



REVIEW ARTICLE

Cytotoxic and genotoxic alterations as an important biomarker for cadmium and mercury induced stress in plants- A review

Monalisa Mohanty^{1*}, Pragyanshree Maharana¹ & Sonali Nayak²

¹Department of Biotechnology, Laboratory of Plant and Environmental Engineering, Rama Devi Women's University, Bhubaneswar 751 022, Odisha, India

²Department of Life Sciences, Rama Devi Women's University, Bhubaneswar 751 022, Odisha, India

*Email: monalisamohanty@rdwu.ac.in

OPEN ACCESS

ARTICLE HISTORY

Received: 22 July 2025
Accepted: 03 November 2025
Available online
Version 1.0 : 17 February 2026

Additional information

Peer review: Publisher thanks Sectional Editor and the other anonymous reviewers for their contribution to the peer review of this work.

Reprints & permissions information is available at https://horizonpublishing.com/journals/index.php/TCB/open_access_policy

Publisher's Note: The article processing was done by atom e-Publishing, Thiruvananthapuram, India. Horizon e-Publishing Group remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Copyright: © The Author(s). This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution and reproduction in any medium, provided the original author and source are credited (<https://creativecommons.org/licenses/by/4.0/>)

CITE THIS ARTICLE

Monalisa M, Pragyanshree M, Sonali N. Cytotoxic and genotoxic alterations as an important biomarker for cadmium and mercury induced stress in plants- A review. Trends in Current Biology. 2025; 4(1): 42-51. <https://doi.org/10.14719/tcb.9436>

Abstract

Cadmium (Cd) and Mercury (Hg) as toxic metallic mutants can cause various genetic and chromosomal disturbances in plants. Their overproduction due to various domestic and industrial demands leads to excess release to the environment and consequently pose detrimental genotoxic effects on plants and other life forms. They cause phytotoxic problems by directly or indirectly impeding plant growth because of their damage to DNA or cellular components of cell cycle viz. spindle apparatus, DNA polymerases and DNA repair systems. They impose detrimental effects on the genome and cause overproduction of Reactive Oxygen Species (ROS), physiological and/or biochemical irregularities such as decreased membrane stability and photosynthesis, suppression or inhibition of cell division and gene expression. These disruptions lead to impaired growth, delayed development and in long run it results into irreversible cellular damage, programmed cell death. Such conditions ultimately impact overall organismal productivity and survival. Due to their mutagenic or genotoxic effects on plants and other organisms, these are referred to as genotoxins. Genotoxic substances cause DNA damage, which can either be repaired or result in DNA changes. This review is a comprehensive survey on their sources, occurrence, industrial use, exposure impacts and various phytotoxic, cytotoxic and genotoxic effects on plants.

Keywords

cadmium; cytotoxic effects; exposure impacts; genotoxic effects; mercury; phytotoxicity

Introduction

Plants are subject to a variety of environmental stresses (biotic and abiotic stress) like heavy metal stress, acidity, salinity, radiations (UV-A, UV-B, UV-C), etc which obstruct the growth of the plant either directly or indirectly through inducing oxidative stress and overproduction of ROS (1).

Heavy metal elements are discharged into the environment as a result of increased anthropogenic activity, urbanization and rapid industrialization (2,3). Heavy metals such as Cd and Hg pose significant risk to plant health. Even at low concentration they can lead to detrimental effects on plant growth, metabolism and overall yield. Cadmium (Cd) in soil is commonly present at an average concentration of 0.1 mg kg⁻¹, whereas Hg in various types of soils from all over the world range between 0.58 and 1.8 mg kg⁻¹ and the worldwide mean content is estimated at 1.1 mg kg⁻¹ (4-7). When soil Cd concentration exceeds 8 mg kg⁻¹ or when bioavailability of Cd surpass 0.001 mg kg⁻¹ toxic symptoms appeared in the plants (8). For many plant species like Lettuce and Raphanus, Cd concentration of about 5-20 mg kg⁻¹ often produces visible stresses such as reduction in root, shoot length, chlorophyll and carotenoid content as well as reduction in fresh and dry weight (9,10). The recent

experiments on *Nigella sativa* exposed to Cd stress revealed pronounced alteration in ultrastructure of stomatal apparatus which results into reduction in stomatal density and closure of stomatal aperture. Furthermore, cd stress induced noticeable changes in plant's morphology and significantly decrease seed and fruit production. In addition, Cd accumulation in seed exceed WHO's permissible limit 0.3 mg kg⁻¹ (11). Similar to Cd, exposure of wheat plants to 10 and 30 mg L⁻¹ of Hg caused significant decline in growth parameters (12).

Cd is a hazardous heavy metal with a lengthy biological half-life (13). Because of its great mobility and solubility, it enters the food chain directly. It is capable of inducing genotoxicity in plants by binding to the functional sites of DNA. It damages DNA by causing breaks in single and double strands, changed bases, DNA-protein crosslinks, oxidised bases and bulky adducts (13).

Hg is one of the most dangerous and carcinogenic heavy metals found in the environment. It reacts with other elements to generate inorganic and organic mercury (14). It is capable to induce oxidative stress in plants which may lead to overproduction of ROS, ultimately leading to genotoxicity. Depending upon the concentration and types of tissue, Hg induced stress can affect both growth and developmental processes of plants like photosynthesis, nucleic acid production and lipid metabolism (15).

Primary genotoxicity occurs as a result of direct reactivity of genotoxins (like heavy metals) with DNA whereas secondary genotoxicity can occur due to the formation of ROS as reported by researchers (1). Heavy metals like Cd and Hg have genotoxic effect on plants through oxidative pathways that involves free radicals (16). Oxidative stress is produced as a result of increased ROS production under metal stress situations. These ROS can oxidise many macromolecules such as lipids, proteins, DNA and RNA which results in cellular dysfunctions (1,16,17). Heavy metal induced oxidative stress in plants causes mutations such as point mutation or deleted mutation or can cause DNA damage which may lead to the amplification of new bands of DNA, disappearance of normal bands and DNA strand breaks (15).

Concepts of heavy metals

Heavy metals are naturally occurring metallic elements (18). which have high atomic weight and atomic number probably greater than 20 (19). They have high densities that are at least 5 times higher than water (18). It accumulates in the environment and have toxic effects that serves as a threat to various lifeforms (19). Heavy metals have an impact on cellular components such as the cell membrane, mitochondria, lysosome and numerous enzymes involved in metabolism, as well as the damage and repair mechanisms. These metal ions interact with the cellular components causing damage and brings about change in the structures (18). Low concentration of these metals is beneficial to plants but at high concentration they may induce phytotoxicity, morphological toxicity, cytotoxicity, genotoxicity in plants. The Cd and Hg induce toxicity in plants are discussed in this review work.

Cadmium- properties, exposure limit and occurrence

Cd is a transition metal element that belongs to the group-12 of the d-block elements in the periodic table. It is found in nature in the form of mineral and is obtained from Cd ore, called Greenockite (Fig. 1, Table 1) (20,21).



Fig. 1. Greenockite ore.

