An Overview of Nickel (Ni^{2+}) Essentiality, Toxicity and Tolerance Strategies in Plants

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Authors' contributions

This work was carried out in collaboration between both authors. Author PS collected literature and wrote the draft manuscript under supervision of author NL. Both authors read and approved the final manuscript.

Review Article

ABSTRACT

Heavy metals (HMs) toxicity has an unavoidable threat to environment and public health due to their increasing contamination and accumulation in atmosphere which ultimately passes to the living beings by the route of food chain. Heavy metals are increasing rapidly in soil and water by weathering of rocks and anthropogenic activities and are now emerging as a major health hazard to humans and plants. Among them Nickel (Ni^{2+}) is a controversial element because of debate on its essentiality or non-essentiality in plants. Ni^{2+} is an important constituent (micronutrient) of many metallo-enzymes including urease, Ni-Fe hydrogenase, Ni-superoxide dismutase etc. while at higher level it affects all cellular and metabolic processes and causes retardation of germination, competition with other essential metal ions, osmotic imbalance, alteration of many enzymatic activities, disruption of cell structure and wilting, reduced photosynthetic activity, oxidative stress etc. Plants also possess some natural and stress-induced strategies to cope up with Ni^{2+} excess/toxicity. These strategies include growth regulators, antioxidative enzymes, amino acids as osmoprotectant, and chelation of Ni^{2+} with metalloproteins and metallothionins. This review focuses on researches done on the morpho-biochemical alterations induced by elevated Ni^{2+} concentration in plants and as well as the strategies adapted by plants to survive and neutralize the effects of these alterations.

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1. INTRODUCTION

Heavy metals (HMs) are present naturally throughout the world at different background states, due to their variable concentrations in the bedrock. Some HMs are used as essential micronutrients by the plants for the completion of their life cycle (respiration, photosynthesis, N_2 metabolism etc.), while others have neutral, deterring and toxic effects on floral visitor communities, pathogens and insect-pests even at trace/smaller (micro molar) concentrations [1]. Toxicity posed by HMs is potentially dangerous to health of biotic and abiotic components of the environment and has become a major concern due to their translocation and bioaccumulation in food chain (including plant products) used for human consumption [2]. The phytotoxicity of various HMs differs and the order of toxicity in plants reveals $As^{5+} < As^{3+} < Cr^{6+} < Co^{2+} < Ni^{2+} < Cu^{2+} < Ti^{+} < Hg^{2+} < Cd^{2+} < Ag^{+}$ [3]. The higher concentrations of these metals in plant cells results in alterations at the physiological, biochemical and cellular levels leading to the severe damage to plants [2,3,4].

Nickel (Ni^{2+}), is one of 23 metals that are of a concern to environmental and human health. Ni^{2+}, first discovered by Swedish chemist A.F. Cronstedt (1751), as a 24th most abundant element (hard, ductile and silver white) forming about 0.008% of the earth's crust. It has several oxidation states ranging from -1 to +4, but its bivalent (Ni^{2+}) form is the most common in biological systems. Ni^{2+} occurs either as a free metal in igneous rocks or in combination with irons. The major Ni^{2+} ores are garnierite [$(Ni,Mg)_3Si_2O_5(OH)_4$] and pentlandite [$(Ni,Fe)_9S_8$]. Ni^{2+} is ubiquitously present heavy metal emitted to the environment from both natural and anthropogenic sources. Natural sources include weathering of rocks whereas metal mining, smelting, vehicle emissions, fossil fuel burning, municipal and industrial waste, electrical batteries, metallurgical and electroplating industries are anthropogenic sources.

Generally, Ni^{2+} is uniformly distributed through the soil profile but typically accumulates at the surface from deposition by industrial and improper agricultural practices. Ni^{2+} deposition may represent a major problem in land near towns, industrial areas and agricultural lands receiving wastes such as sewage sludge. Ni^{2+} content in soil varies in a wide range from 3 to 1000 mg.kg^{-1} [5,6]. Naturally, it is present in soil in the range of 3 to 100 ppm and in water 0.0 to 0.005 ppm, respectively. However, Ni^{2+} polluted soils may exhibit Ni^{2+} concentrations in the range of 200 to 26,000 mg.kg^{-1} (20 to 30 fold higher than the natural range, i.e., 10-1000 mg.kg^{-1}) [5]. Considering elevated Ni^{2+} deposition in atmosphere, efforts should be made to systematically estimate/predict sustainable concentration of Ni^{2+} in plants and unravel the mechanism of interaction between plant and various biological compounds that help in combating Ni^{2+} induced stresses in plants.

