Bull Environ Contam Toxicol (2011) 86:1–17
DOI 10.1007/s00128-010-0171-1

Nickel: An Overview of Uptake, Essentiality and Toxicity in Plants

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Received: 1 November 2010 / Accepted: 29 November 2010 / Published online: 19 December 2010

Abstract Nickel even though recognized as a trace element, its metabolism is very decisive for certain enzyme activities, maintaining proper cellular redox state and various other biochemical, physiological and growth responses. Study of the aspects related with uptake, transport and distributive localization of Ni is very important in various cellular metabolic processes particularly under increased nitrogen metabolism. This review article, in core, encompasses the dual behavior of Ni in plants emphasizing its systemic partitioning, essentiality and ill effects. However, the core mechanism of molecules involved and the successive physiological conditions required starting from the soil absorption, neutralization and toxicity generated is still elusive, and varies among the plants.

Keywords Nickel · Transport · Distribution · Essentiality · Toxicity · Oxidative stress · Antioxidant system

Nickel (Ni) occurs abundantly in igneous rocks as a free metal or as a complex with iron. It stands at twenty-second position amongst most abundant elements in the earth crust (Sunderman and Oskarsson 1991). Swedish chemist Ronstadt in 1751 discovered Ni with an atomic number 28 and an atomic weight of 58.71 but it exists in a number of oxidation states. Ni^{2+} form is stable over a wide range of pH and redox conditions prevailing in the soil. In general, naturally occurring concentration of Ni in soil and surface waters is lower than 100 and 0.005 ppm, respectively (McGrath 1995). Additionally, anthropogenic activities further release Ni into the soil through various sources such as smelting, burning of fossil fuel, vehicle emissions, disposal of house hold, municipal and industrial wastes, metal mining, fertilizer application, and organic manures (Alloway 1995; Salt et al. 2000). However, majority of Ni released into the environment includes raw material used in metallurgical and electroplating industries, as a catalyst in the chemical and food industry and as a major component of electrical batteries (Easton 1992). During the last decades, Ni has become a serious concern as its concentration has reached up to 26,000 ppm in polluted soils (Alloway 1995; McGrath 1995) and 0.2 mg/L in polluted surface waters (Astros and Bjorklund 1996; Zwolsman and Van Bokhoven 2007) i.e. 20–30 times higher than found in unpolluted areas. The toxicity of Ni in plants has become a world-wide problem threatening sustainable agriculture as well. Ni, in contrast to other toxic trace (heavy) metals like cadmium, lead, mercury, copper and chromium has received little attention from plant scientists due to its dual character and complex electronic chemistry which is a major hurdle in disclosing its toxicity mechanism in plants. The critical toxicity level of Ni is more than 10 mg kg^{-1} dry mass (DM) in sensitive species (Kozlow 2005), >50 mg kg^{-1} DM in moderately tolerant species (Bollard 1983; Asher 1991) and >1,000 mg kg^{-1} DM in Ni hyper accumulator plants such as *Alyssum* and *Thalspi* species (Kupper et al. 2001; Pollard et al. 2002).

The impact of Ni toxicity on the physiology of plants depends on the type of plant species, growth stage, cultivation conditions, Ni concentration and exposure time (Krupa et al. 1993; Xylander and Braune 1994; Marschner 1995; Kabata-Pendias and Pendias 2001; Assuncao et al. 2003) in the soil. The toxic effects of higher concentration of Ni are observed at multiple levels, these include inhibition of mitotic activities (Rao and Sresty 2000), reduction

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in plant growth (Molas 2002), plant water relation and photosynthesis (Chen et al. 2009), inhibition of enzymatic activities as well as nitrogen metabolism (Gajewska et al. 2009), interference with the uptake of other essential metal ions (Chen et al. 2009), induction of oxidative stress (Chen et al. 2009). All of these alter physiological processes culminating ultimately in reduced fruit yield and quality (Gajewska et al. 2006).

Nickel in the Environment

In nature, Ni is mostly present in the form of nickelous ion, Ni^{2+}. The hydrated form as Ni $(H_2O)_6^{2+}$, is the most common form of Ni found in the soil solution. About 20% of the world's Ni supply comes from the soils of Ontario, Canada which is rich in Pentlandite, a Ni ore containing sulfide of Cu, Fe and Ni. The metal is extracted from its ore for various industrial, chemical and biological applications. Natural weathering of igneous and metamorphic rocks also releases Ni, which is largely retained in the weathered profile in association with clay minerals and as hydrous ions or as a complex with oxide of manganese. Free Ni concentration in the soil is controlled primarily by precipitation reactions with the hydrous oxides of Mn and Fe metals.

Ni also occurs in water bodies and in other atmospheres, usually in trace amounts. The release of municipal and industrial effluents significantly contributes Ni content to the soil and water but relative concentration depends on the source of effluent. Due to uncontrolled industrial and municipal discharges, some of the rivers in India and other countries are becoming highly polluted with Ni and other toxic metals, which sediment in the river bed to toxic levels. The concentration of Ni in river water and in sediments of upper Ganges (India), has been estimated to be between 35 and 211 and 70,900 and 511,000 ppm, respectively (Israili 1992). Ni is added into atmosphere primarily as pollutant particle, released along with other metals from the chimneys and air flows from metallurgical sites as well as cement clinkers (Orlov et al. 2002) which ultimately settle down on the soil, water or plant surfaces.

Nickel Uptake, Transport and Distribution in Plants

The uptake of Ni in plants is mainly carried out through the root system via passive diffusion and active transport (Seregin and Kozhevnikova 2006). The ratio of uptake between active and passive transport varies with the species, form of Ni and concentration in the soil or nutrient solution (Dan et al. 2002; Vogel-Mikus et al. 2005). The overall uptake of Ni by plants depends on the concentration of Ni^{2+}, plant metabolism, the acidity of soil or solution, the presence of other metals and organic matter composition (Chen et al. 2009). However, uptake of Ni usually declines at higher pH values of the soil solution due to the formation of less soluble complexes (Temp 1991). For example, the uptake of Ni^{2+} by *Lathyrus sativus* reportedly increased with increasing pH up to 5.0 and decreased as the pH is increased further up to 8.0 (Pandaa et al. 2007). Moreover, Ni^{2+} ion may also compete with other essential metal ions when it is absorbed by roots.

