2010), or sequestration in the vacuoles of rhizodermal and cortical cells (Seregin et al. 2004; Kopittke et al. 2007).

However, these reasons are not sufficient to explain the low rate of lead translocation from root to shoot. The endoderm, which acts as a physical barrier, plays an important role in this phenomenon. Indeed, following apoplastic transport, lead is blocked in the endodermis by the Caspary strip and must follow symplastic

transport. In endodermis cells, the major part of lead is sequestered or excreted by plant detoxification systems (c.f. Section 5.2).

Several hyperaccumulator plant species, such as *Brassica pekinensis* and *Pelargonium*, are capable of translocating higher concentrations of lead to aerial plant parts, without incurring damage to their basic metabolic functions (Xiong et al. 2006; Liu et al. 2008; Arshad et al. 2008). A specific hyperaccumulator species can accumulate more than 1000 ppm lead (Maestri et al. 2010). Indeed, these plants exude substances from roots that dissolve metals in soil (Arshad et al. 2008) that increases uptake and translocation (by employing certain metal cation transporters/genes). Moreover, they can tolerate higher concentrations of lead ions because they have various detoxification mechanisms, which may include selective metal uptake, excretion, complexation by specific ligands, and compartmentalization.

In addition, translocation of lead to aerial plant parts increases in the presence of organic chelators like ethylenediaminetetraacetate (EDTA) (Liu et al. 2008; Zaier et al. 2010; Barrutia et al. 2010) or certain species of micro-organisms (Arias et al. 2010; Punamiya et al. 2010). Recently, Liu et al. (2010) reported that, in 30 *B. pekinensis* cultivars, increased soil lead levels also increased the percent translocation to aerial plant parts. High concentrations of lead are known to destroy the physical barrier formed by the Caspary strip.

Transportation of metals from plant roots to shoots requires movement through the xylem (Verbruggen et al. 2009) and, when it occurs, is probably driven by transpiration (Liao et al. 2006). Arias et al. (2010) demonstrated high lead deposition in xylem and phloem cells of mesquite plants by using X-ray mapping. After penetrating into the central cylinder of the stem, lead can again be transported via the apoplastic pathway. The lead is then translocated to leaf areas via vascular flow (Sharma and Dubey 2005; Krzesłowska et al. 2010). While passing through the xylem, lead can form complexes with amino or organic acids (Roelfsema and Hedrich 2005; Vadas and Ahner 2009; Maestri et al. 2010). However, lead may also be transferred in inorganic form, as is cadmium. To express the degree of lead translocation, some authors have used a translocation factor (lead in aerial parts/lead in roots) (Arshad et al. 2008; Uzu et al. 2009; Liu et al. 2010). When this factor is used, the numeric value is normally rather low, which indicates that lead has been sequestered in the roots (Uzu et al. 2009; Liu et al. 2010).

4 General Effects of Lead on Plants

4.1 Effects on Germination and Growth

When plants are exposed to lead, even at micromolar levels, adverse effects on germination and growth can occur (Kopittke et al. 2007). Germination is strongly inhibited by very low concentrations of Pb²⁺ (Tomulescu et al. 2004; Islam et al. 2007). Lead-induced inhibition of seed germination has been reported in *Hordeum*

vulgare, *Elsholtzia argyi*, *Spartina alterniflora*, *Pinus halepensis*, *Oryza sativa*, and *Z. mays* (Tomulescu et al. 2004; Islam et al. 2007; Sengar et al. 2009). At higher concentrations, lead may speed up germination and simultaneously induce adverse affects on the length of radical and hypocotyl in *E. argyi* (Islam et al. 2007). Inhibition of germination may result from the interference of lead with protease and amylase enzymes (Sengar et al. 2009).

Lead exposure in plants also strongly limits the development and sprouting of seedlings (Dey et al. 2007; Gichner et al. 2008; Gopal and Rizvi 2008). At low concentrations, lead inhibits the growth of roots and aerial plant parts (Islam et al. 2007; Kopittke et al. 2007). This inhibition is stronger for the root, which may be correlated to its higher lead content (Liu et al. 2008). Lead toxicity may also cause swollen, bent, short and stubby roots that show an increased number of secondary roots per unit root length (Kopittke et al. 2007). Recently, Jiang and Liu (2010) reported mitochondrial swelling, loss of cristae, vacuolization of endoplasmic reticulum and dictyosomes, injured plasma membrane and deep colored nuclei, after 48–72 h of lead exposure to *A. sativum* roots. Arias et al. (2010) reported significantly inhibited root elongation in Mesquite (*Prosopis* sp.).