The most common symptoms of Ni^{2+} toxicity in plants are inhibition of growth, seed germination, photosynthesis, sugar transport [7] and induction of chlorosis, necrosis and wilting [8]. Keeping in view the increasing Ni^{2+} toxicity to crop plants and significant importance of cereals, oilseeds, grain legumes and vegetables as source of low cost food, the present article discusses various aspects of stress measurements of Ni^{2+} toxicity to plants and their adaptation strategies to cope with these stresses.

2. Ni^{2+} IN PLANTS

In plants, Ni^{2+} is naturally present as an important constituent of some metalloenzymes including ureases, glyoxalases (family I), peptide deformylases, methyl Co-M reductases, hydrogenases and a few superoxide dismutases [9]. It plays important role in various metabolic processes including ureolysis, hydrogen metabolism, methane biogenesis and acetogenesis [10]. In small amounts, Ni^{2+} enhances the growth and yield of plants and is also essential for the biosynthesis of anthocyanins [11,12]. Ni^{2+} deficiency in soybean (*Glycine max* L.) leads to accumulation of toxic level of urea in their leaflet tips because of decrease in urease activity in the leaves [13]. Ni^{2+} deficiency is also found associated with the reduced symbiotic hydrogenase activity in *Rhizobium leguminosarum* that may directly affect the symbiotic N_2 fixation [14,15]. Thus, Ni^{2+} is an essential micronutrient for N_2 metabolism in plants. Excess nickel adversely affects germination process and seedling growth traits of plants by hampering the activity of the enzymes such as amylase and protease as well as disrupting the hydrolyzation of storage food in

germinating seeds [16,17]. Several studies in plants including maize [18] and cowpea [19] have confirmed that Ni toxicity can result in inhibited lateral root formation and subsequent development. Khan and Khan [20] investigating the toxic effect of nickel and cobalt on chickpea (*Cicer arietinum* L.) showed that toxicity of Ni on the biomass production was more pronounced than Co and both metals led to poor germination, growth and biomass production, chlorophyll content and resulted in the reduced yield. Root nodulation was suppressed and number of functional nodules appreciably decreased at 100 ppm and higher levels of Ni^{+2}. Al-Qurainy [21] also demonstrated that Ni at the concentration 150 $\mu g \cdot g^{-1}$ of soil severely reduced biomass, root and shoot length, plant height and leaf area in *Phaseolus vulgaris*.

The uptake of Ni^{2+} in plants is carried out mainly by root system via passive diffusion and as well as active transport [22]. However, the relative uptake mechanisms of Ni^{2+} through active or passive transport differ with plant species, soil acidity, oxidation state, presence of other metals and availability (concentration) of Ni^{2+} in the soil or nutrient medium [23,24]. Ni^{2+} may be delivered to roots by basipetal transport (primarily via epidermal and cortical cell layers) in the phloem [25] and is then further translocated into expanding leaves and root parts behind the meristem (growing tip) [26]. Ni^{2+} is rapidly redistributed to the youngest (expanding) plant parts throughout vegetative growth and the reproductive phase [27]. Furthermore, the micro flora of soil may also enhance Ni^{2+} uptake by plants. In a study by Ma et al. [28], Ni-resistant plant growth promoting bacteria (PGPB) *Psychrobacter* species have been reported to promote the plant growth and Ni^{2+} uptake by *B. juncea* (Indian mustard) and *B. oxyrrhina* (Smooth-stem turnip) in soil contaminated with 450 $mg.kg^{-1}$ Ni^{2+}. The accessibility of Ni^{2+} to the plants usually declines at high pH values of the soil due to formation of less soluble complexes. For example, in a study with *Lathyrus sativus,* Ni^{2+} uptake was reported to increase up to pH 5.0 and then progressively decrease as pH reached up to 8.0 [29]. Nickel readily forms complexes with organic acids and other dissolved organic matters which enhance Ni^{2+} solubility in soil.

Ni^{2+} uptake is competitively inhibited by Copper (Cu^{2+}) and Zinc (Zn^{2+}), because these three soluble metal ions seem to be absorbed by the same cation transporters. Soluble forms of Ni^{2+} complexes could be also intaken competitively by Mg^{2+} ion transporter in many plants that have ultimate adverse effects on photosynthetic activity [8]. At high concentrations, Ni^{2+} can readily transport through phloem (vascular tissue conducting sugar and metabolic products downwards) and Xylem (vascular tissue conducting water and dissolved nutrients upwards), therefore simply translocate to the upper part of plants from the root. Over 50% of the Ni^{2+} absorbed by the plant is retained in the roots due to sequestration in the cation exchange site of walls of xylem parenchyma cells and immobilization in the vacuoles of roots [22]. Eighty % of root Ni^{2+} is present in the vascular cylinder, while less than 20% in the cortex, which shows a high mobilization of Ni^{2+} in the xylem and phloem [25]. In addition to absorption via roots, Ni^{2+} can also enter the plants via the leaves. Nickel in stem and leaves is mainly located in the vacuoles, cell wall and epidermal trichomes associated with citrate, malate and malonate accumulation. Ni at excess competes with several cations, in particular, Fe^{2+} and Zn^{2+}, preventing them from being absorbed by plants, which ultimately causes deficiency of Fe^{2+} or Zn^{2+} and results in chlorosis expression in plants [20].

