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

Priti Sachan\(^1\) and Nand Lal\(^1\)*

\(^1\)Department of Life Sciences, C.S.J.M. University, Kanpur – 208024, India.

*Corresponding author: E-mail: nl_pr@yahoo.co.in

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.
Keywords: Nickel; reactive oxygen species (ROS); antioxidant enzymes; osmoprotectants; Ni$^{2+}$ hyperaccumulators.

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$^{4+}$ < 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 24$^{th}$ 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)$_3$Si$_2$O$_5$(OH)$_2$] and pentlandite [(Ni,Fe)$_9$S$_8$]. 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 functional nodules appreciably decreased at 100 ppm and higher levels of Ni 

Furthermore, the micro-algae [21] and inhibits ion transporter in Ni 

5.0 and then progressively decreased up to 8.0 [29]. The accessibility of Ni 

complexes could be also intaken competitively by Mg 

upwards), therefore simply translocate to the conducting water downwards and Xylem (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 

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 

is present in the vascular cylinder, while less than 20% in the cortex, which shows a high mobilization of Ni 

in the xylem and phloem [25]. In addition to absorption via roots, Ni 

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 

and Zn 

, preventing them from being absorbed by plants, which ultimately causes deficiency of Fe 

or Zn 

and results in chlorosis expression in plants [20].

3. Ni 

TOXICITY IN PLANTS

At higher concentrations, Ni 

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 

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 

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 

deficiency and excess/toxicity in different crop plants are presented in Table 1.
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, chlorophyll, 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. radiate and V. mungo | 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] |
|               | Brassica napus L. | Decreases chlorophyll content, stomatal conductance and CO_{2} fixation | [9] |
|               | Helianthus annuus L. | Reduction in antioxidant enzymes activity | [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] |
|               | 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 (LHCCI) and the amounts of xanthophylls and carotenoids [44]. Nickel induced photosynthetic alterations include reduction in chloroplast size and numbers; disorganized chloroplast ultrastructure with the ablated 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$_2$O (10–20 g.m$^{-2}$). 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$_o$) by disrupting the structure of carriers and reaction center proteins such as plastoquinone (Q$_A$) [45]. At the cellular level, Ni$^{2+}$ also decreases the contents of cytochromes b$_{56}$ 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 perrniciously 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].
Plants Adaptation Strategies to Ni\textsuperscript{2+} stress

- Physical barriers
- Phytochelatins & Metallothiones
- N-containing compounds
- Growth regulators
- Antioxidant enzymes
- S-containing compounds

Cuticle Trichomes etc.

Proline Arginine Polyamines Abscisic acid Jasmonic acid Salsyllic acid SOD Catalase APOX GPOX

Glutathione H\textsubscript{2}S S-rich proteins Glucosinolates

Fig. 2. Various biochemical adaptation and tolerance strategies to Ni\textsuperscript{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 millimolar 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\textsuperscript{2+} [60].

Nasibi et al. [61] in their study on *Hyocyamus niger* found that Ni\textsuperscript{2+} showed decrease in chlorophyll a and total chlorophyll which was further maintained/recovered by Arginine pre-treatment in Ni\textsuperscript{2+} stressed plants. Pietrini et al. [62] in a study on *Amaranthus paniculatus* L. reported that the exposure of plants to increasing Ni\textsuperscript{2+} in the growth solution caused a significant increase in free polyamine content in roots and leaves of test plant at 25 μM NiCl\textsubscript{2}, whereas a decrease in the PAs (Spermidine and Spermine) content of plants at higher Ni\textsuperscript{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\textsuperscript{2+} accumulation in ryegrass (*Lolium perenne* L.) and maize (*Zea mays* L.) and reported 5 to 7 fold increased accumulation of Ni\textsuperscript{2+} in shoots of ryegrass than in maize grown at 20 to 80 μM Ni\textsuperscript{2+} whereas Ni\textsuperscript{2+} concentration in roots of ryegrass was found only 1 to 2 fold higher at 0.1 to 40 μM Ni\textsuperscript{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