Plant biomass can also be restricted by high doses of lead exposure (Gopal and Rizvi 2008; Gichner et al. 2008; Islam et al. 2008; Piotrowska et al. 2009; Sing et al. 2010). Under severe lead toxicity stress, plants displayed obvious symptoms of growth inhibition, with fewer, smaller, and more brittle leaves having dark purplish abaxial surfaces (Islam et al. 2007; Gupta et al. 2009). Plant growth retardation from lead exposure may be attributed to nutrient metabolic disturbances (Kopittke et al. 2007; Gopal and Rizvi 2008) and disturbed photosynthesis (Islam et al. 2008). In most cases, the toxic effect of lead on plant growth is time and dose dependent (Dey et al. 2007; Gupta et al. 2009, 2010). However, the effect of low concentrations is not clearly established, and the observed growth inhibition is not necessarily correlated to a reduction in biomass (Kosobrukhov et al. 2004; Yan et al. 2010). Moreover, the effect of lead toxicity varies with plant species, i.e., hyperaccumulators naturally tolerate more lead toxicity than do sensitive plants (Arshad et al. 2008).

4.2 Effects on Proteins

Similar to what occurs with other heavy metals, lead interacts with cytoplasmic proteins. The effect of lead on the total concentration of protein is unclear, although high concentrations may decrease the protein pool (Chatterjee et al. 2004; Mishra et al. 2006; Garcia et al. 2006; Piotrowska et al. 2009). This quantitative decrease in total protein content is the result of several lead effects: acute oxidative stress of reactive oxygen species (ROS) (Piotrowska et al. 2009; Gupta et al. 2009), modification in gene expression (Kovalchuk et al. 2005), increased ribonuclease activity (Gopal and Rizvi 2008), protein utilization by plants for the purposes of lead detoxification (Gupta et al. 2009), and diminution of free amino acid content

(Gupta et al. 2009) that is correlated with a disturbance in nitrogen metabolism (Chatterjee et al. 2004). However, certain amino acids, like proline, increase under lead stress (Qureshi et al. 2007). Such proteins play a major role in the tolerance of the plant to lead. In contrast, low concentrations of lead increase total protein content (Mishra et al. 2006). This protein accumulation may defend the plant against lead stress (Gupta et al. 2010), particularly for proteins involved in cell redox maintenance. If true, then such proteins act in a way similar to how ascorbate functions or similar to how metals are sequestered by glutathione (GSH) or phytochelatins (PCs) (Brunet et al. 2009; Liu et al. 2009; Yadav 2010; Jiang and Liu 2010). In addition to a quantitative change, lead can affect the qualitative composition of cell proteins. The protein profile of root cells in bean seedlings was modified after lead exposure (Beltagi 2005). Such modification can be correlated to the change that occurs in the transcriptome profile of several enzymes including isocitrate lyase, cysteine proteininase *SAG12*, serine hydroxymethyltransferase, and arginine decarboxylase (Kovalchuk et al. 2005).

4.3 Water Status Effects

The disruption of plant water status after lead treatment has been addressed in many studies (Brunet et al. 2009). Results of such exposures show a decrease in transpiration, as well as reduction of the moisture content (Barcelo and Poschenrieder 1990; Patra et al. 2004). Reduced transpiration may result from reduced leaf surface area for transpiration that is caused by decreased leaf growth (Elibieta and Miroslawa 2005). However, some plant species that have high stomatal density are capable of coping with such effects (Kosobrukhov et al. 2004; Elibieta and Miroslawa 2005). Lead reduces plant cell wall plasticity, and thereby influences the cell turgor pressure. The decrease in concentrations of molecules that control cell turgor, such as sugars and amino acids, further accentuates the phenomenon of lead influence on turgor pressure (Barcelo and Poschenrieder 1990). The change in turgor pressure, particularly in the guard cells, interferes with stomatal opening and closing. To maintain cell turgor pressure, plants synthesize high concentrations of osmolytes, particularly proline under lead stress conditions (Qureshi et al. 2007).

Stomatal opening/closing is controlled by abscisic acid (ABA), a phytohormone (Roelfsema and Hedrich 2005). The presence of Pb²⁺ ions causes a large accumulation of ABA in roots and aerial plant parts (Parys et al. 1998; Atici et al. 2005; Cenkci et al. 2010), leading to stomatal closure (Mohan and Hosetti 1997). Stomatal closure strongly limits gas exchange with the atmosphere, and water losses by transpiration (Parys et al. 1998). According to Elibieta and Miroslawa (2005), the foliar respiration of plants is also reduced by lead exposure, because the deposition of a cuticle layer, for example, on *Glycine max* leaf surfaces, is affected. Moreover, a CO₂/O₂ imbalance in plants from lead-induced oxidative phosphorylation and respiratory disorders may also disrupt plant water status.