The uptake of heavy metals from the soil solution is strongly affected by calcium ion. Ca^{2+} lowered the absorption of Ni^{2+} in *Arabidopsis bertolonii*, an endemic plant of serpentine soils, but promoted Ni^{2+} absorption in *Berkheya coddii* (Gabbirielli and Pandolfini 1984; Boyd and Martens 1998). The inhibitory effect of various metal ions on absorption and translocation of Ni^{2+} from roots to shoots varied as $Fe^{3+} > Co^{2+} > Ca^{2+} > Mg^{2+} > NH_4^+ > K^+ > Na^+$ (Temp 1991). Besides being absorbed by roots, Ni can also enter into the plants via leaves. When a radioisotope of ^{63}Ni was applied on the leaves of *Helianthus annus*, 37% of the total amount was translocated to other plant organs (Sajwan et al. 1996). Similar trend was also observed when oat, soybean, tomato and egg plant leaves were sprayed with Ni salt solution (Hirai et al. 1993). The path of Ni transport in plants is from root to shoot (Peralta-Videaa et al. 2002) and makes an exit through transpiration stream (Neumann and Chamel 1986) via xylem. Organic acids and amino acids have frequently been reported to be the potential metal chelators, which most likely facilitate metal translocation through xylem (Cataldo et al. 1988; Briat and Lebrun 1999). Without being chelated by ligands, movement of metal cations from root to shoot is expected to be severely retarded as xylem cell walls have a high cation exchange capability.

During the last 30 years, numerous authors established that Ni in addition to its ionic form (Sagner et al. 1998) is translocated from root to shoot in several other forms (Homer et al. 1991; Kramer et al. 1996; Sagner et al. 1998; Persans et al. 1999). The complexes with citrate and malate are most widely distributed (Homer et al. 1991; Sagner et al. 1998). Besides this, Ni complexes with amino acids, like histidine (Persans et al. 1999; Kerkeb and Kramer 2003) and peptides (Cataldo et al. 1988) because Ni has high affinity wth imidazole ring (Martin 1986). The amount of these complexes considerably varies in diverse plant species (Kersten et al. 1980). *Alyssum lesbiscum*, a hyperaccumulator plant, was grown in 0.3 mM Ni $(NO_3)_2$ solution, the histidine content in the xylem sap increased 36-fold, whereas in *A. montanum*, a susceptible species, grown under the same conditions, histisine content did not increase (Kramer et al. 1996). Similarly increase in histidine content was also reported in *Brassica juncea* (Kerkeb

and Kramer 2003) and *Thalpsi caerulescene* (Assuncao et al. 2003) in response to Ni stress. Many Ni hyperaccumulators constitutively accumulate high concentrations of organic acids in their leaves (Kramer et al. 1996). Lee et al. (1977) found a linear correlation between Ni and citrate content in field-collected leaves of a variety of New Caledonian nickel hyper accumulator. At pH values typically found in the cytosol, amino acids, peptides and other metabolites have a higher affinity for metals than do for organic acids, therefore organic acids would not offer any protection from metal toxicity in the cytoplasm. In vacuoles and apoplast, at pH values typically below 6, co-ordination of metal cations with amino acids becomes less stable while co-ordination with organic acids, especially citrate, could decrease the chemical activity of the metal cations (Salt and Kramer 1999). Three distinct Ni^{2+} metallochaperones (metalloproteins that aid in the insertion of the appropriate metal ion into a metalloenzyme), including Hyp B, Coo J and Ure E proteins, have been identified in bacteria (Fu et al. 1995; Hausinger 1997; Kerby et al. 1997; Watt and Ludden 1998). It is likely that similar Ni binding proteins might also occur in plants. Recently, it was reported that yellow stripe-like proteins (YSLs) may act as transporters, particularly for Ni–Fe, in a metal hyperaccumulating plant, *Thlaspi caerulescens* (Gendre 2007).

Survey of literature reveals that distribution of Ni in plant tissues mainly deals with its localization in the shoots of hyperaccumulator plant species as evidenced through data obtained using histochemical (dimethyl glyoxime) methods (Heath et al. 1997; Sagner et al. 1998, 2007; Kupper et al. 2001; Bhatia et al. 2004), energy dispersive spectrometry and atomic absorption spectrometry (Nabais et al.1996; Psaras and Manetas 2001; Seregin et al. 2007), micro-processor induced X-ray emission spectrometry (Bhatia et al. 2004; Bidwell et al. 2004), scanning electron microscopy (Heath et al. 1997; Küpper et al. 2000), fluorescent and absorption-edge computed microtomography (McNear et al. 2005). There is less evidence indicating the distribution of Ni in the roots of excluders and hyperaccumulators (Seregin et al. 2003, 2007) though Cataldo et al. (1978) reported that over 50% of Ni absorbed by plants is retained in the roots. This may be due to the sequestration in the cation exchange sites of the walls of xylem parenchyma cells and immobilization in the vacuoles of the roots (Seregin and Kozhevnikova 2006). Moreover, in the roots a high percentage of Ni (over 80%) is present in the vascular cylinder, while less than 20% is present in the cortex (Chen et al. 2009). This distribution suggests high mobility of Ni in xylem and phloem (Marschner 1995; Page and Feller 2005).