| Biomolecules/structures | Plant system | Mechanism of tolerance | References |
|-------------------------|--------------|------------------------|------------|
| Trichomes               | Alyssum corsicum | Accumulate excess amount of Ni^{2+} in roots and shoots and also secrete secondary metabolites | [94] |
| Arbuscular mycorrhizal fungi/Glomalin Phytochelatins and Metallothionines | Festuca arundinacea | Alleviating Ni-induced stress by reducing Ni transport from roots to shoots | [95] |
| Proline                 | Thlaspi      | Binds to Ni^{2+} and sequesters it into vacuoles | [52] |
| Proline                 | 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 leaves and K^{+} content of root and shoot | [96] |
| Arginine                | Hyoscyamas niger | Counterbalance peroxidase and lipoxigenase activity of oxidative stress | [61] |
| Polyamines              | Hydrocharis dubia | Prevent Ni^{2+}-induced lipid peroxidation, electrolyte leakage and reduced Ni^{2+} accumulation | [97] |
| 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] |
| Ethylene                | Triticum aestivum | Induce the activities of enzymes (SOD, POD, CAT) | [98] |
| 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 NiSO₄. Ni²⁺ caused decrease in total SOD activity in both leaves and roots. The activity of APOX in leaves treated with 100 and 200 μM Ni²⁺ increased whereas in roots the enzymatic activity was reduced significantly. Catalase activity remained unaffected in both the organs in response to Ni²⁺. The activity of GST in Ni²⁺ 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 Ni²⁺ 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 Ni²⁺ exposure. JA with or without Ni²⁺ 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 Ni²⁺ stress as compared to control. Ali et al. [70] studied modulatory role of 24-epibrassinolide (EBL) in Brassica juncea exposed to NaCl and NiCl₂ 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 NiCl₂ 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 synthases (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 Ni²⁺-induced growth inhibition and oxidative stress (ROS) which suggests that high levels of GSH conferred tolerance to Ni²⁺-induced oxidative stress in Thlaspi Ni²⁺ 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 Ni²⁺ 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 Ni²⁺ toxicity. Thus, the involvement of MT2a, MT2c, MT2d and MT3 in Ni²⁺ homeostasis is evident from this study.

5.8 Ni²⁺ Phytoremediation

Phytoremediation of metal contaminated soil offers a low cost method for soil amendment. Several recent studies on Ni²⁺ hyperaccumulator plants have reflected their potential to sequester high levels of Ni²⁺ 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\textsuperscript{2+} hyperaccumulators plants have been identified, including members of the Acanthaceae, Asteraceae, Brassicaceae, Caryophyllaceae, Fabaceae, Flacourtiaeae, Meliaceae, Myristicaceae, Ochnaceae, Poaceae, Rubiaceae, Sapotaceae and Stackhousiaceae [76,77]. These above said families have higher requirements for Ni\textsuperscript{2+} as micronutrient (e.g. up to 500 mg Ni\textsuperscript{2+}/kg) than normal plants. The family with the most Ni\textsuperscript{2+} hyperaccumulator species is the Brassicaceae, with more than 80 species which are capable of accumulating Ni\textsuperscript{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, Eichornia, 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\textsuperscript{2+} can be transported quickly into shoots and leaves of hyperaccumulators and then sequestrated in the vacuole [81]. For these features, Ni\textsuperscript{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\textsuperscript{2+} in a wide range of physiological processes, starting from seed germination to the productivity. Moreover, without adequate supply of Ni\textsuperscript{2+}, plant life cycle cannot be completed and proves it as an essential micronutrient. Elevated levels of Ni\textsuperscript{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.