3. Ni^{2+} TOXICITY IN PLANTS

At higher concentrations, Ni^{2+} is reported to have deleterious effects on plant growth and metabolism and produces visible signs of toxicity. High nickel concentration in plants accounts for retardation of germination, competition with other essential metal ions, alteration of many enzymatic activities, disruption of cell structure and dehydration/wilting, oxidative stress etc. Ni^{+2} stress reduces germination, shoot and root growth, biomass production, development of branching system and induces abnormal flower shape, mitotic root tip disturbance, leaf spotting and foliar necrosis [30]. Excess Ni^{2+} also affects nutrient absorption by roots [31] and inhibits photosynthesis, transpiration and transport of photo assimilates from leaves [22,32]. An overview of various Nickel-induced alterations in plant growth and key metabolic functions are shown in Fig. 1. Decrease in all the key metabolic processes coupled with oxidative stress ultimately leads to reduction in growth and yield of crop plants. Various visual/morphological and metabolic effects of Ni^{2+} deficiency and excess/toxicity in different crop plants are presented in Table 1.
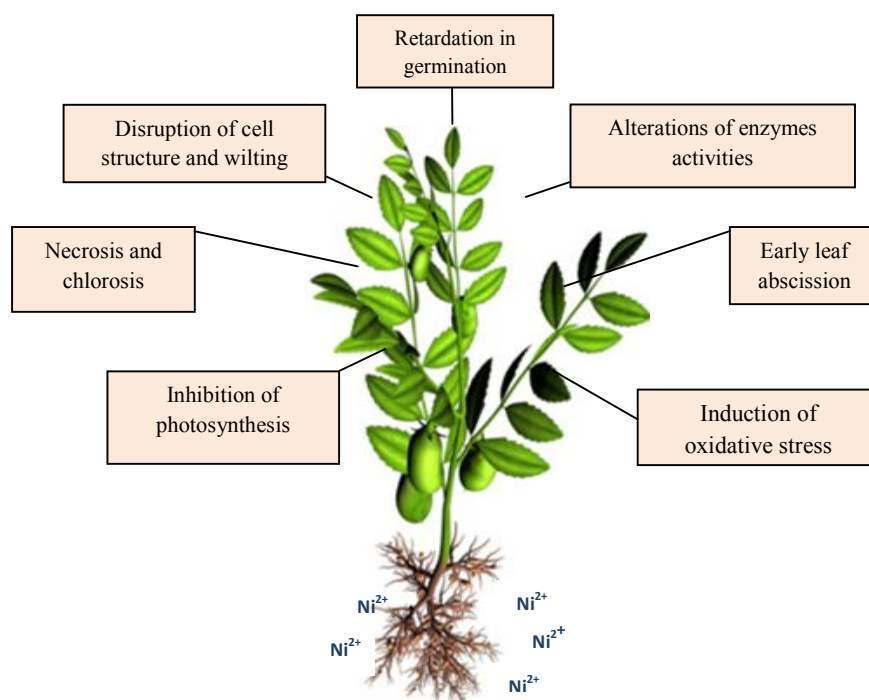
Fig. 1. Ni^{2+} induced alterations in plant growth and functions

4. MORPHO-BIOCHEMICAL EFFECTS

High doses of Ni^{2+} negatively affect plant growth and physiological processes and also induce visible toxicity symptoms. Most of the morphological characters such as root and shoot length, root nodules, leaf area, fresh weight and dry weight, chlorophylls, carotenoids, total sugar, amino acid, proline and protein contents decrease with increasing nickel chloride concentration [33]. The reason for decrease in all these parameters could also be the reduction in cell division in meristematic cells present in this region and activity of certain enzymes of cotyledon and endosperm. In Ni^{2+} treated plants, leaf size and leaf area are found to decrease which is also related to the accumulation of nickel in leaves. Accumulation of excess Ni^{2+} in plant tissues has been reported to cause leaf necrosis and chlorosis of plants [34]. Chlorosis and vein necrosis appeared in newly developed leaves of water spinach after plants were treated with 0.085 to 0.255 mM (5–15 ppm) Ni for a week [35]. Ni^{2+} at a concentration of 0.5 mM produced dark brown necrotic spots along the leaf margins resulting in wilting of outer leaves and necrosis of inner leaves in cabbage [8]. Similarly, Barley grown in presence of 0.1 mM Ni^{2+} for 14 days also showed chlorosis and necrosis of leaves [36]. Such chlorosis of leaves results from decreased synthesis of chlorophyll due to deficiency of Fe^{2+} and Mg^{2+} in Ni^{2+}-treated plants [22].