4.4 Mineral Nutrition Effects

Results from multiple studies demonstrate that nutrient uptake by plants is significantly affected by the presence of lead (Chatterjee et al. 2004; Sharma and Dubey 2005; Gopal and Rizvi 2008). Although data are insufficient to allow a definitive conclusion to be drawn, it is known that lead affects plant mineral uptake. It is also known that lead exposure decreases the concentration of divalent cations (Zn^{2+} , Mn^{2+} , Mg^{2+} , Ca^{2+} , and Fe^{2+}) in leaves of *Z. mays* (Seregin et al. 2004), *O. sativa* (Chatterjee et al. 2004), *Brassica oleracea* (Sinha et al. 2006), *Medicago sativa* (Lopez et al. 2007), *V. unguiculata* (Kopittke et al. 2007), and *Raphanus sativus* (Gopal and Rizvi 2008). But, it is not possible to conclude if this decrease results from blockage of root absorption, a decrease in translocation from roots to aerial plant parts, or a change in distribution of these elements in the plant. The effect of lead on mineral accumulation in aerial plant parts, in most cases, follows a common trend. In roots, the trend varies according to plant species or the intensity of the imposed stress (Lopez et al. 2007; Kopittke et al. 2007; Gopal and Rizvi 2008).

The decreased absorption of nutrient in the presence of lead may result from competition (e.g., those with atomic size similar to lead) or changes in physiological plant activities. According to Sharma and Dubey (2005), the strong interaction of K^+ ions with lead could result from their similar radii (Pb^{2+} : 1.29 Å and K^+ : 1.33 Å): these two ions may compete for entry into the plant through the same potassium channels. Similarly, lead effects on K^+ -ATPase and -SH groups of cell membrane proteins cause an efflux of K^+ from roots. However, lead does not cause nitrogen efflux. The general reduction in the concentration of inorganic nitrogen in all plant parts could be induced by the reduced activity of nitrate reductase, the rate-limiting enzyme in the nitrate assimilation process (Xiong et al. 2006; Sengar et al. 2009). Xiong et al. (2006) reported that lead exposure (4 and 8 mmol kg^{-1}) significantly decreased shoot nitrate content (70 and 80%), nitrate reductase activity (100 and 50%), and free amino acid content (81 and 82%) in *B. pekinensis*.

4.5 Photosynthesis Effects

Photosynthesis inhibition is a well-known symptom of lead toxicity (Xiong et al. 2006; Hu et al. 2007; Liu et al. 2008; Piotrowska et al. 2009; Sing et al. 2010; Cenkci et al. 2010). This inhibition is believed to result from the following indirect effects of lead rather than from a direct effect:

- distorted chloroplast ultrastructure from the affinity lead has for protein N and S ligands (Elibieta and Miroslawa 2005; Islam et al. 2007),
- decreased ferredoxin $NADP^+$ reductase and delta-aminolevulinic acid dehydratase (ALAD) activity at the origin of chlorophyll synthesis inhibition (Gupta et al. 2009; Cenkci et al. 2010),

- inhibition of plastoquinone and carotenoid synthesis (Kosobrukhan et al. 2004; Chen et al. 2007; Liu et al. 2008; Cenkci et al. 2010),
- obstruction of the electron transport system (Qufei et al. 2009),
- inadequate concentration of carbon dioxide via stomatal closure (Romanowska et al. 2002, 2005, 2006),
- impaired uptake of essential elements such as Mn and Fe (Chatterjee et al. 2004; Gopal and Rizvi 2008) and substitution of divalent cations by lead (Gupta et al. 2009; Cenkci et al. 2010),
- inhibition of Calvin cycle enzymatic catalysis (Mishra et al. 2006; Liu et al. 2008), and
- increased chlorophyllase activity (Liu et al. 2008).

However, these different effects vary by plant species. Generally, chlorophyll b is more sensitive than chlorophyll a (Xiong et al. 2006). The mechanism of chlorophyll breakdown into phytol, magnesium and the primary cleavage product of the porphyrin ring occur in four consecutive steps. This reaction is catalyzed by chlorophyllase, Mg-dechelatase, pheophorbide a oxygenase, and red chlorophyll catabolite reductase. Loss of the typical chlorophyll green color occurs only after cleavage of the porphyrin ring (Harpaz-Saad et al. 2007). The decrease observed in photosynthetic activity is often a more sensitive measure than is pigment content.

4.6 Respiration Effects

When exposed to lead, photosynthetic plants usually experience harmful effects on respiration and adenosine triphosphate (ATP) content. Unlike the photosynthetic activity, the effect of lead on respiratory activity has been little studied (Seregin and Ivanov 2001). All the studies carried out on respiratory activity deal with leaves, whereas the effect of the Pb^{2+} ions on the respiratory activity of roots remains unknown. Lead is reported to affect the activity of ribulose-bisphosphate carboxylase in C_3 plants that control CO_2 assimilation, without affecting oxygenase activity (Assche and Clijsters 1990). Therefore, it is quite possible that photosynthesis is significantly reduced without any effect on photorespiration being induced, thus increasing the relative ratio of photorespiration to photosynthesis. Parys et al. (1998) reported that the CO_2 concentration of *P. sativum* in leaves increased significantly after exposure to lead nitrate, most probably from the reduced photosynthetic and increased respiration activity. Romanowska et al. (2002) stressed that Pb^{2+} -induced increases in respiration resulted only from dark (mitochondrial) respiration, while photorespiration was unaffected. The stimulation of dark respiration by lead was observed in leaves or protoplasts of *P. sativum* and *H. vulgare* (Romanowska et al. 2002, 2005, 2006). Moreover, the stimulation of respiration was well correlated with increased production of ATP in mitochondria, resulting in the high energy demands of the plant to combat lead effects being met.