ACKNOWLEDGEMENTS

Authors are thankful to Vice Chancellor, C.S.J.M. University, Kanpur for permission to carry out the research work and extending necessary facilities.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Ali H, Khan E, Sazad MA. Phytoremediation of heavy metals – Concepts and application. Chemosphere. 2013;91:869-881.
2. Singh M, Kumar J, Singh S, Singh VP, Prasad SM, Singh MPVVB. Adaptation strategies of plants against heavy metal toxicity: A short review. Biochemical Pharmacology (Los Angel). 2015;4:161-167.
3. Assche FV, Clijsters H. Effects of metals on enzyme activity in plants. Plant Cell and Environment. 1990;13:195.
4. Lal N. Molecular mechanisms and genetic basis of heavy metal toxicity and tolerance in plants. In M. Ashraf, M. Ozturk and M.S.A. Ahmad (Eds.), Plant Adaptation and Phytoremediation, Springer-Verlag + Buisiness Media B.V., Dordrecht. 2010;35-58.
5. Izosimova A. Modeling the interactions between calcium and nickel in the soil-plant system. FAL Agricultural Research Special Issue. 2005;288:99.
6. Cempel M, Nikel G. Nickel: A review of its sources and environmental toxicology. Polish Journal of Environmental Studies. 2006;15:375-382.
7. Ali MA, Ashraf M, Athar HR. Influence of nickel stress on growth and some important physiological/biochemical attributes in some diverse canola (Brassica napus L.) cultivars. Journal of Hazardous Materials. 2009;172:964-969.
8. Pandey N, Sharma CP. Effect of heavy metals Co\textsuperscript{2+}, Ni\textsuperscript{2+} and Cd\textsuperscript{2+} on growth and metabolism of Cabbage. Plant Science. 2002;163:753-758.
9. Aziz E, Gad N, Badran N. Effect of cobalt and Ni on plant growth, yield and flavonoids content of Hibiscus sabdariffa L. Australian Journal of Basic and Applied Sciences. 2007;1:73-78.

10. Mulrooney SB, Hausinger RP. Nickel uptake and utilization by microorganisms. FEMS Microbiology Reviews. 2003;27:239-261.

11. Ragsdale SW. Nickel biochemistry. Current Opinion in Chemical Biology. 1998;2:208-215.

12. Lopez MA, Magnitskiy S. Nickel: The last of the essential micronutrients. Agronomia Colombiana. 2011;29:49-56.

13. Eskew DL, Welch RM, Cary EE. Nickel: An essential micronutrient for legumes and possibly all higher plants. Science. 1983;222:621-623.

14. Ureta AC, Imperial J, Ruiz-Argueso T, Palacios JM. Rhizobium leguminosarum biovar vicieae symbiotic hydrogenase activity and processing are limited by the level of nickel in agricultural soils. Applied and Environmental Microbiology. 2005;71:7603-7606.

15. Zobiole LHS, Oliveira RS Jr, Kremer RJ, Constantin J, Yamada T, Castro C, Oliveira FA, Oliveira A Jr. Effect of glyphosate on symbiotic N2 fixation and nickel concentration in glyphosate-resistant soybeans. Applied Soil Ecology. 2009;44:176-180.

16. Aydinalp C, Marinova S. The effects of heavy metals on seed germination and plant growth on alfalfa plant (Medicago sativa), Bulgarian Journal of Agricultural Science. 2009;15:347-350.

17. Sethy SK, Ghosh S. Effect of heavy metals on germination of seeds. Journal of Natural Science, Biology and Medicine. 2013;4:272-275.

18. Seregin V, Kozhevnikova AD, Kazyumina EM, Ivanov VB. Nickel toxicity and distribution in maize roots. Russian Journal of Plant Physiology. 2003;50:711-717.

19. Kopittke PM, Dart PJ, Menzies NW. Toxic effects of low concentrations of Cu on nodulation of cowpea (Vigna unguiculata). Environmental Pollution. 2007;145:309-315.

20. Khan MR, Khan MM. Effect of varying concentration of nickel and cobalt on the plant growth and yield of chickpea. Australian Journal of Basic and Applied Sciences. 2010;4:1036-1046.

21. Al-Qurainy F. Toxicity of heavy metals and their molecular detection on Phaseolus vulgaris (L.). Australian Journal of Basic and Applied Sciences. 2009;3:3025-3035.

22. Seregin IV, Kozhevnikova AD. Physiological role of nickel and its toxic effects on higher plants. Russian Journal of Plant Physiology. 2006;53:257-277.

23. Fageria NK, Baligar VC, Clark RB. Micronutrients in crop production. Advances in Agronomy. 2002;77:185-268.

24. Tack FMG. Trace elements; general soil chemistry, principles and processes. In P.S. Honda (Ed.), Trace Elements in Soils (pp. 9-37). Blackwell Publishing Ltd, John Wiley and Sons Publishing, Sussex, UK; 2010.

25. Page V, Feller U. Selective transport of zinc, manganese, nickel, cobalt and cadmium in the root system and transfer to the leaves in young wheat plants. Annals of Botany. 2005;96:425-434.