4.1 Inhibition of Growth

The toxic effects of Ni^{2+} and other heavy metals are primarily manifested by the inhibition of plant growth and germination [37] and this inhibition gains strength at higher metal concentrations. Singh et al. [38] and Talukdar [39] reported that presence of excess Ni^{2+} shows alterations in all energy driven cellular processes during germination thus, slows down emergence of radicles and plumules (embryonic shoots). In Scot Pine seedlings exposed to Ni^{2+}, reduced root sink activity was observed with reduced starch hydrolysis and sucrose transport may result in the accumulation of photo assimilate in leaves [40]. In Ni^{2+} excluder species, root growth is inhibited more strongly than the growth of shoots because Ni^{2+} mostly accumulates in their root cells [41,42]. Ni^{2+} stress has been also found associated with a substantial decrease in all macro and micronutrients in leaves and achenes of sunflower (*Helianthus annuus* L.) with a marked reduction in root and shoot fresh biomass and a consistent decrease in the contents of N, Fe, K, Zn, Mn, Ca and Cu with increasing level of Nickel [43]. Rahman et al. [36]

reported decrease in uptake of Zn, Cu, Fe, and Mn in barley shoots with increasing Ni^{2+} concentration in nutrient solution from 1 to 100 mM. This reduction in uptake of Zn, Cu, Fe, and Mn was observed due to Ni^{2+} accumulation in roots.

Table 1. Visual and metabolic symptoms of Ni^{2+} deficiency and excess in plants

Ni^{2+} level	Plant system	Visual and metabolic symptoms	References
Deficiency	*Glycine max* L.	Accumulation of toxic levels of urea in leaflet tips, leaf tip necrosis	[11]
	Legumes and higher plants	Early senescence, reduced Fe uptake, delayed nodulation and reduced efficiency of N fixation	[82,83]
	Cereals	Poor grain-filling and maturation process	[84]
	Carya illinoinensis	Development of "mouse-ear" leaves, bronzing, chlorosis, rosetting, and tip necrosis	[85]
	Triticum aestivum	Diminished plant resistance to leaf and stem rust	[86]
Excess	*Vigna cylindrica*, *V. mungo* and *V. Radiate*	Reduced seed germination and seedling emergence	[87]
	Brassica oleracea and *Triticum aestivum*	Inhibition of growth, chlorosis, necrosis, and wilting	[8]
	Cicer arietinum L.	Poor germination, growth and biomass production and chlorophyll content, Reduced yield. suppressed Root nodulation and number of functional nodules	[20]
	Triticum aestivum	Reduction in size of vascular bundle, width of epidermal cells and mesophyll thickness	[88]
	Vigna mungo (L.) Hepper	Reduction in photosynthetic pigments (chlorophyll and carotenoids)	[89]
	Cajanus cajan L.	Induction of oxidative stress (reactive oxygen species)	[50,90]
		Decreases chlorophyll content, stomatal conductance and CO_2 fixation	
	Brassica napus L.	Reduction in antioxidant enzymes activity	[9]
	Helianthus annuus L.	Decrease in the concentration of other micro/macro nutrients (N, K, Zn, Mn, Cu)	[43]
	Ipomoea aquatica	Chlorosis and along-vein necrosis in newly developed leaves	[35]
	Brassica oleracea	Dark brown necrotic spots along the leaf margins	[8]
	Hordeum vulgare L	Chlorosis and necrosis of leaves	[36]
	Lolium perenne	Reduction in plant nutrient acquisition, decrease in shoot yield, chlorosis	[92]
	Phaseolus vulgaris L.	Chlorotic leaves with gray spots that coalesce and become necrotic	[21,93]
		Reduced biomass, root and shoot length, plant height and leaf area	
	Soanum nigrum L.	Membrane damage and Ni^{+2} accumulation in root cells	[74]
	Various wild and cultivated plant species	neutral, deterring and toxic effects on floral visitor communities, pathogens and insect-pests	[99,100,101]