In case of *Thlaspi pindicum*, a hyper accumulator, seeds collected from serpentine soils showed the presence of Ni in seed coat, with the maximum accumulation around micropyle and in the epidermis and lower amount in the mesophyll cells of cotyledons (Psaras and Manetas 2001). This evidence strengthens the understanding that Ni is accumulated in the leaf epidermis of the hyperaccumulator species *Hybanthus flouribundus* (Severne 1974), *T. montanum* (Heath et al. 1997), *Senecio coronative* (Raskin and Ensley 2000), *T. goesingense*, *Alyssum bertolonii*, and *A. lesbiacum* (Kupper et al. 2001), an in other *Alyssum* species (Broadhurst et al. 2004a, b). Using the radiolabelled Ni (^{63}Ni), it was demonstrated that following root absorption, Ni was highly mobile in soybean plants, with leaves being the mjor sink in the shoot, during vegetative growth. However, contrary to this, towards maturity, 70% of Ni in the shoot was remobilized to the seeds (Cataldo et al. 1978). They also studied the distribution of Ni in the resulting seeds and noted that 87.5% of the total Ni accumulated in cotyledons, followed by hull (8.5%) and embryo (4.0%). In a similar study in wheat, Ni quickly decreased in the older parts of the roots, moved to the newly formed parts of root system and also accumulated transiently in the expanding leaves (Page and Feller 2005). These findings suggested that Ni is a high mobile trace metal that tends to accumulate in newly formed plant parts as well as the seeds. In contradiction to these findings, Ni accumulation was more pronounced in roots rather than the shoot in barley (Brune and Deitz 1995) and maize (Baccouch et al. 2001). As the uptake of Ni predominates via roots, it is of primary importance to unravel the pattern of Ni distribution in the underground organs. A modified method, employing dimethylglyoxime, was used to demonstrate the concentration of Ni in all the tissues of maize root following 2 days of exposure. It was observed that all tissues homogenously possessed 35 μM of Ni. Moreover, irrespective of root region, Ni content in the cell protoplast exceeded that of the cell wall and the highest metal concentration was found in the endodermis and pericycle. It indicates that the endodermal cells do not block Ni translocation into the stellar tissue, as observed in case of Cd and Pb. After 7-day exposure to Ni, its content considerably increased in all the tissues of root as compared to 2-day exposure; however, the general pattern of Ni distribution in tissue did not change. In the longitudinal sections of root, large clusters of Ni-dimethylglyoxime crystals at the xylem vessels perforation were observed. Although, the mechanism of such a phenomenon is yet to be explored but it possibly indicates a mechanism for limiting Ni-transport into the shoots in the excluder plant species (Seregin et al. 2003). With the help of electron microscopic techniques, Ni accumulation in the cell wall and vacuoles in the cortical cells of onion root was also observed (Liu and Kottke 2003). Therefore, distribution of Ni in plant tissues is different from the characteristic pattern of certain other heavy metals, such as Cd and Pb where endodermis limits the

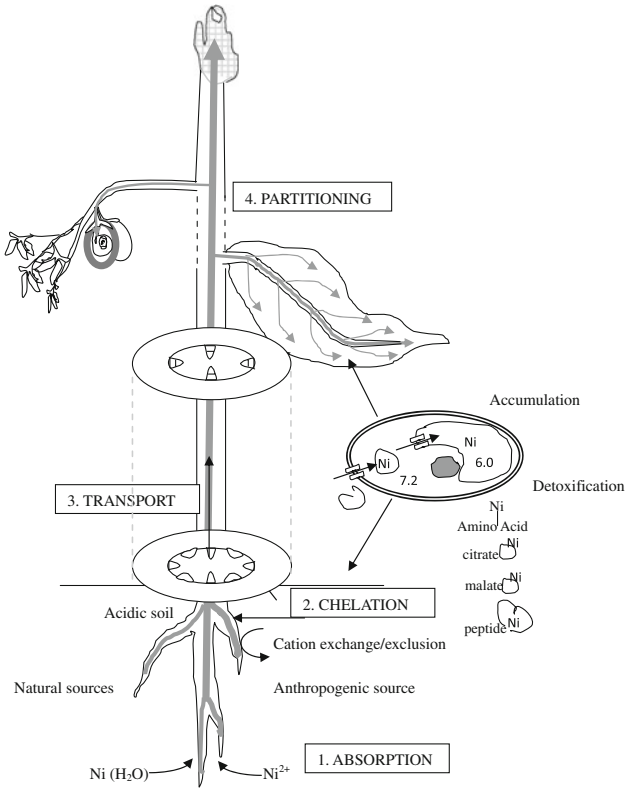

Fig. 1 Nickel uptake, transport and distribution in plants

movement of the later into the central cylinder. Contrary to this Ni is freely translocated into the stellar tissues and can easily reach the above ground parts of the accumulator plants, which might partly determine a specificity of its toxic effects (Fig. 1).

These contradictory observations give an impression that the distribution of Ni in various plant organs, after being absorbed by the roots is still debatable and the problem needs to be addressed in further details.

Essentiality of Nickel in Plants

A metal is considered as an essential nutrient in cases where plants cannot complete their life cycle in its absence and also it cannot be substituted with any other element (Eskew et al. 1983; Andreeva et al. 2001). The essentiality of Ni for higher plants is well documented (Eskew et al. 1983; Brown et al. 1987a, b; Marschner 2002). It was in 1987, when Ni was first established as an essential nutrient for the completion of the life cycle. Brown et al. (1987a, b) reported that Ni deficiency decreased the capacity of barley plants to develop viable seeds because of hindered embryo growth. Moreover, embryonic root was poorly developed or even failed to develop; in addition to this several other anomalies were also reported in the development of endosperm together with decreased activity of dehydrogenase. The critical Ni concentration in barley tissues that reduced the yield by 15% was 90 ± 10 ng/DM (Brown et al. 1987b). Besides this, Ni an important component of many enzymes, where it coordinates either with S-ligands and O-ligands (e.g. Urea), S-ligands (cystein residue e.g. hydrogenase) or ligands of tetrapyrol structure (Marschner 2002). However, urease is the only enzyme in higher plants that has been reported to possess Ni as an integral component, in stoichiometric quantities (Dixon et al. 1980).