It has been also shown that divalent cations (e.g., Pb, Zn, Cd, Co, and Ni) can bind to mitochondrial membranes, disrupting the electron transport that could lead to decoupling of phosphorylation (Romanowska et al. 2002, 2006). An increase in the respiratory rate of 20–50% was observed by Romanowska et al. (2002) in the detached leaves of C₃ plants (*P. sativum* and *H. vulgare*) and C₄ plants (*Z. mays*), when they were exposed to 5 mM Pb(NO₃)₂ for 24 h. Glycine, succinate, and malate substrates were more fully oxidized in mitochondria, isolated from lead-treated *P. sativum* leaves, than in mitochondria from control leaves (Romanowska et al. 2002). Lead caused an increase in ATP content as well as an increase in the ATP/ADP ratio in *P. sativum* and *H. vulgare* leaves (Romanowska et al. 2005, 2006). Rapid fractionation of *H. vulgare* protoplasts, incubated under conditions of low and high CO₂, indicated that the increased ATP/ADP ratio in lead-treated leaves mainly resulted from the production of mitochondrial ATP. The activity of NAD⁺-malate dehydrogenase in protoplasts of barley leaves treated with lead was threefold higher than that in protoplasts from control leaves (Romanowska et al. 2005). Lead also significantly inhibited Hill reaction activity in spinach chloroplasts, in addition to photophosphorylation; moreover, lead had a more conspicuous effect on cyclic photophosphorylation than on noncyclic photophosphorylation (Romanowska et al. 2008). Recently, Qufei and Fashui (2009) reported that the accumulation of Pb²⁺ in photosystem II resulted in damage to its secondary structure and induced decreased absorbance of visible light and inhibited energy transfer among amino acids. Moreover, Jiang and Liu (2010) reported mitochondrial swelling, loss of cristae, and vacuolization of endoplasmic reticulum and dictyosomes during a 48–72 h lead exposure in *A. sativum*.

4.7 Genotoxicity

The antimitotic effect of lead is one of its best known toxic effects on plants (Patra et al. 2004; Shahid et al. 2011). Indeed, Hammett (1928) demonstrated long ago that lead induces a dose-dependent decrease in mitotic activity in root cells of *Allium cepa*, which was later described in detail by Wierzbicka (1999) and Patra et al. (2004). In *V. faba* roots, lead shortened the mitotic stage and prolonged interphase, thus lengthening the cell cycle (Patra et al. 2004). The first step by which lead induces plant toxicity is the binding of the Pb²⁺ ion to cell membranes and to the cell wall. This produces rigidity in these components and reduces cell division. The second step is the disruption of microtubules that are essential for mitosis. Lead exposure induces disturbances in the G₂ and M stages of cell division that leads to the production of abnormal cells at the c-mitosis (colchicine-mitosis) stage. This phenomenon is thought to be accentuated by direct or indirect interactions of lead with the proteins involved in the cell cycle, such as cyclins. Cyclin activity is indirectly dependent on the concentration of GSH. The spindle activity disturbances caused by lead may be transient in some cases, returning the mitotic index to initial levels.

Unlike antimitotic mechanisms, the mechanisms by which lead causes genotoxicity are complex and not yet well understood. At a low concentration, lead did not induce a significant effect on mitosis, but did induce aberrations (chromosomal bridges during anaphase), loss of acentric fragments during meiosis, chromosomal fragmentation, and micronucleus formation (National Toxicology Program 2003; Patra et al. 2004; Cecchi et al. 2008; Marcato et al. 2009; Grover et al. 2010; Barbosa et al. 2010; Shahid et al. 2011). The induction of chromosomal aberrations by lead can be explained, in part, by its action of disrupting the microtubule network. Results of in vitro studies have demonstrated that lead creates breaks in single and double strands of DNA and thereby affects horizontal DNA–DNA or DNA–protein links (Rucińska et al. 2004; Gichner et al. 2008; Shahid et al. 2011).

Lead may enter the nucleus (Małecka et al. 2008) and bind directly to the DNA or indirectly to protein. After binding to DNA, lead disrupts DNA repair and replication mechanisms. Lead does not induce direct genotoxic effects until it becomes attached to naked DNA (Valverde et al. 2001). Lead can also affect replication by replacing the zinc in the Zn-finger pattern of the enzymes that intervene in DNA repair (Gastaldo et al. 2007). Recently, Cenksı et al. (2010) used a random amplified polymorphic DNA (RAPD) assay that amplifies random DNA fragments of genomic DNA, and they reported that genomic template stability was significantly affected by lead exposure in *Brassica rapa*.