Table 1. Physicochemical properties of cadmium

Physicochemical Properties	Analytical Data
Atomic number	48
Atomic mass	112.4 g/mol
Colour	Silvery White
State at room temperature	Solid
Atomic radii	1.71 Å
Ionic radii	0.95Å
Isotopes	15
Common Oxidation States	+2
Electronic configuration	[Kr] 4d105s2
Vander Waals radii	0.154nm
Electronegativity	1.69
Density	8.6 g/cm³
Melting Point	321 °C
Boiling Point	765 °C
First ionization energy	866 kJ/mol
Enthalpy of fusion	6.192 kJ/mol
Latent heat of vapourisation	99.570 kJ/mol

Cd is naturally contained in the earth's crust at a concentration of roughly 0.1 mg kg⁻¹(18). It is found in the form of ores combined with zinc, lead and copper. It is mainly extracted as a by-product during refining of zinc (22).

China, Japan and Korea are the world's largest producer of Cd. Netherlands, India, USA, UK, Peru and Germany along with fifteen other countries produces Cd in smaller amounts (Fig. 2) (23).



Fig. 2. Distribution of cadmium reserves in the world.

The concentration and exposure limit for Cd in plants is 0.02 mg kg⁻¹ as recommended by Food and Agricultural Organisation (FAO) and WHO (Table 2) (24–31).

Mercury- properties, exposure limit and occurrence

Hg is a transition metal element in the periodic table that belongs to group-12 of the d-block elements. It is a heavy, white and silvery liquid metal (Table 3) (32). It is recovered from cinnabar ores by heating and then the vapour is condensed and collected (Fig. 3) (33).

Hg has a worldwide production of around 8000 tonnes per year and the mineable reserves are around 600000 tonnes. Hg is ranked third among the most toxic elements or substances on the planet by the US Government Agency for Toxic Substances and Disease Registry (34). The average concentration of Hg in the earth's crust is 0.5 mg kg⁻¹ both in its free state as well as in the form of ores such as cinnabar, corderoite and livingstonite (22).

The concentration and exposure limit for Hg in plants is 0.03 µg g⁻¹ as recommended by Food and Agricultural Organisation (FAO) and WHO (Table 4) (24–26, 35–37).

The world's major producers of Hg are Algeria, Kyrgyzstan, Spain, USA and China (Fig. 4) (38).

Sources of Cd and Hg

Heavy metals are prevalent in the earth's crust and are dispersed unevenly (39). They may exist in the form of colloids, ions, particulates or dissolved phases (40). The presence of these metals is currently a major source of concern. The high concentration of these metals may have adverse effect on various lifeforms as well as the environment. The sources of these heavy metals can be natural as well as man-made (41) (Fig. 5).

The natural sources of these metals are mostly the result of volcanic eruptions, weathering of the parental metal rock, forest fires or they are naturally present in sedimentary rocks, marine phosphates or in the form of ores (18,19,41). Cd is widely dispersed in the earth's crust at a concentration of 0.1 mg kg⁻¹, while Hg is found at a value of 0.5 mg kg⁻¹. Sedimentary rocks have the highest concentrations of Cd and Hg. There is about 15 mg of Cd/kg present in the marine phosphates (18). About 7000 tonnes per year of Cd and 475 tonnes per year of Hg is emitted globally through coal combustion (19). Cd emissions from all sources (natural and anthropogenic) are estimated to be roughly 22000 metric tonnes per year while Hg emissions are estimated to be 5000–8000 metric tonnes per year (41,42).

Table 3. Physicochemical properties of mercury

Physicochemical Properties	Analytical data
Symbol	Hg
Atomic number	80
Group number	12
Colour	Silvery white
Atomic mass	200.59 g mol ⁻¹
Element classification	Transition metal
Ore	Cinnabar
State at room temperature	Liquid
Density	13.6 g cm ⁻³ at 20°C
Electronic configuration	[Xe] 4f14 5d10 6s2
Electronegativity	1.9
Thermal conductivity	Poor
Electrical conductivity	Good
Standard potential	+ 0.854 V (Hg ²⁺ / Hg)
Melting point	- 38.9 °C
Boiling point	356.6 °C
Oxidation states	+2, +1
Isotopes	56 total – 7 stable and 33 radioactive
Ionic radii	0.11 nm (+2)
Vander Waals radii	0.157 nm
Atomic radii	2.23 nm



Fig. 3. Cinnabar ore.

Table 2. Concentration and exposure limits for Cd in the environment by different safety standards

Sample	Safety Standard	Recommendation Level for Cadmium	References
Drinking water	Environmental Protection Agency (EPA) 2013	0.05ppm	(24)
Drinking Water	WHO 2011	3 µg/L	(25)
Waste water	World Health Organisation (WHO) 2011	0.003ppm	(25)
Agricultural soil	World Health Organisation (WHO) 2007	0.003ppm	(26)
Plants	Food and Agricultural Organisation (FAO) & WHO 2007	0.02 mg/kg	(26)
Leafy Vegetables	FAO/WHO 1996, European Commission 2006	0.2mg/kg	(27, 28)
Leafy Vegetables	FSSAI 2020	0.3mg/kg	(29)
Root Vegetables	FAO/WHO 1996	0.3mg/g	(27)
Root Vegetables	European Commission 2006	0.1mg/kg	(28)
Root Vegetables	FSSAI 2020	0.2mg/kg	(29)
Cereals	ATSDR 2012	0.1 µg/kg	(30)
Cereals	EU 2021	0.04mg/kg	(31)
Spices (Turmeric powder)	FSSAI 2020	0.1ppm	(29)

Table 4. Concentration and exposure limits for Hg in the environment by different safety standards

Sample	Safety standards	Recommendation level for mercury	References
Drinking water	Environmental Protection Agency (EPA) 2013	2 parts per billion (ppb)	(24)
Drinking water	WHO 2005	0.006 mg/L	(35)
Waste water	World Health Organisation (WHO) 2011	0.0019 ppm	(25)
Agricultural soil	World Health Organisation (WHO)2007	0.05ppm	(26)
Vegetables	Food and Agricultural Organisation (FAO) & (WHO)2007	0.03 µg/g	(26)
Cereals	WHO/FAO 2001	0.10 mg/kg	(36)
Cereals	EU 2018	0.01 mg/kg	(37)