26. Riesen O, Feller U. Redistribution of nickel, cobalt, manganese, zinc and cadmium via the phloem in young and maturing wheat. Journal of Plant Nutrition. 2005;28:421-430.

27. Page V, Weisskopf L, Feller U. Heavy metals in white lupin: Uptake, root-to-shoot transfer and redistribution within the plant. New Phytologist. 2006;171:329-341.

28. Ma Y, Rajkumar M, Freitas H. Improvement of plant growth and nickel uptake by nickel resistant plant growth promoting bacteria. Journal of Hazardous Materials. 2009;166:1154-1161.

29. Panda GC, Das SK, Bandopadhyay TS, Guha AK. Adsorption of nickel on husk of Lathyrus sativus: Behavior and binding mechanism. Colloids and Surfaces. 2007;57:135-142.

30. Gajewska E, Sklodowska M, Slaba M, Mazur J. Effect of nickel on antioxidative enzyme activities, proline and chlorophyll contents in wheat shoots. Biologia Plantarum. 2006;50:653-659.

31. Hasinur R, Shamima S, Shah A, Shigenao KW. Effects of nickel on growth and composition of metal micronutrients in barley plants grown in nutrient solution. Journal of Plant Nutrition. 2005;28:393-404.

32. Shi GR, Cai QS. Photosynthetic and anatomic responses of peanut leaves to cadmium stress. Photosynthetica. 2008;46:627-630.
33. Kaveriammal S, Subramani A. Toxic effect of Nickel Chloride (NiCl₂) on the growth behavior and biochemical constituent of groundnut seedling (Arachis hypogaea L.). International Journal of Research in Botany. 2013;3:48-52.
34. Chen C, Huang D, Liu J. Functions and toxicity of nickel in plants: Recent advances and future prospects. CLEAN-Soil, Air, Water. 2009;37:304-313.
35. Sun EJ, Wu FY. Along-vein necrosis as indicator symptom on water spinach caused by nickel in water culture. Botanical Bulletin of Academia Sinica. 1998;39:255-259.
36. Rahman H, Sabreen S, Alam S, Kawai S. Effects of nickel on growth and composition of metal micronutrients in barley plants grown in nutrient solution. Journal of Plant Nutrition. 2005;28:393-404.
37. Poonkothai M, Vijayavathi BS. Nickel as an essential element and a toxicant. International Journal of Environmental Sciences. 2012;1:285-288.
38. Singh PP, Mall M, Singh J. Impact of fertilizer factory effluent on seed germination, seedling growth and chlorophyll content of gram (Cicer arietinum). Journal of Environmental Biology. 2006;27:153-156.
39. Talukdar D. Effect of arsenic-induced toxicity on morphological traits of Trigonella foenum-graecum L. and Lathyris sativus L. during germination and early seedling growth. Current Research Journal of Biological Sciences. 2011;3:116-123.
40. Roitto M, Raulto P, Julkunen-Titto R, Kukkola E, Huttenen S. Changes in the concentrations of phenolics and phytoalexins in Scots pine (Pinus sylvestris L.) seedlings exposed to nickel and copper. Environmental Pollution. 2005;137:603-609.
41. Seregin IV, Ivanov VB. Physiological aspects of cadmium and lead toxic effects on higher plants. Russian Journal of Plant Physiology. 2001;48:523-544.
42. Samantaray S, Rout GR, Das P. Tolerance of rice to nickel in nutrient solution. Biologia Plantarum. 1997;40:295-298.
43. Ahmad MS, Ashraf M, Hussain M. Phytotoxic effects of nickel on yield and concentration of macro-and micro-nutrients in sunflower (Helianthus annuus L.) achenes. Journal of Hazardous Materials. 2011;185:1295-1303.
44. Molas J. Changes in morphological and anatomical structure of cabbage (Brassica oleracea L.) outer leaves and in ultrastructure of their chloroplasts caused by an In vitro excess of nickel. Photosynthetica. 1997;34:513-522.
45. Bhalerao SA, Sharma AS, Poojari AC. Toxicity of nickel in plants. International Journal of Pure and Applied Biosciences. 2015;3:345-355.
46. Sreepanth TVM, Nagajyothi PC, Lee KD, Prasad TNVK. Occurrence, physiological responses and toxicity of nickel in plants. International Journal of Environmental Science and Technology. 2013;10:1129-1140.
47. Ghasemi F, Heidari R, Jameii R, Purakbar Boominathan R. Toxicity of nickel in plants. Journal of Stress Physiology and Biochemistry. 2012;8:55–61.
48. Siedlecka A, Krupa Z. Functions of enzymes in heavy metal treated plants. In M.N.V. Prasad and K. Strza1ka (Eds.), Physiology and Biochemistry of Metal Toxicity and Tolerance in Plants, Kluwer Academic Publishers. 2002;303-324.
49. Kabala K, Janicka-Russak M, Burzynski M, Klobus G. Comparison of heavy metal effect on the proton pumps of plasma membrane and tonoplast in cucumber root cells. Journal of Plant Physiology. 2008;165:278-288.
50. MadhavaRao KV, Sresty TV. Antioxidative parameters in the seedlings of pigeonpea ( Cajanus cajan (L.) Millspaugh) in response to Zn and Ni stresses. Plant Science. 2000;157:113-128.
51. Boominathan R, Doran PM. Ni-induced oxidative stress in roots of the Ni hyperaccumulator, Alysum bertolonii. New Phytologist. 2002;156:205–215.