4.8 Oxidative Stress and Lipid Peroxidation

ROS are produced during normal cell metabolism in the chloroplast, either as by-products of the reduction of molecular oxygen (O_2) or because of excitation in the presence of highly energized pigments. These ROS, such as superoxide radicals ($O_2^{\bullet-}$), hydroxyl radicals ($\bullet OH$), and hydrogen peroxide (H_2O_2), are also generated following exposure to certain environmental agents. The production of ROS in the cells of aerobic organisms, defined as oxidative stress, is a well-known feature of the toxicity of heavy metals, including lead (Pourrut et al. 2008; Liu et al. 2008; Grover et al. 2010; Yadav 2010; Singh et al. 2010). However, the degree to which this feature is important is dependent on the metal type, specific form of the metal, plant species, exposure time, etc. When ROS forms exhaust cellular antioxidant reserves, they can rapidly attack and oxidize all types of biomolecules, such as nucleic acids, proteins, and lipids (Reddy et al. 2005; Clemens 2006; Hu et al. 2007; Wang et al. 2007; Yadav 2010). Such attacks lead to irreparable metabolic dysfunction and cell death.

Lead causes marked changes in the lipid composition of different cell membranes (Liu et al. 2008; Piotrowska et al. 2009; Grover et al. 2010; Yan et al. 2010; Singh et al. 2010). The polyunsaturated fatty acids and their esters that are present in lipids show high susceptibility to ROS (Dey et al. 2007; Gupta et al. 2009). Indeed, ROS removes hydrogen from unsaturated fatty acids and forms lipid

radicals and reactive aldehydes, ultimately causing distortion of the lipid bilayer (Mishra et al. 2006). Lead-induced changes in lipid composition and potassium ion leakage were reported in *Z. mays* (Malkowski et al. 2002). Lead ions are known to induce lipid peroxidation, decrease the level of saturated fatty acids, and increase the unsaturated fatty acid content of membranes in several plant species (Singh et al. 2010).

The oxidation of bis-allylic hydrogens on polyunsaturated fatty acids by ROS involves three distinct stages: initiation (formation of the lipid radical), progression (formation of lipid peroxy radical by reaction between lipid radical and oxygen), and termination (formation of non-radical products after bimolecular interaction of lipid peroxy radicals) (see details in the reviews of Gurer and Ercal 2000 and Bhattacharjee 2005). These lipid membrane changes cause the formation of abnormal cellular structures, such as alterations in the cell membrane (Dey et al. 2007; Islam et al. 2008; Gupta et al. 2009), organelles (e.g., mitochondria), peroxisomes (Małecka et al. 2008; Liu et al. 2008), or chloroplasts (Choudhury and Panda 2004; Elibieta and Miroslawa 2005; Hu et al. 2007).

5 Mechanisms of Lead Tolerance

Plants respond to noxious effects of lead in various ways, such as selective metal uptake, metal binding to the root surface, binding to the cell wall, and induction of antioxidants. There are several types of antioxidants to which plants may respond: non-protein thiol (NP-SH), cysteine, glutathione, ascorbic acid, proline, and antioxidant enzymes, such as superoxide dismutase (SOD), ascorbate peroxidase (APX), guaiacol peroxidase (GPX), catalase (CAT), and glutathione reductase (GR). However, the response varies with plant species, metal concentration, and exposure conditions.

5.1 Passive Mechanisms

Even when small amounts of lead penetrate root cell membranes, it interacts with cellular components and increases the thickness of cell walls (Krzesłowska et al. 2009, 2010). Pectin is a component of plant cell walls. Lead complexation with pectin carboxyl groups is regarded as the most important interaction by which plant cells can resist lead toxicity (Meyers et al. 2008; Jiang and Liu 2010). Krzesłowska et al. (2009) observed that binding of lead to JIM5-P (within the cell wall and its resultant thickening) acted as a physical barrier that restricted lead access to the plasma membrane in *F. hygrometrica* protonemata. However, later, these authors stated that lead bound to JIM5-P within the cell can be taken up or remobilized by endocytosis, together with this pectin epitope (Krzesłowska et al. 2010).

5.2 Inducible Mechanisms

Recently, several authors have reported the presence of transporter proteins among plant cells that play an important role in metal detoxification, by allowing the excretion of metal ions into extracellular spaces (Meyers et al. 2008; Vadas and Ahner 2009; Maestri et al. 2010). The human divalent metal transporter 1 (DMT1), expressed in yeast, has been shown to transport lead via a pH-dependent process (Bressler et al. 2004) in plants. Simultaneously, several ATP-binding cassette (ABC) carriers, such as AtATM3 or AtADPR12 at ATP-binding sites in *Arabidopsis*, were involved in resistance to lead (Kim et al. 2006; Cao et al. 2008). Although, suspected to act against lead, this detoxification mechanism has not yet been clearly confirmed. Transcriptome analysis has shown that the gene expression of these carriers is stimulated by lead (Liu et al. 2009).