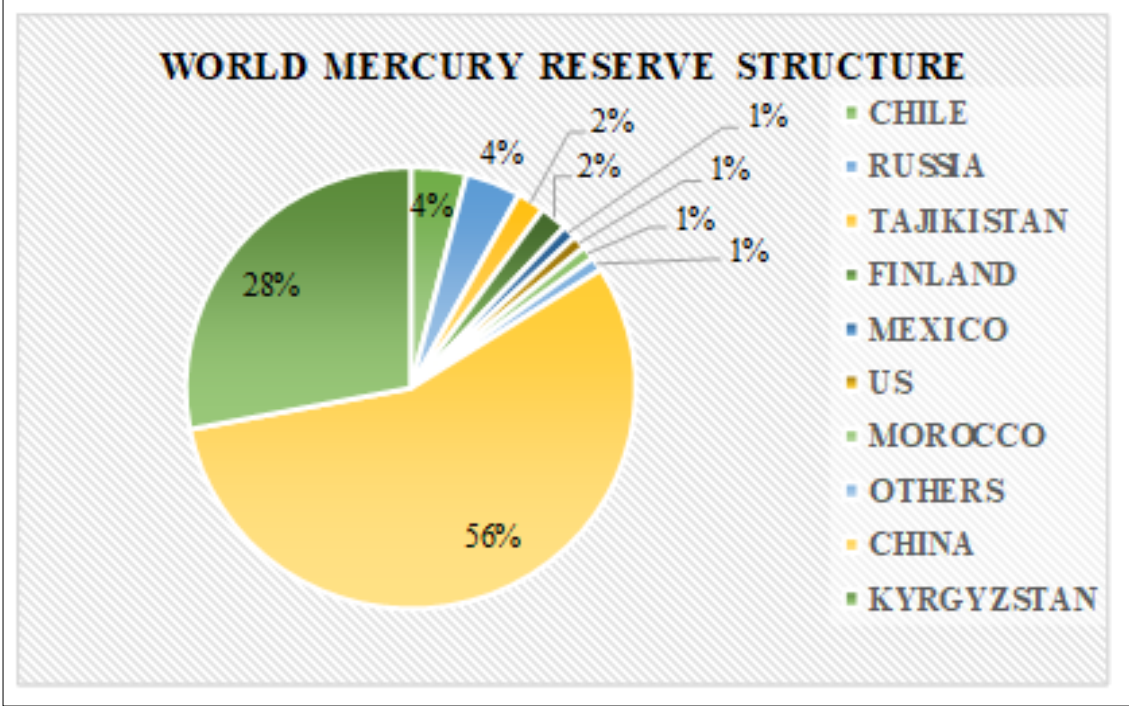


Fig. 4. Distribution of cadmium reserves in the world.

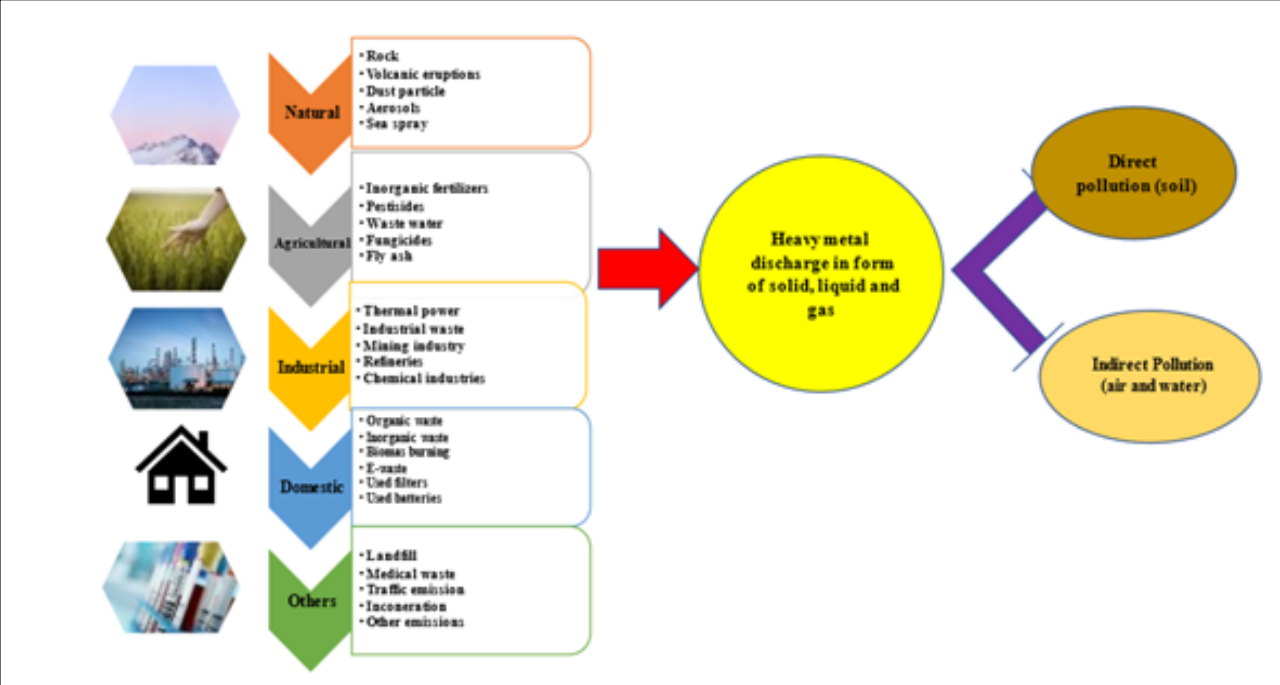


Fig. 5. Heavy metal sources and its discharge causing pollution in the environment.

Cd concentration in soil and water rises due to both anthropogenic and geogenic sources (43). Mining, smelting, wastewater irrigation, industries and vehicular emission, are the major sources of Cd pollution in soil and ground water. Unlike other heavy metals Cd is highly mobile in nature. This high mobility contributes faster release of Cd from soil into

groundwater than other heavy metal (43). Hg is released into the environment through industrial waste particularly from mining and manufacturing processes such as cement and non-ferrous metal production (44). Once released these heavy metals persist in nature contaminating aquifers and surface water bodies due to their non-biodegradable nature (45).

Industrial production and use of Cd and Hg

Cd is mainly used in industries for the manufacturing of alloys, pigments and batteries (18). Cd is employed in the fabrication of rechargeable Cd-Ni batteries to the tune of 80 %. Paint pigments containing cadmium sulphide, usually known as yellow Cd, are used. Due to its physical property of being able to absorb neutrons it is used in nuclear reactor control to keep the reactions under control. It is alloyed with silver to make solder for indoor sprinkler system. It is used as a stabilizer for PVC products and in electroplating of steel (22).

Hg is utilized in the electrical sector to make thermostats, barometers, switches and batteries, among other things. It is alloyed with other metals to form amalgams, zinc amalgam is used in dry batteries whereas silver amalgam is used in dentistry to make dental fillings. Fluorescent lamps, streetlights and other electronic devices utilise Hg vapours. Hg batteries are made from Hg oxide. It is used to make caustic soda.

Impact of cadmium and mercury

The high toxicity of heavy metals has been known for a long time and have remained a cause of major concern (46). There is a drastic global increase in heavy metal contamination of soil, water and air over the last few decades due to the increased demand of modernization (47). The presence of these metals at extreme concentrations in the environment have a negative impact on overall biological life and the ecosystem. They affect the biological system by inducing toxicity and inhibiting cellular activities.

Effect on humans and animals

The main threat to human health may be the exposure to Cd and Hg (18) depending on where the heavy metal is bound in the body (48). Heavy metals are transported and compartmentalised into body cells and tissues, where they bind to proteins and nucleic acids, destroying them and disrupting their cellular functions. Cd exposure may lead to kidney damage, skeletal damage, lung emphysema and inflammation, hepatic damage. Pregnant women, infants and young ones are more susceptible to Cd exposure. During pregnancy exposure of Cd can lead to many detrimental effects such as low birth weight, preterm birth and developmental disorders (49). Hg can harm neurocognitive functions like fine motor skills, speech function and memory. Acute Hg poisoning can cause lung damage. Neurological and psychological signs of chronic poisoning include tremor, personality changes, restlessness, anxiety, sleep disturbances and depression (22).