52. Freeman JL, Persans MW, Nieman K, Albrecht C, Peer W, Pickering IJ, Salt DE. Increased glutathione biosynthesis plays a role in nickel tolerance in Thlaspi nickel hyperaccumulators. The Plant Cell. 2004;16:2176-2191.
53. Gomes-Juniora RA. Nickel elicits a fast antioxidant response in Coffea arabica cells. Plant Physiology and Biochemistry. 2006;44:420-429.
54. Gajewska E, Sklodowska M. Effect of nickel on ROS content and antioxidative
enzyme activities in wheat leaves. Bio Metals. 2007;20:27-36.
55. Nishida S, Tsuzuki C, Kato A, Aisu A, Yoshida J, Mizuno T. AtIR1, the primary iron uptake transporter in the roots, mediates excess nickel accumulation in Arabidopsis thaliana. Plant Cell Physiology. 2011;52:1433-3499.
56. Hall JL. Cellular mechanisms for heavy metal detoxification and tolerance. Journal of Experimental Botany. 2002;53:1-11.
57. Harada E, Kim JA, Meyer AJ, Hell R, Clemens S, Choi YE. Expression profiling of tobacco leaf trichomes identifies genes for biotic and abiotic stresses. Plant Cell Physiology. 2010;51:1627-1637.
58. Hauser MT. Molecular basis of natural variation and environmental control of trichome patterning. Frontiers in Plant Science. 2014;5:1-7.
59. Ashraf M, Foolad MR. Roles of glycine betaine and proline in improving plant abiotic stress resistance. Environmental and Experimental Botany. 2007;59:206-216.
60. Hayat S, Hayat Q, Alyemeni MN, Ahmad A. Proline enhances antioxidative enzyme activity, photosynthesis and yield of Cicer arietinum L. exposed to cadmium stress. Acta Botanica Croatica. 2013;2:323-335.
61. Nasibi F, Heidari T, Asrar Z, Mansoori HJ. Effect of arginine pre-treatment on nickel accumulation and alleviation of the oxidative stress in Hyoscyamus niger. Soil Science and Plant Nutrition. 2013;13:680-689.
62. Pietrini F, Iori V, Cheremisina A, Shevyakova NI, Radyukina N, Kuznetsov VV, Zacchini M. Evaluation of nickel tolerance in Amaranthus paniculatus L. plants by measuring photosynthesis, oxidative status, antioxidative response and metal-binding molecule content. Environmental Science and Pollution Research. 2015;22:482-494.
63. Shahid MA, Balal RM, Pervez MA, Abbas T, Aqueel MA, Javaid MM, Garcia-Sanchez F. Exogenous proline and proline-enriched Lolium perenne leaf extract protects against phytoxic effects of nickel and salinity in Pismum sativum by altering polyamine metabolism in leaves. Turkish Journal of Botany. 2014;38:914-926.
64. Yang XE, Baligar VC, Foster JC, Martens DC. Accumulation and transport of nickel in relation to organic acids in ryegrass and maize with different nickel levels. Plant and Soil. 1997;196:271-276.
65. Hossain MA, Piyatida P, Silva JATD, Fujita M. Molecular mechanism of heavy metal toxicity and tolerance in plants: Central role of glutathione in detoxification of reactive oxygen species and methylglyoxal and in heavy metal chelation. Journal of Botany. 2012;37. Article ID: 872875. DOI: 10.1155/2012/872875
66. Yadav G, Srivastava PK, Singh VP, Prasad SM. Light intensity alters the extent of arsenic toxicity in Helianthus annuus L. seedlings. Biological Trace Element Research. 2014;158:410-42.
67. Xu J, Yin H, Li X. Protective effects of proline against cadmium toxicity in micropropagated hyperaccumulator, Solanum nigrum L. Plant Cell Report. 2009;28:325-333.
68. Gajewska E, Sklodowska M. Antioxidative responses and proline level in leaves and roots of pea plants subjected to nickel stress. Acta Physiologiae Plantarum. 2005;27:329-339.
69. Sirhind CD, Mir MA, Sharma P, Gill SS, Kaur H, Mushtaq R. Modulatory role of jasmonic acid on photosynthetic pigments, antioxidants and stress markers of Glycine max L. under nickel stress. Physiology and Molecular Biology of Plants. 2015;21:559-565.
70. Ali B, Hayat S, Fariduddin Q, Ahmad A. 24-Epibrassinolide protects against the stress generated by salinity and nickel in Brassica juncea L. Chemosphere. 2008;72:1387-1392.
71. Lee S, Moon JS, Domier LL, Korban SS. Molecular characterization of phytochelatin synthase expression in transgenic arididopsis. Plant Physiology and Biochemistry. 2002;40:727-733.
72. Shukla D, Tiwari M, Tripathi RD, Nath P, Trivedi PK. Synthetic phytochelatins complement a phytochelatin deficient Arabidopsis mutant and enhance the accumulation of heavy metal(loid)s. Biochemical and Biophysical Research Communications. 2013;434:664-669.
73. Cobbett C, Goldsbrough P. Phytochelatins and metallothioneins: Roles in heavy metal detoxification and homeostasis. Annual Review of Plant Biology. 2002;53:159-182.
74. Ferraz P, Fidalgo F, Almeida A, Teixeira J. Phytostabilization of nickel by the zinc and cadmium hyperaccumulator Solanum nigrum L. are metallothioneins involved?
Plant Physiology and Biochemistry. 2012;57:254-260.