Cellular sequestration is considered to be an important aspect of plant metal homeostasis and plant detoxification of heavy metals (Maestri et al. 2010). The lead, that could be bound by certain organic molecules (Piechalak et al. 2002; Vadas and Ahner 2009), is sequestered in several plant cell compartments: vacuoles (Malecka et al. 2008; Meyers et al. 2008), dictyosome vesicles (Malone et al. 1974), endoplasmic reticulum vesicles (Wierzbicka et al. 2007), or plasmabubules (Wierzbicka 1998).

Cysteine and glutathione (GSH) are known to be non-enzymatic antioxidants in plants. An increase in cysteine content, in response to lead toxicity, has been demonstrated in *Arabidopsis thaliana* (Liu et al. 2009). Glutathione protects plants from lead stress by quenching lead-induced ROS (Verbruggen et al. 2009; Liu et al. 2009). Moreover, as the substrate for phytochelatin (PC) biosynthesis, the glutathione-related proteins play an important role in heavy metal detoxification and homeostasis (Liu et al. 2009). Lead treatment can induce different GSH genes, including glutathione-synthetase, -peroxidase, and -reductase, and glutamylcysteine synthetase. Glutathione can also enhance accumulation of proline in stressed plants, a role that is associated with reducing damage to membranes and proteins (Liu et al. 2009). Gupta et al. (2010) reported the role of GSH in lead detoxification in *S. alfredii*, although this was accomplished without any induction of PC. This suggests that GSH may play an important role in detoxifying lead, under stress conditions where PCs are absent.

PCs and metallothioneins (MTs) are the best characterized metal-binding ligands in plant cells. These ligands belong to different classes of cysteine-rich heavy metal-binding protein molecules. PCs, the most frequently cited metal protective proteins in plants, are low molecular weight, metal-binding proteins that can form mercaptide bonds with various metals (Maestri et al. 2010) and play an important role in their detoxification in plants (Brunet et al. 2009; Liu et al. 2009; Gupta et al. 2010; Yadav 2010; Jiang and Liu 2010). These thiols are biologically active compounds, whose function is to prevent oxidative stress in plant cells (Verbruggen et al. 2009; Gupta et al. 2010). Their general structure is $(\gamma\text{-glutamyl-cys})_n$ Gly where $n = 2\text{--}11$, and they are synthesized by the action of γ -glutamylcysteine dipeptidyl transpeptidase (phytochelatin synthase; PCS) from GSH (Yadav 2010).

Lead is known to stimulate the production of PC and activate PCS (Mishra et al. 2006; Clemens 2006; Andra et al. 2009; Vadas and Ahner 2009; Sing et al. 2010). It has been proposed that in vivo, phytochelatins are involved in the cellular detoxification and accumulation of several metals, including lead, because of their ability to form stable metal–PC complexes (Clemens 2006; Yadav 2010). Phytochelatin sequesters soluble lead in the cytoplasm before transporting it to vacuoles and chloroplasts (Piechalak et al. 2002; Małecka et al. 2008; Jiang and Liu 2010), thus reducing the deleterious effect of Pb²⁺ in the cells. The mechanism regulating the passage of the lead–PC complex through the tonoplast is, however, not yet known. Gisbert et al. (2003) reported significantly increased uptake and tolerance to lead and Cd following the induction and over-expression of a wheat gene encoding for phytochelatin synthase (*TaPCS1*), in *Nicotiana glauca*.

5.3 Antioxidant Enzymes

To cope with the increased production of ROS and to avoid oxidative damage, plants have a system of antioxidant enzymes that scavenge the ROS that are present in different cell compartments (Brunet et al. 2009; Singh et al. 2010; Gupta et al. 2010). Lead-induced toxicity may inhibit the activity of these enzymes or may induce their synthesis (Table 1). However, lead-induced inhibition or induction of antioxidant enzymes is dependent on metal type, specific form of the metal, plant species type, and the duration/intensity of the treatment (Islam et al. 2008; Gupta et al. 2009; Singh et al. 2010).

Generally, lead inhibits enzymatic activities and, when this occurs, the values of the inactivation constant (K_i) range between 10^{-5} and 2×10^{-4} M (i.e., 50% of enzymatic activities are inhibited in this concentration range) (Seregin and Ivanov 2001). Enzyme inhibition results from the affinity lead has for -SH groups on the enzyme (Sharma and Dubey 2005; Gupta et al. 2009). This is true for more than 100 enzymes, including ribulose-1,5-bisphosphate carboxylase oxygenase (RuBisCO) and nitrate reductase. Inactivation results from a link at either the catalytic site or elsewhere on the protein and produces an altered tertiary structure. Lead can also produce the same effect by binding to protein-COOH groups (Gupta et al. 2009, 2010). Lead also interacts with metalloid enzymes. Indeed, lead can disrupt plant absorption of minerals that contain zinc, iron, manganese, etc., which are essential for these enzymes. Lead and other divalent cations also can substitute for these metals, and thereby inactivate enzymes, as occurs with ALAD (Gupta et al. 2009; Cenkci et al. 2010). The effect lead has on ROS constitutes another mechanism by which lead exposure affects protein behavior (Gupta et al. 2009, 2010).