4.2 Inhibition of Photosynthesis

Heavy metals are directly related to the inhibition of photosynthesis, by several direct/indirect ways i.e. disorganized chloroplast structure, blocked chlorophyll biosynthesis, disordered electron transport, inhibited activities of the Calvin cycle enzymes, and CO_2 deficiency caused by stomatal closure [41]. The adverse impact of toxic levels of Ni on the photosynthetic apparatus and performance is conspicuous. At the biochemical level, Ni^{2+} affects light-harvesting complex II (LHCII) and the amounts of xanthophylls and carotenoids [44]. Nickel induced photosynthetic alterations include reduction in chloroplast size and numbers; disorganized chloroplast ultrastructure with the abated numbers of grana and thylakoids and altered membrane lipid composition have been confirmed in *Brassica oleracea* plants grown on agar media in the presence of $NiSO_4.7H_2O$ (10–20 $g.m^{-3}$). These changes in chloroplast result from the Ni^{2+} induced oxidative stress which further causes peroxidation of membrane lipids [44]. A detailed study revealed Ni^{2+} to inhibit electron transport from pheophytin to plastoquinone (Q_A) and Fe to plastoquinone (Q_B) by disrupting the structure of carriers and reaction center proteins such as plastoquinone (Q_B) [45]. At the cellular level, Ni^{2+} also decreases the contents of cytochromes b_6f and b_{559}, as well as ferredoxin (Fd) and plastocyanin (PC) in the thylakoids which consequently further reduces the efficiency of electron transport chain [48]. Sreekanth et al. [46] reported that Ni toxicity can lead to reduced chlorophyll content and interruption of electron transport. Ghasemi et al. [47] in maize (*Zea mays* L.) showed that excess Ni perniciously influenced photosynthetic protein complexes and the rate of Hill reaction diminished by increasing Ni concentration.

4.3 Induction of Oxidative Stress (ROS)

Oxidative stress is a complex physiochemical phenomenon that causes overproduction and accumulation of reactive oxygen species (ROS) responsible for abiotic stresses in higher plants. At cellular and molecular levels, Ni^{2+} binds strongly to oxygen (O), nitrogen (N), and sulfur (S) atoms present in different parts of plants. Ni^{2+} also shows high affinities towards sulfhydryl groups and disulfide bonds which cause damage to the secondary structure of proteins and also affect the activities of cellular enzymes, leading

to the disturbance of various metabolic pathways [48,49]. Excessive amount of Ni^{2+} significantly accelerate the concentration of hydroxyl radicals, superoxide anions, nitric oxide and hydrogen peroxide [50,51]. Since Ni^{2+} is not a redox-active metal, it cannot directly generate these reactive oxygen species but interferes with a number of antioxidant enzymes [8] such as superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPOX), glutathione reductase (GR), peroxidase (POD), guaiacol peroxidase (GOPX), and Ascorbate peroxidase (APX). Exposure of plants to Ni^{2+} at low concentrations and/or for short times has been shown to increase the activities of SOD, POD, GR, and GOPX to enhance the activation of other antioxidant defense's and finally leads to the removal (or scavenging) of ROS [52,53]. Lipid peroxidation may be a major contributing factor in Ni^{2+}-induced tissue oxidative stress. Ni^{2+}-induced oxidative stress in plants may be also associated with the competition between Ni and Fe in biochemical and physiological processes and also due to Ni-mediated modulation of the activities of antioxidant Fe enzymes (e.g., Fe SOD and CAT) [22,8,54,55]. An increase in Ni^{2+} concentration has been found to reduce the activity of many cellular antioxidant enzymes, both *in vitro* and *in vivo*, and plant's capability to scavenge ROS, leading to ROS accumulation and finally oxidative stress in plants [45].

5. ADAPTATION STRATEGIES TOWARDS Ni^{2+} TOXICITY IN PLANTS

Plants possess a sophisticated and interconnected network of biochemical defense strategies to avoid/tolerate Nickel intoxication as presented in Fig. 2 and Table 2. Some of these defense mechanisms used by plants against Nickel and other HMs are being discussed categorically in next section.

5.1 Physical Barriers

Physical barriers are naturally occurring defense system of plants against heavy metals. Morphological structures like thick cuticle, biologically active tissues like trichomes, and cell walls as well as arbuscular mycorrhizal fungi symbiosis can act as barriers when plants face HM stress [56,57]. Trichomes are fine outgrowths on plants and can either serve as HM storage site for detoxification purposes or can secrete various secondary metabolites to neutralize hazardous effects of metals [58].
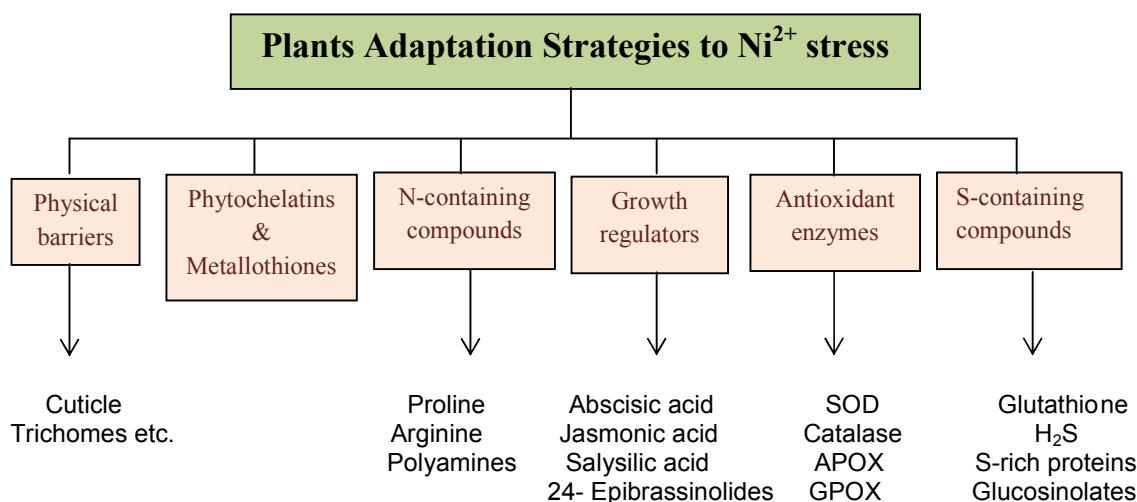
Fig. 2. Various biochemical adaptation and tolerance strategies to Ni^{2+} stress in plants