75. Prasad MNV. Nickelophilous plants and their significance in phytotechnologies. Brazilian Journal of Plant Physiology. 2005;17:113-128.

76. Chaney RL, Angle JS, McIntosh MS, Reeves RD, Li YM, Brewer EP, Chen KY, Roseberg RJ, Perner H, Synkowski EC, Broadhurst CL, Wang S, Baker AJ. Using hyperaccumulator plants to phytoextract soil Ni and Cd. Zeitschrift fur Naturforschung. 2005;60:190-198.

77. Reeves RD, Baker AJM, Borhidi A, Berezain R. Nickel-accumulating plants from the ancient serpentine soils of Cuba. New Phytologist. 1996;133:217-224.

78. Brooks RR, Morrison RS, Reeves RD, Dudley TR, Akman Y. Hyperaccumulation of nickel by Alyssum linnaeus (Cruciferae). Proceedings of Royal Society London B. 1979;203:387-403.

79. Mishra VK, Upadhayaya AR, Pandey SK, Tripathi BD. Heavy metal pollution induced due to coal mining effluent on surrounding aquatic ecosystem and its management through naturally occurring aquatic macrophytes. Bioresource Technology. 2008;99:930-936.

80. Rai PK. Heavy metal pollution in aquatic ecosystems and its phytoremediation using wetland plants: An ecossustainable approach. International Journal of Phytoremediation. 2008;10:131-158.

81. Milner MJ, Kochian LV. Investigating heavy-metal hyperaccumulation using Thlaspi caerulescens as a model system. Annals of Botany. 2008;102:3-13.

82. Ahmad MSA, Ashraf M. Essential roles and hazardous effects of nickel in plants. Reviews in Environmental Contamination and Toxicology. 2011;214:125-167.