In soybean, the activity of the enzyme urease depends on attachment with Ni which is facilitated by the product of two genes i.e. *Eu2 and Eu3*. The mutation in these genes resulted in the loss of urease activity. The product of later encodes a 32-kD protein which interacts with the product of *Eu2* in the course of embryonic urease activation. The interaction of these two auxillary proteins facilitates the attachment of Ni and hence the activity of urease. However, inactivation of the product of *Eu3* results into the nonavailability of Ni attachment to the enzyme, therefore the above evidence suggests that a sufficient quantity of Ni is essential for urease activation (Polaccao et al. 1999; Sirko and Brodzik 2000). A series of experiments with various plant species and growth media were conducted to demonstrate that Ni and urease are essential for plant vital functions. The deficiency of Ni content in the medium and the lower activity of urease, resulting from such a deficiency, upsets nitrogen metabolism and led to the accumulation of toxic level of a urea in shoots. Phenotypically such a process was manifested as necrosis in leaf tips or chlorosis in older leaves (Welch 1981; Eskew et al. 1983; Walker et al. 1985; Gerendas and Sattelmacher 1997a, b, 1999; Dalton et al. 1985; Ali et al. 2009). Similar necrosis of leaf tips was also observed following the decreasing levels of urea in soil. It was urea not ammonia that caused necrosis because the addition of urease inhibitor augmented necrosis (Krogmeier et al. 1989). Leaf injury was especially manifested in plant species that were capable to develop symbiosis with nitrogen fixing bacteria. In such species, nodule development in the roots lagged behind by 2 or 3 days (Eskew et al. 1983). The addition of Ni salts at low concentrations to the nutrient solution alleviated these symptoms. Ni could not be substituted by any other element, such as Al, Cd, Sn, V, Cr or Pb (Eskew et al. 1984). In some legumes, small amount of Ni is essential for root nodule growth and hydrogenase activation. The efficiency of nitrogen fixation depends on largely hydrogenase activity because the oxidation of hydrogen provides ATP required for N reduction to ammonia. Ni deficiency is known to lower the hydrogenase activity in the nodules. On contrary, when soybean plants grown in soil culture and irrigated once in 2 weeks with the nutrient solution containing 1 mM $NiCl_2$, at day 52 the hydrogenase activity in

nodules exceeded that of the control plants by about 45%, although the promoting effect disappeared apparently on approaching day 100, as a result of increasing Ni toxicity (Dalton et al. 1985). Recently, Bai et al. (2006) observed that the foliage of Ni-deficient seedlings exhibited metabolic disruption of nitrogen metabolism, via ureide catabolism, amino acid metabolism and ornithine cycle intermediates. Disruption of ureide catabolism in Ni-deficient plants resulted in the accumulation of xanthine, allantoic acid, ureidoglycolate, and littenlline, but total quantity of ureids, urea concentration and urease activity were reduced. Disruption of amino acid metabolism in Ni-deficient foliage results in accumulation of glycine, valine, isolucine, tyrosine, tryptophan, arginine and total free amino acids but lowered the concentration of histidine and glutamic acid. Ni-deficiency also disrupted the citric acid cycle and decreased the level of citrate. Moreover, lactic and oxalic acids accumulated as a result of disruption of carbon metabolism, thereby causing mouse ear, a key morphological symptom (Bai et al. 2006). The fact that activity of several enzymes depends on the presence of Ni ion that explains the promotional effects of low Ni concentrations on plant growth and development in certain species as zucchini, oilseed rape, cotton, sweet pepper, tomato, potato, and Chinese hemp (Welch 1981; Gerendas and Sattelmacher 1997a, b, 1999). Thus, spraying cotton plants with nickel sulphate solution (234.8 mg/kg) increased the number of buds, flowers, the rate of ball formation, and oil content of seeds (by 4.6%) (Andreeva et al. 2001). From the above reports, we are in position to conclude that Ni has vital roles in a wide range of physiological processes, starting from seed germination, through vegetative growth culminating ultimately to the seed development. Moreover, plants can not complete their life cycle without the adequate supply of this metal. Ni, therefore occupies a due position in a list of essential micronutrients (Marschner 2002).

Toxicity of Nickel in Plants

Plants rely on a range of transition (heavy) metals as essential micronutrients for normal growth and development. These elements are essential for most redox reactions which in turn, are fundamental to cellular functions. However, these transition metals including Ni, above the permissible limit, interfere with the functions of many cellular components, thereby, altering the normal metabolism, causing cellular injuries, and in extreme cases cause death of plant. Moreover, on the basis of toxicological and biochemical analysis, a large number of target molecules in the cells whose structure/activity is inhibited, modified or enhanced by transition metals (Ni) have been identified. According to Ochiai (1977) there are at least three events that play a pivotal role in generating toxicity by transition (heavy) metals including Ni. These are (a) displacement of essential components in the biomolecules by the metal (b) blocking of essential biological functional group of the molecules and (c) modification of enzyme/proteins, plasma membrane and/or membrane transporters structure/function. These enzymes and proteins contain several mercapto ligands close enough to chelating structures with the metal, and thereby lose their functional property. Beside this, heavy metals also generate oxidative stress, mediated through the generation of free radicals (Di Toppi and Gabbrielli 1999; Schutzendubel and Polle 2002). The toxic symptoms generated by Ni include chlorosis, necrosis, inhibition of shoot and root growth and decrease in leaf area (Shaw et al. 2004). In the following section, several metabolic and physiological processes affected by Ni in plants are being reviewed.

Growth and Development

Plant growth and development are essential processes of life and propagation of species. They are continuous and mainly depend on external resources present in soil and/or air. Growth is primarily expressed as a function of genotype and environment, which consists of external and internal growth factors. Presence of excess Ni in the external environment leads to changes in the growth pattern and development of plants. These effects are summarized in Table 1.

Seed Germination and Seedling Growth

Seed germination and subsequent seedling growth are the initial events in the life of a plant, projecting the extent of future physiological and biochemical processes. Seed germination is the most resistant phase to heavy metals (Seregin and Kozhevnikova 2006). However, there have been many reports on the toxic effects of Ni on germination and seedling growth in plants. Zhang et al. (2007) reported that leaves having high amount of Ni, shed by the Ni hyperaccumulator *Alyssum murate,* inhibit seed germination of the competing plants. The germination of pigeonpea decreased by 20% in a 1.5 mM solution of Ni and the germination percentage decreased in proportion to the concentration of Ni (Rao and Sresty 2000). 42 day-old cabbage plants exposed to 0.5 mM of Ni for 8 days did not produce any noticeable difference in growth, but their subsequent growth was retarded (Panday and Sharma 2002). Growth of *Zea mays* L. seedlings decreased with the increasing concentration of Ni as compared to control (Bhardwaj et al. 2007). Application of 100 and 200 μM Ni reduced the wheat shoot mass by 20% and 26% below the