Effect on microbes

Heavy metals in the environment can lead to a decline in both the amount and variety of the microbial community (48) by altering their physiological and biochemical properties (50). Cd can cause oxidative damage and denaturation in microorganisms, as well as reducing their ability to bio remediate (50). It can disrupt enzyme activities and prevent DNA-mediated transformation in microorganisms (51). Cd toxicity in microbial cells is thought to be caused by protein depletion of glutathione and sulfhydryl groups, interactions with nucleic acids, oxidative damage caused by ROS and inactivation of metalloproteins due to displacement of Zn and Fe ions (52). Hg toxicity causes protein

denaturation, cell envelope disruption, inhibition of cell division and enzyme activities, nucleic acid destruction and transcription inhibition in microorganisms (52).

Effect on plants

Plant roots readily absorb heavy metals and transport them to various plant organs. Excessive amounts of heavy metals in soil commonly cause stress symptoms in plants, such as reduced growth, mineral nutrition and carbohydrate metabolism disturbances (53). Higher plants can absorb metals from the atmosphere through their shoots and leaves and enter the soil via roots and rhizomes. When metals move from the soil to the plant roots, they are transported and stored in various locations throughout the plant, causing toxicity. The total amount of potentially available or bioavailability of the metal (quantity factor), the activity as well as the ionic ratios of elements in soil solution (intensity factor) and the rate of element transfer from solid to liquid phases and to plant roots all play a role in the transfer of HMs from soils to plants (reaction kinetics) (47). Some metals are required for normal plant growth and development as cofactors and/or structural and catalytic components of proteins and enzymes at appropriate concentrations. Toxic levels of heavy metal ions, on the other hand, cause a variety of cellular stress responses as well as damage to various cellular components such as membranes, proteins and DNA. When heavy metals build up in plant tissues, they affect vital growth processes like mineral nutrition, transpiration, photosynthesis, enzyme activities related to chlorophyll metabolism and biosynthesis, nucleic acids and seed germination (54).

Heavy metal can affect plants in numerous ways. It may cause phytotoxicity, oxidative stress, cytotoxicity and genotoxicity in plants. This paper deals with the genotoxic effect of heavy metal such as Cd and Hg in plants.

Phytotoxicity

Heavy metal phytotoxicity is caused by an imbalance between an element's uptake and the metabolism's ability to cope with its cellular, particularly cytosolic, concentrations (55). Hg^{2+} may not have a significant impact on plant growth at low concentrations, but at greater concentrations, it becomes highly phytotoxic to plant cells, causing obvious damage and physiological abnormalities (56). Photosynthesis, transpiration, water intake and chlorophyll synthesis can all be affected by Hg exposure. Hg, both organic and inorganic, has been shown to induce potassium, magnesium and manganese loss, as well as iron accumulation (57).

Cd is considered to be one of the most phytotoxic heavy metals (57). It has a deleterious impact on all elements of photosynthesis, including the Electron Transport Chain (ETC), photosystems (PSI and PSII), chl-protein complexes and CO_2 reduction pathways in the stroma. The ultrastructure of chloroplasts is similarly altered by Cd-induced toxicity, with swollen and disordered thylakoids (58). Cd produces a large reduction in carbon metabolism, which leads to a shift in photosynthesis due to a lack of CO_2 and low carbon levels interfere with ETC and thylakoid membranes, as well as photosynthetic enzymes. Cd toxicity affects photosystems (PSI and PSII), interacts with the PSII core complex and supramolecular entities and slows photoactivation (59).

Cytotoxicity

Heavy metals induce both oxidative and genotoxic stress responses in plants, resulting in cytotoxicity and damage to various cellular components such as proteins, membranes and nucleic acids, resulting in a typical abiotic stress response. Extreme heavy metal toxicity frequently affects the cellular redox system and causes increased formation of ROS as a result of oxidative stress (60).

Cd has been identified as a key element in the development of oxidative stress through the creation of ROS and the weakening of non-enzymatic antioxidant defences. Through membrane lipid peroxidation, Cd-mediated oxidative stress has been demonstrated to regularly affect bio-membrane function in plants. In plants it damages cell membranes, chloroplast ultrastructure, photosynthetic pigments and finally impairs antioxidant metabolism, causing severe growth retardation. Through oxidative stress induced ROS formation, high levels of Hg cause membrane lipid peroxidation, mitochondrial dysfunction and ultimately cellular metabolism in plants (60). High concentration of Hg substantially reduces the carbohydrate and protein level and also disrupts vital physiological processes. Moreover the activity of essential antioxidant enzymes like catalase, superoxide dismutase was greatly reduced under Hg stress (61).

Morphological toxicity

Plants respond to metal stress with morphological and metabolic changes that are thought to be adaptive responses. The main morphological and structural effects of metal toxicity in roots can be summarised as follows: (i) decreased root elongation, biomass and vessel diameter, (ii) tip damage, (iii) root hair collapse or decrease in number of roots, (iv) increased or decreased lateral root formation, (v) enhanced suberification and lignification and (vi) changes in hypodermis and endodermis structure. Cd slows growth while also altering physiological and biochemical features such as water balance, food intake and photosynthetic electron transport around photosystems. Cd causes morphological changes in older leaves, such as drying and chlorosis and necrosis in younger leaves (40). Seed germination, germination relative index, root and shoot length and dry weight, shoot root ratio and seedling vigour index, plant

height, phytomass, number of leaves and branches, leaf area and chlorophyll contents and nitrate and nitrite reductase activity have all been shown to be negatively affected by Cd (42).

Genotoxicity

Several lines of evidence show that a range of substances cause oxidative stress by producing ROS, as well as genotoxicity via ROS-mediated DNA damage and genomic instability (Fig. 6). ROS assault DNA and cause alterations in DNA bases, resulting in further DNA damage such as single and double strand breaks in the DNA molecule that may not be repaired.

Under ROS stress, DNA Double-Strand Breaks (DSBs) are one of the most common types of DNA damage, resulting in genomic instability and DNA fragmentation (17). Overproduction of ROS/RNS causes a decrease in the quantity of cellular antioxidants, resulting in an imbalance between free radicals and the cellular antioxidant-mediated defence mechanism, which causes oxidative stress-mediated DNA damage (1). Overall, DNA damage has the potential to be both genotoxic and cytotoxic to the cell.

