Preface

Heavy metals (HM) are conventional elements with properties like ductility, conductivity, stability as cations, ligand specificity, etc., and an atomic number >20. HM such as Cu, Zn, Mn, Fe, Ni, and Co are essential micronutrients for plant metabolism but when present in excess, these, as well as low levels of non-essential HM such as Cd, Hg, and Pb, can become extremely toxic.

Tolerance to HM in plants may be defined as the ability to survive in a soil/water that is toxic to other plants and is manifested by an interaction between a genotype and its environment (Macnair et al. 2000). To protect themselves from metal poisoning, plants must develop mechanisms by which the HM entering the cytosol are either immediately excluded or complexed and neutralized, thus preventing the metal from inactivating catalytically active or structural proteins, presumably by adopting mechanisms that may also be involved in the general homeostasis of essential mineral ions and to tolerate them. The strong effect of HM on oxidative processes is the base for other connections with signaling response and genetic diversity. Plant tolerance to HM depends largely on plant efficiency in uptake, translocation, and further sequestration of HM in specialized tissues or in trichomes and cell organelles (Gupta and Sandalio 2012). Metals which are complexed and sequestered in cellular structures become unavailable for translocation to the shoot (Lasat et al. 1998). HM binding to the cell wall is not the only plant mechanism responsible for metal immobilization into roots and subsequent inhibition of ion translocation to the shoot. The vacuole is generally considered to be the main storage site for metals in yeast and plant cells and there is evidence that phytochelatin-metal complexes are pumped into the vacuole in plants (Yang et al. 2005a).

Complexation with ligands is a process associated to HM pollutants, and it can be an extracellular or an intracellular molecular event. These ligands can be chelators as organic acids or peptides such phytochelatins (PCs), methallothioneins (MTs), or glutathione (GSH) (Mello-Farias and Chaves 2008). PCs are a class of nuclear encoded cysteine-rich peptides that play a pivotal role in HM tolerance in plants and fungi by chelating these substances thus decreasing their free concentrations (Vatamaniuk et al. 1999).

Transport of metals and alkali cations across plasma membrane and organelle membranes is essential for plant growth, development, signal transduction, and

toxic metal phytoremediation (Cherian and Oliveira 2005). Although there is no direct evidence for a role for plasma membrane efflux transporters in HM tolerance in plants, recent research has revealed that plants possess several classes of metal transporters that must be involved in metal uptake and homeostasis in general and, thus, could play a key role in tolerance (Yang et al. 2005a). Several classes of proteins have been implicated in HM transport in plants. These include the HM (or CPx-type) ATPases that are involved in the overall metal-ion homeostasis and tolerance in plants, the natural resistance-associated macrophage protein (Nramp) family of proteins, the cation diffusion facilitator (CDF) family proteins (Williams et al. 2000), and the zinc–iron permease (ZIP) family proteins, etc. (Yang et al. 2005a, b).

One of the major consequences of HM action in the cell is the enhanced generation of reactive oxygen species (ROS) which usually damage the cellular components such as membranes, nucleic acids, chloroplast pigments, and alteration in enzymatic and non-enzymatic antioxidants. Complementary, a new family of molecules designated a reactive nitrogen species (RNS) starts to be new elements involved in the mechanism of response against HM where molecules such as nitric oxide (NO), peroxynitrite (ONOO–), and *S*-nitrosoglutathione (GSNO) can mediate protein function by specific post-translational modifications (Leterrier et al. 2012).

It is an intriguing question whether the toxicity effect induced by HM was the result (at least partially) of signaling pathways evolving the action of the formed substances, or parallel direct HM action and signaling pathways. The molecular mechanisms of signal transduction pathways in higher plant cells are essential to vital processes such as hormone and light perception, growth, development, stress resistance, and nutrient uptake from soil and water. HM interfere with cell signaling pathways. In fact, it might be hypothesized that HM-induced deregulation of signaling events significantly participates in the HM toxicity response, as well as in damage development.

It is always like an adventure for scientists all over the world to work with HM and plants. The main purpose of the book is to present comprehensive and concise knowledge of the recent advancement in the field of metal transport and how the genetic diversity affects the HM transport in plants. Other key futures of the book are related to metal toxicity and detoxification mechanism, biochemical tools toward HM remediation processes, molecular mechanism for HM detoxification, how metallomics and metalloproteomics are affected by HM stress in plants, and role of ROS metabolism in alleviation of HM. Some chapters are focusing on recent development in the field of phytoremediation. Overall the information compiled in this book will bring very depth knowledge and advancement in the field of HM toxicity in plants in recent years.

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Granada, Spain

Dr. Dharmendra K. Gupta
Dr. Francisco J. Corpas
Dr. José M. Palma

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