Table 1 Effects of nickel on plant growth and development

Nickel concentration	Crop/plants	Site of action	Reference
100 mM	Onion	Inhibition of root growth	Liu et al. 1994
1–10 mM	Onion roots	Expanded nuclei (histochemical study)	Liu et al. 1994
>10 mM	Onion roots	Nuclei attained irregular shape	Liu et al. 1994
200 mM	Barley seedlings	Root biomass severely affected	Brune and Dietz 1995
400 mM	Barley seedlings	Not survived	Brune and Dietz 1995
Ni alone and/or in combination with Pb, Zn, Cu, Cd and Cr	Root system of maize	Reduction in dry matter production	Baccouch et al. 1998
>50 mM	Soybean seedlings	Fresh and dry mass of both root and shoot	El-Shintinawy and El-Ansary 2000
100 μM	Wheat	Decrease fresh weight of root	Gajewska et al. 2009

control level, respectively (Gajewska and Sklodowska 2008). Germination of seeds and seedling growth of *Brassica juncea* were significantly reduced by Ni (25, 50, 100 mg dm^{-3}) treatment (Sharma et al. 2008). Moreover, the roots of *Nicotiana tabacum* became dark brown within 7–10 days of exposure to Ni (0.43 mM) and the growth of plants was severely inhibited (Boominathan and Doran 2002). The reason for the above toxic effect of Ni on seed germination and seedling growth might be due to ill effect generated by Ni on varied metabolic processes, poor elasticity of cell walls and disturbed cell proliferation (Tripathy et al. 1981; Seregin and Kozhevnikova 2006) as well as the suppression of the activity of hydrolytic enzymes (Walker et al. 1985).

Root Growth

Since roots are the primary target of metal anions, their growth is usually more severely affected than that of the aerial parts (Panday and Sharma 2002). In excluder species which accumulate Ni mostly in their roots, root growth is inhibited more heavily than the growth of shoots (Seregin et al. 2003; Samantaray et al. 1997) and therefore root test is widely used for evaluating the toxicity of various toxicants including heavy metals (Seregin and Ivanov 2001; Seregin and Kozhevnikova 2006). Unlike root growth, lateral root initiation is very resistant to most heavy metals (Seregin and Ivanov 2001; Ivanov 1994), due to the endodermal barrier and the characteristics structure of central cylinder (Seregin and Ivanov 1997, 1998). However, the number of lateral roots decreased considerably in rice and maize and apparently Ni^{2+} could cross the endodermal barrier and got accumulated in the cells of pericycle (Samantaray et al. 1997; Seregin et al. 2003).

Treatment of wheat seedling with 100 and 200 mM Ni reduced root growth by 37 and 53%, respectively, over the control (Gajewska and Sklodowska 2008). Exposure of the wheat seedling to excess Ni caused a rapid accumulation of this metal in roots (Gajewska et al. 2006). Moreover, Ni (10 μM) had no significant effect on root elongation in wheat seedlings whereas exposure of seedlings to 200 μM of Ni, a significant inhibition of root elongation was observed (Gajewska et al. 2006). In *Brassica juncea* plants, length of root decreased by 33% in the presence of 100 μM Ni (Alam et al. 2007).

Stem Growth

It is evident from available literature that heavy metals particularly Ni affects plant growth at cellular, organ and the organism level (Sheoran et al. 1990a; Bishnoi et al. 1993). However, Ni has been tested in a limited number of studies, thus the information available on this topic is scarce. We have tried to establish a relationship between Ni and stem growth in Table 2.

Stem growth (plant growth) inhibited by Ni and other heavy metals results from general metabolic disorders and immediate inhibition of cell division. However, it is not clear whether Ni enters cell nuclei at high concentrations and, if it does, how important is immediate interference of Ni with DNA and nuclear proteins. By discussing these issues, we will be able to give some insight of Ni toxicity and its after effects on plant growth and morphogenesis.

Leaf Growth

Leaf growth, leaf area and total leaf number decisively determines the yield of crops. In the absence of Ni, plants develop specific deficiency symptoms, like leaf tip burn due to excessive accumulation of urea whereas its excess has been reported to cause leaf necrosis and chlorosis of plants (Van Assche and Clijsters 1990; Mcllveen and Negusanti 1994; Marschner 1995; Seregin and Kozhevnikova 2006). Exposure to 0.085–0.255 mM (5–15 ppm) Ni, for a week, developed chlorosis and necrosis along the veins in newly developed leaves of water spinach (Sun and Wu 1998). Ni at a concentration of 0.5 mM produced dark brown necrotic spots along the leaf margins and decreased water

Table 2 Effect of nickel on stem growth in different plants

Nickel concentration	Crop/plant	Effect	Reference
100 μM $NiSO_4$	*Triticum aestivum*	Decrease mesophyll thickness, size of vascular bundle, and width of epidermal cells	Kovacevic et al. 1999
200 μM Ni	Wheat seedlings	Lowered shoot length by 44%	Gajewska et al. 2006
>50 mM Ni	Soybean seedlings	Decrease the fresh and dry mass of the plant	El-Shintinawy and El-Ansary 2000
100 μM Ni	*Triticum aestivum*	Reduced shoot growth appearance of chlorosis and necrosis	Gajewska and Sklodowska 2007

potential and transpiration rate, resulting in the wilting of outer leaves (Panday and Sharma 2002). Similarly, barley grown in 0.1 mM Ni for 14 days had foliar chlorosis and necrosis (Rahman et al. 2005). Bashmakov et al. (2006) also observed a significant decrease in leaf area even at lower doses (50 and 0.1 mM) of Ni. Possibly delay in seed germination is caused by the inhibitory action of Ni ions on the processes governing its metabolism and elongation of the resulting embryonic axis, as Ni is known to inhibit cell division and their elongation (Seregin and Ivanov 2001). Similarly, deformation of leaves is apparently an expression of the irregular cell elongation. It may be suggested that leaf growth traits might serve as suitable bioindicators of heavy metal pollution and in the selection of resistant plant species.

Total Dry Matter Production

The first prerequisite for higher yields in plants is an increase in biomass production in terms of dry matter. Carbon compounds account for 80%–90% of the total dry matter produced by planst. Larger source size and improved photosynthetic process was found to be the basis for the building up of organic substances and dry matter production. In a study conducted on *Brassica juncea* to evaluate the Ni accumulation and toxicity in relation to biomass production, it was found that 100 μM Ni decreased dry mass of the plants (Alam et al. 2007). The other important characteristics determining total dry matter production is ratio of shoot to root mass. It was revealed that at low concentrations of Ni^{2+} (10 and 50 μM) shoot to root ratio increased in maize seedlings (Baccouch et al. 2001).