Mercuric ions can cause oxidative stress in plants by causing the production of ROS such as superoxide radicals ($O_2^{\cdot-}$), hydrogen peroxide (H_2O_2) and hydroxyl radicals ($OH^{\cdot}$) (62). ROS are produced continually by a variety of intracellular oxidative activities and have the ability to affect enzyme performance, modulate intracellular signalling and cause damage to biological macromolecules such as DNA. Antioxidants, ROS-eliminating enzymes and an effective DNA repair mechanism are all present in cells. When ROS levels surpass the capacity of defence or antioxidant systems, DNA suffers oxidative damage, which frequently results in DNA mutations, DSBs and chromosomal deletions (53). According to this theory, different types of stress elicit similar adaptive responses, implying that Reactive Oxygen Intermediate (ROI) production plays an important role. ROI are important in plant defence, signal transmission, plant growth and development, but they can also cause genotoxicity and Programmed Cell Death (PCD) by damaging genetic material (1). There are few studies on Hg's genotoxicity and the cellular and molecular mechanisms underlying this metal's toxicity are

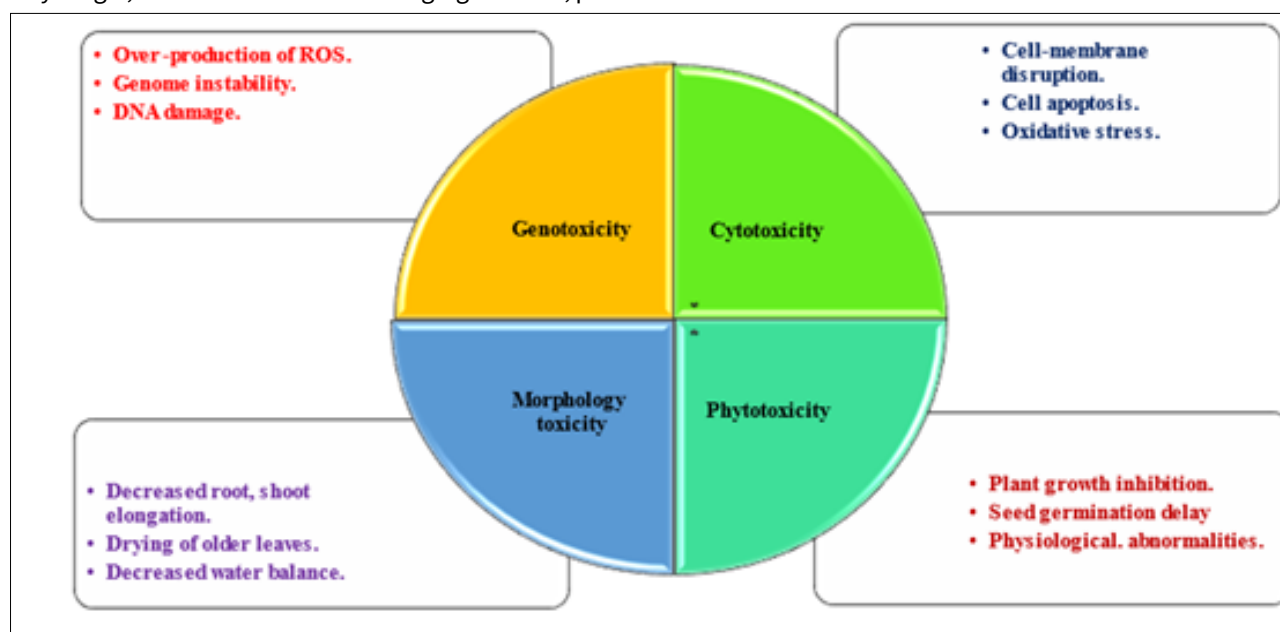


Fig. 6. Plant response to cadmium and mercury stress.

mostly unknown. Because of their easily malleable outer electron shells, Hg ions tend to establish covalent connections within cells. Depending on environmental factors such as ionic strength, the presence of competing ions and base makeup, DNA contains a variety of potentially reactive sites for Hg bonding (57). The effects of Hg's forms on plants are dependent on the concentration and length of exposure, with the most pronounced effects in the S-phase, when it can cause damage and severe clastogenicity. Low dosages of Hg have been shown to cause c-mitosis, sister chromatid swaps, chromosomal abnormalities and spindle modifications in plants (57). Several N and O atoms can be found in ribose/ribosephosphate groups and purine/pyrimidine bases, which could be possible Hg binding sites. The ligation of Hg to nucleobases can result in nucleobase impairment; this phenomenon has been linked to Hg's mutagenic potential. Hg can affect the amino acid sequences of proteins that are to be produced in this way. Despite all of this, many elements of Hg's genotoxicity remain unexplained, including the mechanism, target and degree of its effects on plants (57).

Heavy metals have been proven to have direct and/or indirect effects on DNA and chromosomal synthesis and duplication. In plant cells, metal genotoxicants can also cause chromosomal abnormalities. The metal species and contamination dosage can have a big impact on the genotoxic effects. Polyploidy, C-Karyokinesis, chromosomal fragmentation, chromosome fusion, micronuclei production and nuclear breakdown are all symptoms of Cd and Hg exposure in beans and onions (*Allium cepa*). Cd has also been linked to cell death in tobacco (*Nicotiana tabacum*) due to the formation of superoxide anions (O_2^-) from mitochondria and membrane peroxidation. Cd has been shown to have genotoxic and cytotoxic effects and high amounts of Cd can cause oxidative stress in plants by inducing lipid peroxidation (42). Furthermore, genotoxicants in some plants can disrupt the sequence of DNA processing activities such as replication, methylation and the repair mechanism, resulting in mutations (42). In a dose-dependent way, mercuric ions appear to establish covalent connections with DNA, causing sister chromatid exchange, lower mitotic index and increased frequency of chromosomal abnormalities (60).

Effect on enzymes

Heavy metals have been found to inhibit enzymes involved in the photosynthetic carbon reduction cycle, as well as all three phases of the Calvin cycle (carboxylation, reduction and regeneration), particularly the carboxylation phase, in plants (40). Cd toxicity affects cell membranes and inactivates enzymes, potentially by interacting with proteins belonging to the SH-group, which reflects the inhibitory effects of Pb^{2+} , Cd^{2+} , Zn^{2+} and Cu^{2+} on chloroplast enzyme activity (40). Catalase activity is lowered at increasing metal concentrations, while SOD activity is unaltered. Cd interferes with antioxidant enzymes in a negative way (40). Following Cd treatment, there was a considerable increase in SOD, APX and GR activity, as well as a decrease in CAT. Increased SOD activity and inhibition of CAT and POD resulted in a high level of H_2O_2 , which is most likely the main cause of oxidative stress in plants caused by Cd regimens. The activities of NAD(P)H oxidase, on the other hand, were considerably stimulated in response to Cd exposure (42). Plants have a highly effective antioxidative defense mechanism that includes a variety of enzymatic (such as superoxide dismutase, catalase and

guaiacol peroxidase) and non-enzymatic elements that scavenge ROS and mitigate their harmful effects. Previous research has shown, however, that Cd inhibits plant antioxidant ability, resulting in reduced plant development (63).