5.2 Amino Acid Derivatives (as osmoprotectant)

Plants often synthesize a set of diverse metabolites on exposure to metals. These metabolites accumulate in the range of milimolar concentrations and particularly include specific amino acids such as proline and histidine, peptides such as glutathione and the amines spermine (spm), spermidine (spd), putrescine (put), and nicotinamine. Thus, nitrogen metabolism is central to the response of plants to heavy metals. Proline has been considered as one of the important osmolytes as well as antioxidants found in the cellular system exposed to water stress, salinity stress, metal stress etc. In recent years, the role of proline has also been characterized as scavenger of ROS, generated during stress conditions [59]. Moreover, several studies report that under stress condition proline acts as an osmolyte and may increase the activity of antioxidant enzymes to minimize the adverse effect of oxidative stress caused by elevated Ni^{2+} [60].

Nasibi et al. [61] in their study on *Hyocyamus niger* found that Ni^{2+} showed decrease in chlorophyll a and total chlorophyll which was further maintained/recovered by Arginine pre-treatment in Ni^{2+} stressed plants. Pietrini *et al.* [62] in a study on *Amaranthus paniculatus* L. reported that the exposure of plants to increasing Ni^{2+} in the growth solution caused a significant increase in free polyamine content in roots and leaves of test plant at 25 µM NiCl$_2$, whereas a decrease in the PAs (Spermidine and Spermine) content of plants at higher Ni^{2+} concentrations.

Shahid et al. [63] in a study on *Pisum sativum* reported that the exogenous application of Pro (pure synthetic proline or proline enriched with essential nutrients) on pea protected the plant against phytotoxic impacts of nickel by reducing lipid peroxidation and electrolyte leakage, increase in activities of polyamine biosynthetic enzymes and thus, improving leaf polyamines and increasing concentration of endogenous compatible solutes. It was also concluded that Pro enriched with nutrients was more effective than pure Pro in enhancing plant growth under metal stress.

5.3 Organic Acids

Organic acids are carboxylic group containing compounds that act not only as intermediates in carbon metabolism but also as key components in mechanisms that some plants use to cope with nutrient deficiencies, metal tolerance and plant-microbe interactions operating at the root-soil interphase. Organic acids excreted from plant roots may form stable HM-ligand complexes with HM ions and change their mobility and bioavailability, thus preventing the HM ions from entering plants or avoiding their accumulation as well as translocation in the sensitive sites of shoots and roots. Yang et al. [64] examined the relationship of organic acid to Ni^{2+} accumulation in ryegrass (*Lolium perenne* L.) and maize (*Zea mays* L.) and reported 5 to 7 fold increased accumulation of Ni^{2+} in shoots of ryegrass than in maize grown at 20 to 80 µM Ni^{2+} whereas Ni^{2+} concentration in roots of ryegrass was found only 1 to 2 fold higher at 0.1 to 40 µM Ni^{2+} and 1.5 fold lower at 80 µM than that of maize roots.

Shoot concentrations of citric, malic, oxalic and cis-aconitic acids increased at 20 µM Ni^{2+} and were about 2 to 6 times higher in ryegrass than in maize. Whereas maize roots accumulated greater amount of malic, oxalic and cis-aconitic acids than ryegrass roots specially at Ni^{2+} levels of 40 to 80 µM. Research on several Ni^{2+} hyperaccumulators had shown that Ni^{2+} is predominantly bound to citrate and that the amount of citrate produced is strongly correlated with the accumulated Ni^{2+} [65].