The important parameters contributing to total dry matter production are fresh and dry mass. According to Bashmakov et al. (2006), 21 day old maize plants showed significant loss of fresh mass at even 10 μM Ni. Moreover, an increase in Ni concentration caused almost a linear decrease in shoot and root fresh and dry mass of plants. The water content both of root and shoot significantly decreased at 0.1 mM and 50 μM of Ni^{2+}, that possibly is the main reason assigned for the loss of fresh and dry mass of the maize seedlings.

Physiological Processes

The toxic effects of Ni relating to various physiological processes are summarized in Table 3.

Photosynthesis

Photosynthesis is of the fundamental basis of competitive success in green plants and the principal organ of photosynthesis is leaves of higher plants. In case of heavy metals, it is well established that several direct and indirect paths are known which lead to non-specific metal inhibition of photosynthesis. However, the influence of Ni on photosynthesis is pervasive occurring both in isolated chloroplasts and in the intact plants (Tripathy et al. 1981; Singh et al. 1989; Molas 2002; Boisvert et al. 2007). Almost, at both the levels, Ni damages the photosynthetic apparatus/machinery, including the destruction of mesophyll cells and epidermal tissues (Bethkey and Drew 1992) and decreases the chlorophyll content (chlorophyll a, b, total chlorophyll and chlorophyll a/b ratio) (Gajewska et al. 2006; Ahmad et al. 2007; Alam et al. 2007; Gajewska and Sklodowska 2007). Ni damages the structure of thylakoid membranes and the structure of grana (Szalontai et al. 1999; Molas 2002), reducing the size of grana and increasing the number of non-appressed lamellae (Molas 1997).

The primary functional mechanism of heavy metal toxicity is the displacement of essential ions e.g. Ni displaces Mg ion (Van Assche and Clijsters 1986), therefore, alters the structure and/or activity of chlorophyll molecule (Kupper et al. 1996, 1998) and that of ribulose-1,5-biphosphate carboxylated oxygenase (Van Assche and Clijsters 1986). It also interferes with the photosynthetic electron transport chain (Tripathy et al. 1983; Mohanty et al. 1989) and the availability of its intermediates (such as cytochromes b6F and b559) in leaves (Sheoran et al. 1990b; Krupa et al. 1993). The inhibition of electron transport is mainly on the donor side of photosystem II (PS II) (Tripathy et al. 1983; Singh et al. 1989) and the binding site for Q_B, the secondary quinone acceptor of PS II (Mohanty et al. 1989; El-Sheekh 1993). Moreover, studies

Table 3 Effect of nickel on various physiological processes

Plant/crops	Ni concentration and duration	Effect	References
Phaseolus vulgaris	200 mM Ni for 24 h	Increased stomatal resistance	Rauser and Dumbroff 1981
Barley	500 mM Ni	Decreased the ratio of chlorophyll a:b	Shalygo et al. 1999
Cajanus cajan	1 mM $NiCl_2$	Inhibition of key enzymatic activities of the calcium cycle	Sheoran et al. 1990b
Brassica juncea	100 µM	Decreased chl. content and net photosynthetic rate	Alam et al. 2007
Triticum aestivum	100 µM Ni for 6 days	Decreased 48% chlorophyll content	Gajewska and Sklodowska 2007
Triticum aestivum	100 µM Ni for 9 days	Decreased 61% chlorophyll content	Gajewska and Sklodowska 2007
Triticum aestivum	100 µM Ni for 7 days	Decrease protein content	Gajewska et al. 2009
Triticum aestivum	100 µM Ni	Decrease chlorophyll content (SPAD) and net photosynthetic rate in all five test cultivars	Yusuf et al. 2010

on photosynthetic protein complexes, in vivo studies showed that Ni mainly inactivates photosystem I (PS I) (Singh et al. 1989), whereas, in vitro studies, it primarily targets PSII (Tripathy et al. 1981). A recent in vitro study on spinach leaves discloses that two proteins associated with the oxygen evolving complex of PS II (the extrinsic 16 and 24 kD polypeptides) were depleted under Ni deficiency which recover following the treatment with 1 mM of Ni (Rao and Sresty 2000).

All the impaired events discussed above lead to a loss in photosynthetic rate as Ni has a wide range of toxic effects on electron transport system, chlorophyll pigments degradation/synthesis and the enzymatic machinery involved in photosynthesis.

Water Relations

If transpiration (water relation) is eliminated without stopping photosynthesis, injury from drought would occur and crop plants will not thrive in large areas that are semi-desert. Heavy metals block water transport from roots to the above ground parts leading to a severe dehydration of shoots (Haag-Kerwer et al. 1999; Chen et al. 2004). In general, transition (heavy) metals are known to alter the water relation of plants (Barcelo and Poschenrieder 1990; Prasad 1997) where the effect may be at multiple points such as water uptake, water movement through symplast and apoplast and stomatal functioning (Barcelo and Poschenrieder 2004). However, the stability of plant water pool depends on the balance between water uptake and transpiration. Many authors reported that Ni induced the decline in plant transpiration rate and water content (Sheoran et al. 1990b; Schickler and Caspi 1999; Bishnoi et al. 1993; Molas 1997).

Bishnoi et al. (1993) reported that in 4 day old plants of *Triticum aestivum* in sand culture, with 10 mM Ni added to the nutrient solution, leaf water potential, stomatal conductance, transpiration rate and total moisture content decreased. The toxic effect of Ni^{2+} on plant growth decreased the area of leaf blades, the major transpiring surface (Chen et al. 2009). Such a decrease in leaf area by 40% was reported in *Cajanus cajan* plants, grown in sand with 1 mM Ni added to the nutrient solution (Sheoran et al. 1990b) and also in *Brassica oleracea* plants, grown in the presence of 5.20 g/m^3 $NiSO_4 \cdot 7H_2O$ in agar medium (Molas 1997). The density of stomata may be the reason to be assigned for the loss of transpiration under the stress of Ni but the observations are quite contradictory. The number of stomata may decrease (Molas 1997) or increase (Breckle and Kahle 1991) because of the reduction in leaf area and the size of the epidermal cells. Moreover, closing of stomatal aperture is the primary response of plants exposed to heavy metals that lowers transpiration rate (Molas 1997; Seregin and Ivanov 2001). The presence of Ni in *Phaseolus vulgaris* leaf tissues was shown to elevate the level of ABA, which is known to bring about stomatal closure (Molas 1997). The cumulative effect of diminished transpiration, stomatal closure and elevated ABA level under metal (Ni) stress, could have lead to a shift in the water relation of plants.