Membrane lipid peroxidation

Excessive levels of heavy metals cause changes in membrane fatty acid composition and membrane injury can occur when the peroxidation of membrane lipid rises due to highly hazardous free radicals. Peroxidation of lipids in both the plasma membrane and the chloroplast membrane has been documented as a result of a large quantity of metal ions (42). Increased lipid peroxidation also changes membrane properties including fluidity and permeability, as well as modulating the activities of membrane-bound ATPases. Lipid peroxidation products have been found to have a negative impact on plasma membrane activities, culminating in cell death. The application of various amounts of multiple heavy metal stress (such as Cd^{2+} and Hg^{2+}) accelerated the lipid peroxidation of *Bruguiera gymnorhiza* plants (42). Lipid peroxidation is a highly damaging process that damages DNA while also changing the structure and function of the cellular membrane. Endogenous lesions are caused by lipid peroxidation, which causes the creation of exocyclic DNA base adducts. Lipid peroxidation produces a complex array of nucleophiles capable of interacting with DNA, resulting in the creation of mutagenic adducts (64).

Genotoxicity assays

Several studies have been conducted in recent years using cytological (chromosome aberrations and micronuclei formation assay), molecular (comet assay) and others to assess genotoxicity in plants following exposure to various heavy metals, such as Hg and Cd (60). For the testing of genotoxins, plant-based tests have been recognized reliable bioassays.

The effects on micronuclei formation and cell division frequency by micronucleus test

If cells are actively dividing following exposure to genotoxicants, micronuclei develop, which are visible in non-dividing cells. A drop in the Mitotic Index (MI) compared to the control indicates toxicity and reduces the number of micronuclei. However, if cells are not actively split, the absence or decrease of MN frequencies could lead to erroneous interpretations or conclusions about the safety of the tested chemical. Indeed, a hazardous chemical could have a faster influence on MN development than a genotoxic one. The absence of harmful effects was indicated by a comparison of the MI for all treatments, which were all higher than the unexposed control (65). In plant root cells, Cd was discovered to reduce the MI and to cause chromosomal abnormalities and the development of micronuclei (MN) (66).

Sister Chromatid Exchange (SCE) test

DNA segregation between two sister chromatids of a replicating chromosome, occurs during DNA replication. The hypothesis that sister chromatid exchanges are initiated when daughter strands of a duplicated DNA cluster recombine with the parental strands of a partially replicated cluster was proposed as a model for the generation of sister chromatid exchanges. After exposure to genotoxic/mutagenic chemicals, the frequency of SCEs per chromosome set rises. Plants with a modest number of chromosomes and a large size, such as *Vicia faba* and *Allium cepa*, are used for SCE testing (17).

Comet assay

The comet assay determines the genotoxicity of heavy metals. It is the most widely used *in vivo* test for determining the mutagenicity/genotoxicity of genotoxic substances. In this approach, detergents and salts are used to lyse cells, releasing their genetic material, which is then electrophoresed (1). DNA fragments travel toward the anode using electrophoresis, generating a classic "comet tail." In comparison to the DNA remaining in the head, the amount of DNA in the tail represents DNA strand breakage (17). Cells with more DSBs migrate more quickly to the anode and produce more bands. It can detect a wide range of primary DNA lesions that are difficult to detect with other tests and it only takes a few cells to do so. It can also be used to detect low amounts of DNA damage in a wide range of cells or tissues (1).

Conclusion

Plant cells produce ROS in response to direct or indirect chemical exposure, causing oxidative stress. Heavy metals such as Cd and Hg are capable of inducing oxidative stress which leads to over-production of ROS. It has been found that at high concentrations, heavy metals have more negative impacts on plants than at low concentrations. ROS can stimulate genotoxicity in plants by inducing DNA damage, resulting in a range of cytotoxicity and genotoxicity end points. Genotoxic substances cause DNA damage, which can either be repaired or result in DNA changes. Chromosome aberrations occur when a DNA double strand break is not repaired or is repaired incorrectly. Without telomeres, broken chromosome ends become "sticky" and may fuse with other broken chromosome ends. Acentric fragments, dicentric bridges in mitotic cells of the first cell cycle after mutagenic therapy, or micronuclei in the interphase cell of the next cell cycle are the result of these chromosomal rearrangements. The frequency and kind of chromosome abnormalities in mitotic cells, as well as the frequency of micronuclei in interphase cells, cell division irregularities, chromosomal aberrations, sister chromatid exchange, are the most extensively used plant-based genotoxicity assays for detecting persistent DNA-damage. Genome instability is caused by genotoxic damage, which has a negative impact on plant growth and yield. Plants respond to induced oxidative and genotoxic damage by activating transcription factors, which causes a fast shift in the expression of the responsive genes at the transcriptional level. Seed germination, root elongation and biomass, as well as physiological and biochemical endpoints such as antioxidant enzymes, cell structure and hormones, are currently the focus of heavy metal toxicity studies in plants.

Acknowledgements

The authors greatly acknowledge the e-resource facility by OSHEC, Department of Higher Education, Government of Odisha, DBT, Government of India, DST CURIE (Consolidation of University Research for Innovation & Excellence in Women Universities) Government of India for institutional wi-fi facility to access different literature which facilitate in articulating latest information in this review article.

Authors' contributions

MM contributed in conceptualisation and collection of information and its articulation, along with writing the draft manuscript. PM information collection, designing of graphs, tables and helps in writing few parts of the article. SN contributed to writing, editing and formatting the references of this article. All authors read and approved the final manuscript.

Compliance with ethical standards

Conflict of interest: Authors have no conflict of interest to declare.

Ethical issues: None

References

1. Tabassum J, Jeena AS, Pandey D. Metal induced genotoxicity and oxidative stress in plants, assessment methods and role of various factors in genotoxicity regulation. In: Induced genotoxicity and oxidative stress in plants. 2021:133-49. https://doi.org/10.1007/978-981-16-2074-4_5
2. Mohanty M, Dhal NK, Patra P, Das B, Reddy PS. Phytoremediation: a novel approach for utilization of iron-ore wastes. *Rev Environ Contam Toxicol*. 2010;206:29-47. https://doi.org/10.1007/978-1-4419-6260-7_2
3. Mohanty M, Patra HK. Attenuation of chromium toxicity by bioremediation technology. *Rev Environ Contam Toxicol*. 2010;210:1-34. https://doi.org/10.1007/978-1-4419-7615-4_1
4. Kabata-Pendias A. Trace elements in soils and plants. Boca Raton: CRC Press; 2010. <https://doi.org/10.1201/b10158>
5. U.S. Environmental Protection Agency (EPA). Cadmium Compounds; 2006.
6. U.S. Environmental Protection Agency (USEPA). Integrated Risk Information System. Washington (DC); 2020.
7. Arora NK, Chauhan R. Heavy metal toxicity and sustainable interventions for their decontamination. *Environ Sustain*. 2021;4(1):1-3. <https://doi.org/10.1007/s42398-021-00164-y>
8. Dutta A, Patra A, Jatav HS, Jatav SS, Singh SK, Sathyanarayana E, et al. Toxicity of cadmium in soil-plant-human continuum and its bioremediation techniques. In: Soil contamination-threats and sustainable solutions. 2020:22. <https://doi.org/10.5772/intechopen.94307>
9. Shahzad K, Danish S, Mubeen S, Dawar K, Fahad S, Hasnain Z, et al. Minimization of heavy metal toxicity in radish (*Raphanus sativus*) by strigolactone and biochar. *Sci Rep*. 2024;14(1):13616. <https://doi.org/10.1038/s41598-024-64596-2>
10. Abu-Shahba MS, Mansour MM, Mohamed HI, Sofy MR. Effect of biosorptive removal of cadmium ions from hydroponic solution containing indigenous garlic peel and mercerized garlic peel on lettuce productivity. *Sci Hortic*. 2022;293:110727. <https://doi.org/10.1016/j.scienta.2021.110727>
11. Khan A, Khan AA, Samreen S, Ansari MS, Hasan SA, Irfan M. Morphological, ultrastructural, biochemical and yield attribute variations in *Nigella sativa* L. by cadmium (Cd) stress: implications for human health risk assessment. *Biocatal Agric Biotechnol*. 2025;64:103514. <https://doi.org/10.1016/j.bcab.2025.103514>
12. Dawood MF, Sofy MR, Mohamed HI, Sofy AR, Abdel-Kader HA. N- or/ and P-deprived *Coccomyxa chodatii* SAG 216-2 extracts instigated mercury tolerance of germinated wheat seedlings. *Plant Soil*. 2023;483(1):225-53. <https://doi.org/10.1007/s11104-022-05732-7>
13. Pal S, Kundu R. Heavy metal induced genotoxicity detection by RAPD in alligator weed. *Int J Eng Technol Sci Res*. 2015;2(9):1-2.