5.4 Antioxidants Defense System

In plants, heavy metal toxicity frequently leads to the over production of ROS, resulting in peroxidation of many vital constituents of the cell. Plants develop a number of strategies to overcome with the adverse impacts imposed by heavy metals. To cope up with the situation, plants have an efficient defense system comprising of set(s) of enzymatic as well as non-enzymatic antioxidants. A wide variety of

Table 2. Biomolecules/structures involved in nickel tolerance in various plant species

Biomolecules/ structures	Plant system	Mechanism of tolerance	References
Trichomes	*Alyssum corsicum*	Accumulate excess amount of Ni^{2+} in root and shoot and also secrete secondary metabolites	[94]
Arbuscular mycorhizal fungi/ Glomalin	*Festuca arundinacea*	Alleviating Ni-induced stress by reducing Ni transport from roots to shoots	[95]
Phytochelatins and Metallothionines	*Thlaspi*	Binds to Ni^{2+} and sequesters it into vacuoles	[52]
	Solanum nigrum	Enhanced accumulation of Metallothionin related transcripts	[74]
Proline	*Pisum sativum*	Suppresses lipid peroxidation; electrolyte leakage and accelerating the activities total free amino acids, total soluble sugars, total phenol and tocopherol content	[63]
Histidine and calcium	*Solanum lycopersicum*	Regulate shoot and root length, pigment content of leafs and K^+ content of root and shoot	[96]
Arginine	*Hyoscyamas niger*	Counterbalance peroxidase and lipoxigenase activity of oxidative stress	[61]
Polyamines	*Hydrocharis dubia* *Pisum sativum*	Prevent Ni^{2+}-induced lipid peroxidation, electrolyte leakage and reduced Ni^{2+} accumulation	[97] [63]
Jasmonic acid	*Glycine max* L.	Significantly enhance antioxidant activity, while tightly inhibit stress related parameters responsible for lipid peroxidation	[69]
Salicylic acid	*Brassica napus*	Reduction in Ni^{2+} induced Chlorosis, necrosis of leaves and oxidative stress	[91]
	Triticum aestivum	Induce the activities of enzymes (SOD, POD, CAT)	[98]
Ethylene	*Brassica juncea*	Protects photosynthetic potential by efficient PS II activity and increase the activity of ribulose-1,5-bisphosphate carboxylase	[20]
Citrate, malate and oxalic acid	*Lolium perenne, Zea mays*	Form stable HM-ligand complexes, preventing accumulation and translocation to sensitive sites (roots and shoots)	[64]
Epibrassinosteroids	*Brassica juncea*	Improve membrane stability index and RWC, and increase proline and anti-oxidative enzymes	[70]

enzymatic antioxidants consisting of superoxide dismutase (SOD), peroxidase (POD), catalase (CAT) and glutathione-*s*-transferase (GST) which may efficiently convert the superoxide radicals into hydrogen peroxide and subsequently water and oxygen whereas low molecular weight non-enzymatic antioxidants consisting the proline, ascorbic acid and glutathione which may directly detoxify the ROS [66,67]. These two groups of antioxidants may successfully quench a wide range of toxic oxygen derivatives and prevent the cells from oxidative stress. Gajewska and Sklodowska [65] studied SOD, APOX, CAT and GST activity in leaves and roots of 14 days old pea plants treated with 10, 100, 200 µM $NiSO_4$. Ni^{2+} caused decrease in total SOD activity in both leaves and roots. The activity of APOX in leaves treated with 100 and 200 µM Ni^{2+} increased whereas in roots the enzymatic activity was reduced significantly. Catalase activity remained unaffected in both the organs in response to Ni^{2+}. The activity of GST in Ni^{2+} exposed plants increased in both the organs but markedly in roots. Gajewska and Sklodowska [68] concluded that stimulation of GST activity in tissue is mainly involved in response of pea plants under the Ni^{2+} stress.

5.5 Growth Regulators

Plant hormones are essential components of regulation of growth and development in plants and also play a crucial role in defense strategies against environmental stress. Plants produce reactive oxygen species (ROS) in response to the heavy metal toxicity which further induce the synthesis of several plant hormones such as jasmonic acid (JA), salicylic acid (SA), ethylene, epibrassinosteroids, abscisic acid (ABA) etc. Sirhindi et al. [69] studied modulatory role of JA on photosynthetic pigments, antioxidants and stress markers in *Glycine max* L. seedlings using exogenous application of JA prior to Ni^{+2} exposure. JA with or without Ni^{+2} stress caused amelioration of antioxidant enzyme system (SOD, POD, Catalase and APOX) and several-fold enhancement in cellular Ascorbic acid content. JA made seedlings more tolerant to Ni^{+2} stress as compared to control. Ali et al. [70] studied modulatory role of 24-epibrassinolide (EBL) in *Brassica juncea* exposed to NaCl and $NiCl_2$ alone or in combination. EBL improved the membrane stability index and relative water content, but did not influence electrolyte leakage and lipid peroxidation. The level of proline and anti-oxidative enzymes exhibited significant increase in response to EBL in both, NaCl and $NiCl_2$ stressed plant.