Mineral Nutrition

It is well documented that Ni along with other nutrients such as K, Na, Ca, Mg, Fe, Cu, Zn and Mn is necessary for plant growth (Taiz and Zeiger 2006) and has therefore, been placed in the list of micronutrients. However, the interaction between the toxic heavy metals and other essential plant nutrients determining their availability and uptake needs to be debated. Ni has some similar characteristics to Ca, Mg, Mn, Fe, Cu and Zn. Therefore, Ni may compete with these minerals in absorption, uptake and their subsequent utilization in the plant system (Barcelo and Poschenrieder 1990; Rubio et al. 1994; Chen et al. 2009). As a consequence of competition, Ni, above a threshold level, may inhibit the absorption of these metals, decrease

their concentration and even lead to their deficiency in plants (Van Assche and Clijsters 1990; Rubio et al. 1994; Ahmad et al. 2007). Subsequently, this may lead to perturbation in physiological and biochemical processes, and ultimately results in toxic effects (Genrich et al. 1998; Gajewska et al. 2006; Goncalves et al. 2007) For example, Ni in addition to other toxic heavy metals (Cd, Cr, Co, Zn and Pb) is reported to cause Fe deficiency either by retarding its uptake or by causing its immobilization in roots (Mysliwa-Kurdziel et al. 2004). This may cause retardation of germination, growth suppression, and reduction in yields (Chen et al. 2009). These inhibitory effects of Ni can be partially overcome by additional supplementation of Mg (or Fe) ions (Genrich et al. 1998; Goncalves et al. 2007). In barley plants, the toxic concentration of Ni significantly reduced the contents of Ca, Fe, K, Mg, Mn, P and Zn, both in leaves as well as in roots (Brune and Deitz 1995).

The excess quantity of Ni declined the level of nitrogen in the leaves and roots of chickpea and mungbean. However, the toxicity was more severe in cases where Ni was supplemented in combination with other heavy metals namely Cd, Co, Pb, Zn and Cu (Athar and Ahmad 2002). These authors also noted that the concentration of nitrogen declined more sharply in the shoot than in the roots. The phosphorus content in *Helianthus annus* and *Hyptis suaveolens* also exhibited a significant decrease in response to Ni treatment; it was attributed to an increase in the activity of acid phosphatase and ATPase (Pillay et al. 1996). Many enzymes, e.g. superoxide dismutase (SOD) and catalase (CAT) are metalloenzymes that contain Fe, Cu, Zn or Mn in their prosthetic group. Since excess Ni has been shown to decrease the contents of Fe (Panday and Sharma 2002), Cu and Zn (Parida et al. 2003) in plant tissues, it can therefore be speculated that Ni might have reduced the biosynthesis of these metalloenzymes by causing deficiencies of these essential metals (Gajewska et al. 2006) as heavy metals are recognized to alter the membrane structure and its functions (Schutzendubel and Polle 2002). These alterations at the level of membrane are supposed to cause the real hindrance in the mineral uptake and further translocation to the adjoining tissues and to the sink.

Effect on Metabolites

In soybean seedlings, Ni (200 mM) induced the accumulation of all free amino acids in roots in association with a decrease in alanine aminotransferase and aspartate aminotransferase, reflecting the accumulation of both alanine and aspartic acid. Moreover, phytochelatin constituting amino acid (cystein) accumulated in a huge quantity (17.5% of total free amino acid pool), both in root and shoot (El-Shintinawy and El-Ansary 2000). Accumulation of proline in response to Ni treatment has been found in cabbage (Panday and Sharma 2002), soybean (Prasad et al. 2005; Mishra and Agrawal 2006), pea (Gajewska and Sklodowska, 2005), wheat (Gajewska et al. 2006, 2009), and in rice plants (Maheshwari and Dubey 2007). The Ni-treatment decreased the level of proteins and carbohydrates in sunflower and *Myplis snavelus* (Pillay et al. 1996), maize (Baccouch et al. 2001) and soyabean (El-Shintinawy and El-Ansary 2000). In legumes, Ni is reported to affect the symbiotic association with *Rhizobium* (Athar and Ahmad 2002) which consequently decreased the rate of nitrogen fixation and hence nitrogen content in plants.

The effects of heavy metals on plants may be the result of their direct effect on membranes and the photosynthetic apparatus and/or their indirect effect by altering some signaling pathways (Maksymiec 2007). The increased synthesis of secondary metabolites (Schutzendubel and Polle 2002) and pathogenesis-related proteins, connected with increased energy consumption, may cause the slowdown of basic metabolites and plant productivity (Maksymiec 2007).

Effect of Ni on Plasma Membrane and Lipid Peroxidation

Plasma membrane is the first functional part of the plant cell that comes in contact with toxic transition (heavy) metals where these ions alter the membrane fluidity and structural conformation of membrane bound enzymes (e.g. ATPase) and their activity (Ros et al. 1990, 1992). Ni has also been reported to decline the activity of membrane bound ATPase (Ros et al. 1990, 1992) thus affecting the solute mobility across the membrane (Cakmak and Horst 1991; Yang et al. 1996). These modifications are believed to be the consequence of conformational changes in ATPase, brought about by the metal directly or thoroughly by the changes in the lipid composition, associated with the membrane (Vuletic and Kohler 1990; Strass and Horst 1995). Changes in the plasma membrane integrity/structure resulting from alterations in lipid composition have been observed in rice plants, exposed to Cd and Ni (Ros et al. 1992). In addition to plasma membrane, the heavy metals also adversely affect thylakoid membrane, both structurally and functionally. These modifications are brought about by the changes in lipid content, its composition and peroxidation of chloroplast membrane (Devi and Prasad 2004). Similar observations have also been reported in Ni-stressed maize plants (Baccouch et al. 2001). Thus the membrane dysfunction induced by metals, including Ni, could be due to changes in the level of membrane lipids and/or membrane lipid peroxidation.