14. Engwa GA, Ferdinand PU, Nwalo FN, Unachukwu MN. Heavy metal toxicity in humans. In: Poisoning in the modern world: new tricks for an old dog? London (UK): IntechOpen; 2019. p. 77–99.
15. Malar S, Sahi SV, Favas PJ, Venkatachalam P. Mercury heavy-metal induced physicochemical changes and genotoxic alterations in water hyacinths (*Eichhornia crassipes* (Mart.)). Environ Sci Pollut Res. 2015;22:4597–608. <https://doi.org/10.1007/s11356-014-3576-2>
16. Rout GR, Das P. Effect of metal toxicity on plant growth and metabolism: I. Zinc. Agronomie. 2009;23(1):873–84. https://doi.org/10.1007/978-90-481-2666-8_53
17. Badr A, El-Shazly HH, Mohamed HI. Plant responses to induced genotoxicity and oxidative stress by chemicals. In: Zeba Khan, Mohd Yunus Khalil Ansari, Durre Shahwar, editors. Induced Genotoxicity and Oxidative Stress in Plants. Singapore: Springer; 2021:103–31. https://doi.org/10.1007/978-981-16-2074-4_4
18. Tchounwou PB, Yedjou CG, Patlolla AK, Sutton DJ. Heavy metal toxicity and the environment. In: Luch A, editor. Molecular, clinical and environmental toxicology. Vol. 101. Basel: Springer; 2012:133–64. https://doi.org/10.1007/978-3-7643-8340-4_6
19. Ali H, Khan E, Ilahi I. Environmental chemistry and ecotoxicology of hazardous heavy metals: environmental persistence, toxicity and bioaccumulation. J Chem. 2019;2019(1):6730305. <https://doi.org/10.1155/2019/6730305>
20. Van Horn JD. Cadmium, physical and chemical properties. In: Encyclopedia of Metalloproteins. New York: Springer; 2013:383–4. https://doi.org/10.1007/978-1-4614-1533-6_32
21. Genchi G, Sinicropi MS, Lauria G, Carocci A, Catalano A. The effects of cadmium toxicity. Int J Environ Res Public Health. 2020;17(11):3782. <https://doi.org/10.3390/ijerph17113782>
22. Järup L. Hazards of heavy metal contamination. Br Med Bull. 2003;68(1):167–82. <https://doi.org/10.1093/bmb/ldg032>
23. Metalpedia. Cadmium: resources & production. Asian Metal.
24. U.S. Environmental Protection Agency (EPA). Drinking water contaminants; 2013.
25. World Health Organization. Guidelines for drinking-water quality. 4th ed. Geneva; 2011.
26. Food and Agriculture Organization; World Health Organization. Joint FAO/WHO food standard programme codex alimentarius commission: report of the 38th session of the codex committee on food hygiene. Houston (TX); 2007.
27. Food and Agriculture Organization; World Health Organization. Codex general standard for contaminants and toxins in foods. Joint FAO/WHO food standards programme, codex alimentarius commission. Rome; 1996.
28. The Commission of the European Communities. Commission regulation (EC) No 1881/2006 of 19 December 2006 setting maximum levels for certain contaminants in foodstuffs. Off J Eur Union. 2006;L364:5–24.
29. Food Safety and Standards Authority of India. Food safety and standards (contaminants, toxins and residues) Regulations; 2011.
30. U.S. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for cadmium. Atlanta (GA): Department of Health and Human Services, Public Health Service; 2012.
31. The Commission of the European Union. Commission Regulation (EU) 2021/1323 of 10 August 2021 amending Regulation (EC) No 1881/2006 as regards maximum levels of cadmium in certain foodstuffs. Off J Eur Union. 2021; L288(13):13–18.
32. Kozin LF, Hansen S. Physicochemical properties of metallic mercury. In: Mercury handbook: Chemistry, applications and environmental impact. Cambridge (UK): The Royal Society of Chemistry; 2013:1–35. <https://doi.org/10.1039/9781849735155-00001>
33. Maziek R. The mobile element mercury. ChemTalk; 2021.
34. Rice KM, Walker EM Jr, Wu M, Gillette C, Blough ER. Environmental mercury and its toxic effects. J Prev Med Public Health. 2014;47(2):74–80. <https://doi.org/10.3961/jpmph.2014.47.2.74>
35. World Health Organization (WHO). Mercury in drinking-water. Background document for development of WHO Guidelines for Drinking-water Quality. Geneva; 2005.
36. FAO/WHO. Codex Alimentarius Commission. Food additive and contaminants. Joint FAO/WHO Food Standards Programme. ALINORM 01/12A. 2001. p. 1–289.
37. The Commission of the European Union. Commission Regulation (EU) 2018/73 of 16 January 2018 amending Annexes II and III to Regulation (EC) No 396/2005 as regards maximum residue levels for mercury compounds in or on certain products. Off J Eur Union. 2018;L138:8–20.
38. MCGroup. Mercury: 2025 world market review and forecast to 2034. Merchant Research & Consulting Ltd; 2025.
39. Panda BB, Panda KK. Genotoxicity and mutagenicity of metals in plants. In: Physiology and biochemistry of metal toxicity and tolerance in plants. Dordrecht: Springer Netherlands; 2002:395–414. https://doi.org/10.1007/978-94-017-2660-3_15
40. Shah FU, Ahmad N, Masood KR, Peralta-Videa JR, Ahmad FU. Heavy metal toxicity in plants. In: Plant adaptation and phytoremediation. Dordrecht: Springer; 2010:71–97. https://doi.org/10.1007/978-90-481-9370-7_4
41. Rizvi A, Zaidi A, Ameen F, Ahmed B, AlKahtani MD, Khan MS. Heavy metal induced stress on wheat: phytotoxicity and microbiological management. RSC Adv. 2020;10(63):38379–403. <https://doi.org/10.1039/D0RA05610C>
42. Oves M, Saghir Khan M, Huda Qari A, Nadeen Felemban M, Almeelbi T. Heavy metals: biological importance and detoxification strategies. J Bioremed Biodeg. 2016;7(2):1–5.
43. Kubier A, Wilkin RT, Pichler T. Cadmium in soils and groundwater: a review. Appl Geochem. 2019;108:104388. <https://doi.org/10.1016/j.apgeochem.2019.104388>
44. Xu J, Bravo AG, Lagerkvist A, Bertilsson S, Sjöblom R, Kumpiene J. Sources and remediation techniques for mercury contaminated soil. Environ Int. 2015;74:42–53. <https://doi.org/10.1016/j.envint.2014.09.007>
45. Abdullah M, Ali SF, Khan SU. The overview of the problem of heavy metals contamination in the environment. In: Heavy Metal Contamination in the Environment. CRC Press; 2024:1–18. <https://doi.org/10.1201/9781032685793-1>
46. Cho UH, Park JO. Mercury-induced oxidative stress in tomato seedlings. Plant Sci. 2000;156(1):1–9. [https://doi.org/10.1016/S0168-9452\(00\)00227-2](https://doi.org/10.1016/S0168-9452(00)00227-2)
47. Tuteja N, Gill SS, editors. Crop improvement under adverse conditions. New York: Springer; 2013. <https://doi.org/10.1007/978-1-4614-4633-0>
48. Kepel BJ, Bodhi W, Tallei TE. Heavy metal (As, Cd, Cr, Hg, Pb) analysis and identification of heavy metal resistant bacteria in sediments from Manado Bay. In: IOP Conf Ser: Earth Environ Sci. 2021;926(1):012096. <https://doi.org/10.1088/1755-1315/926/1/012096>
49. Ali W, Bian Y, Zhang H, Qazi IH, Zou H, Zhu J, Liu Z. Effect of cadmium exposure during and after pregnancy of female. Environ Pollut Bioavailab. 2023;35(1):2181124. <https://doi.org/10.1080/26395940.2023.2181124>
50. Igiri BE, Okoduwa SI, Idoko GO, Akabuogu EP, Adeyi AO, Ejiogu IK. Toxicity and bioremediation of heavy metals contaminated ecosystem from tannery wastewater: a review. J Toxicol. 2018;2018:2568038. <https://doi.org/10.1155/2018/2568038>
51. Gonzalez Henao S, Ghneim-Herrera T. Heavy metals in soils and the remediation potential of bacteria associated with the plant microbiome. Front Environ Sci. 2021;9:604216. <https://doi.org/10.3389/fenvs.2021.604216>
52. Salam LB, Obayori OS, Ilori MO, Amund OO. Effects of cadmium perturbation on the microbial community structure and heavy