Lead exposure is also known to stimulate the activities of certain enzymes (Table 1), but the mechanisms of action are, as yet, unclear. It has been proposed that lead activates certain enzymes by modulating gene expression or by restricting the activity of enzyme inhibitors (Seregin and Ivanov 2001). Indeed, antioxidant enzymes scavenge ROS, when they are produced in excess as a consequence of

Table 1 Lead-induced activation (\uparrow) and reduction (\downarrow) of enzymatic activities in different plant species

Plant species	\uparrow Enzyme activity	\downarrow Enzyme activity	References
<i>Najas indica</i>	SOD, GPX, APX, CAT, GR		Sing et al. (2010)
<i>Sedum alfredii</i>	SOD, APX		Gupta et al. (2010)
<i>Zea mays</i>	SOD, CAT, AsA		Gupta et al. (2009)
<i>Lathyrus sativus</i>	APX, GR, GST		Brunet et al. (2009)
<i>Wolffia arrhiza</i>	CAT, APX		Piotrowska et al. (2009)
<i>Raphanus sativus</i>	POD, ribonuclease	CAT	Gopal and Rizvi (2008)
<i>Elsholtzia argyi</i>	CAT	SOD, GPX	Islam et al. (2008)
<i>Kandelia candel</i>	SOD, POD, CAT		Zhang et al. (2007)
<i>Bruguiera gymnorhiza</i>	SOD, POD, CAT		Zhang et al. (2007)
<i>Cassia angustifolia</i>	SOD, APX, GR, CAT		Qureshi et al. (2007)
<i>Zea mays</i>	SOD, POD, CAT		Wang et al. (2007)
<i>Triticum aestivum</i>	SOD, POX	CAT	Dey et al. (2007)
<i>Potamogeton crispus</i>	POD, SOD, CAT	SOD, CAT	Hu et al. (2007)
<i>Ceratophyllum demersum</i>	SOD, GPX, APX, CAT, GR	SOD, GPX, APX, CAT, GR	Mishra et al. (2006)
<i>Helianthus annuus</i>	GR	CAT	Garcia et al. (2006)
<i>Macroteloma uniflorum</i>	SOD, CAT, POD, GR, GST		Reddy et al. (2005)
<i>Cicer arietinum</i>	SOD, CAT, POD, GR, GST		Reddy et al. (2005)
<i>Taxithelium nepalense</i>	APX, GPOX, CAT,		Choudhury and Panda (2004)
<i>Oryza sativa</i>	SOD, GPX, APX, CAT, GR	GR, CAT	Verma and Dubey (2003)

SOD, superoxide dismutase; APX, ascorbate peroxidase; GPX, guaiacol peroxidase; CAT, catalase; GR, glutathione reductase; AsA, ascorbic acid; GST, GSH S-transferase; GSH, glutathione; POD, peroxidase

metal toxicity. Superoxide dismutase, a metallo-enzyme present in various cell compartments, is considered to be the first defense against oxidative stress (Mishra et al. 2006). It catalyses the dismutation of two superoxide radicals to H_2O_2 and oxygen and thus maintains superoxide radicals at steady-state levels (Islam et al. 2008; Gupta et al. 2009). H_2O_2 is a very strong oxidant and requires quick removal to avoid oxidative toxicity; removal is achieved by the action of APX in the ascorbate-glutathione cycle or by GPX and CAT in the cytoplasm and in other cell compartments (Mishra et al. 2006). The role of GSH and glutathione reductase in the H_2O_2 -scavenging mechanism in plant cells (Piechalak et al. 2002) is well established in the Halliwell–Asada enzyme pathway. Moreover, antioxidant enzymes may be activated from the increased concentration of their substrates, instead of direct interaction with lead (Islam et al. 2008).

6 Conclusions and Perspectives

Lead is a major inorganic global pollutant and numerous studies have revealed its biogeochemical behavior and impact on the biosphere. Based on these studies, as cited in this review, it is concluded as follows:

- (1) Lead has been in use since antiquity, because of its many useful properties. The continued use of lead in many industrial processes has increased its concentration to toxic levels in all environmental compartments.
- (2) Lead forms stable complexes with different compounds in soil and tends to be stored in the soil. The fate and behavior of lead in soil is affected by its form, solubility, mobility, and bioavailability and is controlled by many biogeochemical parameters, such as soil pH, redox conditions, cation-exchange capacity, soil mineralogy, biological and microbial conditions, amount and nature of organic and inorganic ligands present, and competing cations.
- (3) Lead enters plants mainly through the roots via the apoplast pathway or calcium ion channels. Lead can also enter plants in small amounts through leaves. Once in the roots, lead tends to sequester in root cells. Only a limited amount of lead is translocated from roots to shoot tissues, because there are natural plant barriers in the root endodermis (e.g., Caspary strips).
- (4) Lead has no biological function and induces various noxious effects inside plants. Excessive lead accumulation in plant tissue is toxic to most plants, leading to a decrease in seed germination, root elongation, decreased biomass, inhibition of chlorophyll biosynthesis, mineral nutrition and enzymatic reactions, as well as a number of other physiological effects. The intensity of these effects varies depending on the duration of exposure, stage of plant development, studied organ, and the concentration of lead used in the exposure assessment.
- (5) Lead-induced production of ROS is the major cause of its toxicity. These free radicals disrupt the redox status of cells, causing oxidative stress and DNA damage through oxidation, and lead to irreparable metabolic dysfunction and cell death.
- (6) Plants defend against lead toxicity through several avoidance or detoxification mechanisms. Plants resist lead entry into their cells via exclusion or they bind lead to their cell walls or other ligands. Plants combat lead-induced increased production of ROS by activating various antioxidant enzymes.
- (7) The efficiency of detoxification mechanisms determines the final tolerance or sensitivity of plants to metal-induced stress. Plants that have efficient detoxification mechanisms are generally characterized as being hyperaccumulators. Such plants are useful in soil bioremediation for many metal types. Conversely, plants that do not efficiently cope with pollutants are sensitive to metal toxicity and are often used in risk assessment studies.

In this review, we raise several questions that need attention if our understanding of the biogeochemical behavior of lead in different environmental compartments is

to be advanced. Lead is known to interfere directly or indirectly with the genetic material to induce ROS and modify (increase or decrease) the activities of certain enzymes in plants. These responses of plants to lead toxicity are often used as tools for risk assessment. However, the mechanisms of action underlying the noxious effects of lead in plants are still unknown.

Moreover, most field work performed on the effects of lead on plants is based almost exclusively on the total metal content in polluted soil, even though this is of little significance from an environmental point of view. Indeed, the potential effects of lead and other toxic elements in the environment depend on insights into their physico-chemical distribution, i.e., speciation. Therefore, if environmental scientists are to become better at predicting what the toxicity or environmental impact of lead may be, then additional research into the form of lead present is essential.

7 Summary

Lead has gained considerable attention as a persistent toxic pollutant of concern, partly because it has been prominent in the debate concerning the growing anthropogenic pressure on the environment. The purpose of this review is to describe how plants take lead up and to link such uptake to the ecotoxicity of lead in plants. Moreover, we address the mechanisms by which plants or plant systems detoxify lead.

Lead has many interesting physico-chemical properties that make it a very useful heavy metal. Indeed, lead has been used by people since the dawn of civilization. Industrialization, urbanization, mining, and many other anthropogenic activities have resulted in the redistribution of lead from the earth's crust to the soil and to the environment.

Lead forms various complexes with soil components, and only a small fraction of the lead present as these complexes in the soil solution are phytoavailable. Despite its lack of essential function in plants, lead is absorbed by them mainly through the roots from soil solution and thereby may enter the food chain. The absorption of lead by roots occurs via the apoplastic pathway or via Ca^{2+} -permeable channels. The behavior of lead in soil, and uptake by plants, is controlled by its speciation and by the soil pH, soil particle size, cation-exchange capacity, root surface area, root exudation, and degree of mycorrhizal transpiration. After uptake, lead primarily accumulates in root cells, because of the blockage by Casparyan strips within the endodermis. Lead is also trapped by the negative charges that exist on roots' cell walls.

Excessive lead accumulation in plant tissue impairs various morphological, physiological, and biochemical functions in plants, either directly or indirectly, and induces a range of deleterious effects. It causes phytotoxicity by changing cell membrane permeability, by reacting with active groups of different enzymes involved in plant metabolism and by reacting with the phosphate groups of ADP or ATP, and by replacing essential ions. Lead toxicity causes inhibition of ATP production,

lipid peroxidation, and DNA damage by over production of ROS. In addition, lead strongly inhibits seed germination, root elongation, seedling development, plant growth, transpiration, chlorophyll production, and water and protein content. The negative effects that lead has on plant vegetative growth mainly result from the following factors: distortion of chloroplast ultrastructure, obstructed electron transport, inhibition of Calvin cycle enzymes, impaired uptake of essential elements, such as Mg and Fe, and induced deficiency of CO₂ resulting from stomatal closure.

Under lead stress, plants possess several defense strategies to cope with lead toxicity. Such strategies include reduced uptake into the cell; sequestration of lead into vacuoles by the formation of complexes; binding of lead by phytochelatins, glutathione, and amino acids; and synthesis of osmolytes. In addition, activation of various antioxidants to combat increased production of lead-induced ROS constitutes a secondary defense system.

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