83. Brown PH. Nickel. In A.V. Barker and D.J. Pilbeam (Eds.), Handbook of Plant Nutrition, CRC Press Taylor & Francis Group, Boca Raton, FL (USA). 2006;395–410.

84. Brown PH, Welch RM, Cary EE. Nickel: A micronutrient essential for higher plants. Plant Physiology. 1987;85:801–803.

85. Liu G, Simonne EH, Li Y. Nickel nutrition in plants; 2017 Available: http://edis.ifas.ufl.edu/hs1191 (Accessed on 27th May 2017).

86. Graham RD, Welch RM, Walker CD. A role of nickel in the resistance of plants to rust. Proc. 3rd Australian Agronomy Conference. Hobart Tasmania, Australia; 1985.

87. Ishtiaq S, Mahmood S. Phytotoxicity of nickel and its accumulation in tissues of three Vigna species at their early growth stages. Journal of Applied Botany and Food Quality. 2011;84:223-228.

88. Kovacevic G, Kastori R, Merkuloj LJ. Dry matter and leaf structure in young wheat plants as affected by Cd, lead, and nickel. Biologia Plantarum. 1999;42:119-123.

89. Dubey D, Pandey A. Effect of nickel (Ni) on chlorophyll, lipid peroxidation and antioxidant enzymes activities in black gram (Vigna mungo) leaves. International Journal of Science and Nature. 2011;2:395-401.

90. Sheoran IS, Singal HR, Singh R. Effect of cadmium and nickel on photosynthesis and enzymes of photosynthetic carbon reduction cycle in poinotea (Cajanus cajan L.). Photosynthesis Research. 1990;23:345-351.

91. Kazemi N, Khavari-Nejad RA, Fahimi H, Saadatmand S, Nejad-Sattari T. Effects of exogenous salicylic acid and nitric oxide on lipid peroxidation and antioxidant enzyme activities in leaves of Brassica napus L. under nickel stress. Scientia Horticulurae. 2010;126:402-407.

92. Khalid BY, Tinsley J. Some effects of nickel toxicity on rye grass. Plant and Soil. 1980;55:139-144.

93. Campanharo M, Monnerat PH, Espindula MC, Rabello WS, Ribeiró G. Toxicity symptoms of nickel in common bean. Revista Ciencia Agronomica. 2010;41:490-494.

94. Aydas SSB, Acik L, Leduc D, Adiguzel N, Ellialtioglu SS, Suludere Z, Kadioglu YK. Localization and distribution of nickel and other elements in in-vitro grown Alyssum corsicum exhibiting morphological changes in trichomes: initial insights into molecular mechanisms of nickel hyperaccumulation. Turkish Journal of Botany. 2013;37:1115-1124.

95. Shabani N, Sabzialian MR, Mostafavi-pour S. Arbucucular mycorrhiza affects nickel translocation and expression of ABC transporter and metallothionein genes in Festuca arundinacea. Mycorrhiza. 2018;26:67-76.

96. Mozafari H, Asrar Z, Rezanejad F, Pourseyedi S, Yaghoubi MM. Calcium and L-histidine interaction on growth
improvement of three tomato cultivars under nickel stress. Acta Biologica Szegediensis. 2013;57:131-144.

97. Zhao J, Shi G, Yuan Q. Polyamines content and physiological and biochemical responses to ladder concentration of nickel stress in Hydrocharis dubia (Bl.) backer leaves. BioMetals. 2008;21:665-674.

98. Siddiqui MH, Al-Whaibi MH, Ali HM, Sakran AM, Basalah MO, Al-Khaishany MYY. Mitigation of nickel stress by the exogenous application of salicylic acid and nitric oxide in wheat. Australian Journal of Crop Science. 2013;7:1780-1788.

99. Meindl GA, Ashman TL. Effect of floral metal accumulation on floral visitor communities: Introducing the elemental filter hypothesis. American Journal of Botany. 2015;102:379-389.

100. War AR, Paulraj MG, Ahmad T, Buhroo AA, Hussain B, Ignacimuthu S, Sharma HC. Mechanism of plant defence against insect herbivores. Plant Signaling and Behaviour. 2012;7:1306-1320.

101. Horger AC, Fones HN, Preston GM. The current status of the elemental defense hypothesis in relation to pathogens. Frontiers in Plant Science. 2013;4:395.