- metal resistome of a tropical agricultural soil. *Bioresour Bioprocess*. 2020;7:1-9. <https://doi.org/10.1186/s40643-020-00314-w>
53. Mediouni C, Houlne G, Chaboute ME, Ghorbel MH, Jemal F. Cadmium and copper genotoxicity in plants. In: *Biosaline agriculture and high salinity tolerance*. Basel: Birkhäuser Basel; 2008:325-33. https://doi.org/10.1007/978-3-7643-8554-5_30
 54. Aras S, Aydin SS, Körpe DA, Dönmez Ç. Comparative genotoxicity analysis of heavy metal contamination in higher plants. *Ecotoxicology*. 2012;21:107-24. <https://doi.org/10.5772/30073>
 55. Ernst WH. Phytotoxicity of heavy metals. In: *Fertilizers and Environment: Proceedings of the International Symposium "Fertilizers and Environment"*, Salamanca, Spain, 26-29 September 1994. 1996:423-30. https://doi.org/10.1007/978-94-009-1586-2_72
 56. Tiwari S, Lata C. Heavy metal stress, signalling and tolerance due to plant-associated microbes: an overview. *Front Plant Sci*. 2018;9:452. <https://doi.org/10.3389/fpls.2018.00452>
 57. Azevedo R, Rodriguez E. Phytotoxicity of mercury in plants: a review. *J Bot*. 2012;2012:848614. <https://doi.org/10.1155/2012/848614>
 58. Zulfiqar U, Jiang W, Xiukang W, Hussain S, Ahmad M, Maqsood MF, et al. Cadmium phytotoxicity, tolerance and advanced remediation approaches in agricultural soils; a comprehensive review. *Front Plant Sci*. 2022;13:773815. <https://doi.org/10.3389/fpls.2022.773815>
 59. Ali E, Hussain A, Ullah I, Khan FS, Kausar S, Rashid SA, et al. Cadmium phytotoxicity: issues, progress, environmental concerns and future perspectives. *Revista la Fac Ciencias Agrar UNCuyo*. 2020;52(1):391-405.
 60. Dutta S, Mitra M, Agarwal P, Mahapatra K, De S, Sett U, et al. Oxidative and genotoxic damages in plants in response to heavy metal stress and maintenance of genome stability. *Plant Signal Behav*. 2018;13(8):e1460048. <https://doi.org/10.1080/15592324.2018.1460048>
 61. Mani V, Gurusamy AK, Perumal V. Toxicological effects of Mercury in radish (*Raphanus sativus*) plants-Biochemical analysis. *Sch Acad J Pharm*. 2025;1:1-8. <https://doi.org/10.36347/sajp.2025.v14i01.001>
 62. Sahu GK, Upadhyay S, Sahoo BB. Mercury induced phytotoxicity and oxidative stress in wheat (*Triticum aestivum* L.) plants. *Physiol Mol Biol Plants*. 2012;18:21-31. <https://doi.org/10.1007/s12298-011-0090-6>
 63. Nouairi I, Jalali K, Essid S, Zribi K, Mhadhbi H. Alleviation of cadmium-induced genotoxicity and cytotoxicity by calcium chloride in faba bean (*Vicia faba* L. var. minor) roots. *Physiol Mol Biol Plants*. 2019;25:921-31. <https://doi.org/10.1007/s12298-019-00681-5>
 64. Tuteja N, Ahmad P, Panda BB, Tuteja R. Genotoxic stress in plants: shedding light on DNA damage, repair and DNA repair helicases. *Mutat Res Rev Mutat Res*. 2009;681(2-3):134-49. <https://doi.org/10.1016/j.mrrev.2008.06.004>
 65. Klein P, Chauvey L, Kallerhoff J, Pinelli E, Morard M, Silvestre J. A tool derived from the *Vicia faba* micronucleus assay, to assess genotoxicity, cytotoxicity or biostimulation of novel compounds used in agriculture. *Agronomy*. 2021;11(2):321. <https://doi.org/10.3390/agronomy11020321>
 66. Ünyayar S, Celik A, Çekiç FÖ, Gözel A. Cadmium-induced genotoxicity, cytotoxicity and lipid peroxidation in *Allium sativum* and *Vicia faba*. *Mutagenesis*. 2006;21(1):77-81. <https://doi.org/10.1093/mutage/gel001>