5.6 Phytochelatins (PCs)

Chelation and compartmentalization of heavy metals by Phytochelatins (PCs) is an ubiquitous detoxification phenomenon described in wide range of plant systems. Phytochelatins are low-molecular weight short chain thiol-rich peptides [71], synthesized from S-rich glutathione (GSH) by the enzyme phytochelatin syntheses (PCS) that have a high affinity to bind to HMs [72]. PCs form complexes with toxic metal ions in the cytosol and subsequently transported them into the vacuole. In transgenic *Arabidopsis*, GSH concentration has been found strongly correlated with increased resistance to Ni^{2+}-induced growth inhibition and oxidative stress (ROS) which suggests that high levels of GSH conferred tolerance to Ni^{2+}-induced oxidative stress in *Thlaspi* Ni^{2+} hyperaccumulators [52].

5.7 Metallothionins (MTs)

Metallothioneins (MTs) belong to the group of intracellular cysteine-rich, metal-binding proteins that have been found in bacteria, plants, invertebrates and vertebrates. Metallothioneins (MT) are gene-encoded metal chelators synthesized as a result of mRNA translation process and participate in the transport, sequestration and storage of metals [73]. MTs are divided into class I (vertebrates), class II (plants and fungi), and class III (higher plants) on the basis of their cysteine content and structure. Ferraz et al. [74] investigated the specific accumulation of MT-related transcripts in *Solanum nigrum* and observed that Ni^{+2} enhanced the accumulation of MT2a and MT2d mRNA (expressed constitutively) as well as *de novo* accumulation of MT2c and MT3-related transcripts in shoots. MT1 gene transcription remained unaffected due to Ni^{+2} toxicity. Thus, the involvement of MT2a, MT2c, MT2d and MT3 in Ni^{+2} homeostasis is evident from this study.

5.8 Ni^{2+} Phytoremediation

Phytoremediation of metal contaminated soil offers a low cost method for soil amendment. Several recent studies on Ni^{2+} hyperaccumulator plants have reflected their potential to sequester high levels of Ni^{2+} in their tissues (from several thousands of mg/ kg up to 5% of dry biomass) without exhibiting phytotoxicity [75]. More than

310 species of Ni^{2+} hyperaccumulators plants have been identified, including members of the Acanthaceae, Asteraceae, Brassicaceae, Caryophyllaceae, Fabaceae, Flacourtiaceae, Meliaceae, Myristicaceae, Ochnaceae, Poaceae, Rubiaceae, Sapotaceae and Stackhousiaceae [76,77]. These above said families have higher requirements for Ni^{2+} as micronutrient (e.g. up to 500 mg Ni^{2+}/kg) than normal plants. The family with the most Ni^{2+} hyperaccumulator species is the Brassicaceae, with more than 80 species which are capable of accumulating Ni^{2+} to concentrations as high as 3% of shoot dry biomass [78].

In addition, it is notable that many aquatic plants such as *Typha*, *Phragmites*, *Eichhornia*, *Azolla* and *Lemna* also have the potential to remove heavy metals from aquatic ecosystems [79,80]. These species have efficient root absorption mechanisms which allow them to specifically accumulate metals from soils and/or water. After root absorption, Ni^{2+} can be transported quickly into shoots and leaves of hyperaccumulators and then sequestrated in the vacuole [81]. For these features, Ni^{2+} hyperaccumulators have been extensively used to remove Ni from polluted soils and/or water.

6. CONCLUSIONS

The present article provides an overview to aspects related to the essentiality of Ni^{2+} in a wide range of physiological processes, starting from seed germination to the productivity. Moreover, without adequate supply of Ni^{2+}, plant life cycle can not be completed and proves it as an essential micronutrient. Elevated levels of Ni^{2+} alter almost all the metabolic activities of the plant and consequently minimize the photosynthetic rate, and biological yield of plants. Excess Ni-concentration also triggers oxidative damage in the plants. However, plants are well equipped with an organized constitutive/inducible defense system to counter the toxic effects that includes exclusion/restriction of entry of the metal into the cell through plasma membrane and chelation of the metal by phytochelatins, metallothionins and nicotianamide, followed by sequestration into the vacuole, making it less toxic for the plants. All these mechanisms are well understood and through integration of genetic engineering, it has been possible to manipulate expression of bacterial/higher plant genes involved in defense against nickel as well as to transfer them into susceptible genotypes leading their stable transformation into transgenic tolerant forms. Such transgenic plants hold great promise for cultivation of crops on contaminated croplands as well as for environmental clean-up and phytomining.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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