Enzymes and Other Compounds

Nitrate Reductase

The values for nitrate reductase (NR) activity in leaves decreased significantly over the control in the presence of different heavy metals such as Cd (Hasan et al. 2008), Cu (Fariduddin et al. 2009), and Ni (Alam et al. 2007). Ni concentrations up to 100 μM resulted in significant inhibition of NR activity in *Brassica juncea* (Alam et al. 2007). Wheat seedling treated with 100 μM Ni showed sharp decline in NR activity (Yusuf et al. 2010). Ni also inhibited the activity of NR in soybean (El-Shintinawy and El-Ansary 2000) which supplies the organic nitrogen to the plants. The above reports revealed that at high Ni level (100 μM), a significant decrease in NR activity was observed that was more significant in leaves than roots. Here the Ni might be hindering the mechanism of nitrogen uptake by plant roots. Both the uptake and transport of nitrates into the cells depend on the availability of metabolic energy, utilized for cell membrane polarization. The main role in this process is played by H^+-ATPase proton pump (McClure et al. 1990a, b). An inhibition of NO^{3-} uptake can result from the action of Ni on H^+- ATPase pump, though it can also affect the carrier of H^+/NO_3^- symport. Moreover, proteins of the NO_3^- uptake system contain -SH groups, and due to that they are sensitive to heavy metals including Ni (Singh et al. 1989; Tan et al. 2000). It is also suggested that NR inhibition is likely to occur because of reduced supply of NADH. This might result from disorganization of chloroplasts, reduced rate of photosynthesis and respiration, NADH oxidation or reduction in NO_3^- supply to the site of the enzyme as a consequence of water stress induced by the heavy metal, or direct effect of heavy metals on protein synthesis as they have a strong affinity for functional –SH group of enzyme (Rai and Rai 1997; Singh et al. 1989; Ahmad and Abdin 1999; Gouia et al. 2000).

Nitrogen Metabolism

Nitrogen is often a limiting nutrient factor for plants; even though molecular nitrogen is abundant in the atmosphere (~79%) but plants do not have the genes, coding the enzymes for the fixation of inert dinitrogen and therefore depend on the nitrogen-fixing activities in the soil or the atmosphere. Nitrogen-fixing organisms of the soil may be free living or form a symbiotic association between the bacteria and the host plant. The invading rhizobia induce the formation of root nodules, where leghaemoglobin ensures a low oxygen environment in which the enzyme dinitrogenase can function. The host plant provides energy in the form of photosynthate and, in exchange, receives a supply of combined nitrogen for its own growth and development.

The product after nitrogen fixation is ammonium, which is rapidly incorporated to form amino acids through the activity of enzyme complex GS/GOGAT, before being exported out of the nodule. P II proteins sense cellular carbon/nitrogen balance and regulate GS/GOGAT activity. Plants that do not form nitrogen-fixing associations with bacteria generally take up nitrogen in the form of nitrate from the soil. Nitrate must first be reduced to ammonia in the plant before it can be incorporated into organic molecules. In photosynthesizing leaves, reducing power is generated that reductes nitrate to NH_4^+.

Gajewska et al. (2009) reported that treatment of wheat seedlings with 100 μM Ni led to a decrease in NR activity, without altering its the activation state. Decline in NiR activity was more pronounced than that of NR after the application of 100 μM Ni. Moreover, the activities of GS and NADH-GOGAT also showed substantial decrease in response to Ni stress with the latter being more susceptible to the metal. In another study, exposure of wheat seedlings to Ni led to the alteration in nitrogen metabolism in the shoots resulting in the accumulation of ammonium and proline in these organs. Induction of NADH-GOGAT and NADH-GDH activities and glutamate producing aminotransferase activities might compensate for the decreased Fd-GOGAT activity, serving as alternative means of glutamate synthesis.

Plasma Membrane H^+-ATPase

One of the membrane bound enzymes which is altered in metal stressed plants seems to be H^+-ATPase. This is the only proton pump operating in plasma membrane, playing a crucial role in the regulation of ion homeostasis. It was observed that the hydrolytic activity of H^+-ATPase in roots of different plants was inhibited by Cd (Kennedy and Gonsalves 1989; Fodor et al. 1995) as well as by Cu (Burzynski and Kolano 2003). Treatment of cucumber seedlings with heavy metals (Cd, Cu and Ni) changed the hydrolytic and transporting activities of plasma membrane bound H^+-ATPase (Janicka-Russak et al. 2008). Higher concentrations of Cd, Cu and Ni (100 μM) in nutrient solution caused a distinct inhibition of H^+-ATPase activity. The greater inhibition (about 60%) of ATP hydrolysis was observed in plasma membrane isolated from cucumber roots, treated with Cd, while in the case of Cu and Ni it was 45 and 20%, respectively. Inhibition of the plasma membrane proton pump in root cells under heavy metal stress could also result from the alteration at the transcriptional as well as the translational levels of the cells.

Increasing evidences indicate that, besides the genetic regulation of the proton pump, its activity might also be

modulated post-translationally at the protein level, mainly through reversible phosphorylation (Schaller and Sussman 1988a, b; Portillo 2000). This important role in the regulation of the plasma membrane bound H^+-ATPase has its autoinhibitory domain in the C-terminal region of the enzyme (Jahn et al. 1997; Baunsgaard et al. 1998; Camoni et al. 1998).

Glutathione Reductase

Chloroplast, cytoplasm and mitochondria of higher plants are the base for glutathione reductase (GR) where it catalyzes the NADPH-dependent reduction of disulphide bond of oxidized glutathione resulting in the generation of GSH. It is involved in defence against oxidative stress, where it plays an important role within the cell system, which includes participation in the ascorbate–glutathione cycle in the breakdown of H_2O_2 (Azevedo et al. 1998), maintenance of the sulfhydryl groups of cysteine in a reduced form, storage of reduced sulphur and a substrate for glutathione-S-transferases (Noctor and Foyer 1998). Some transition (heavy) metals at higher concentration could induce transient depletion of GSH and an inhibition of antioxidative enzymes, especially GR.

GR activity in response to Ni stress is often found to be dose dependent and variable over time and this increased activity helps in maintaining glutathione in the reduced form, prior to its incorporation into phytochelatins, and/or the activation of the ascorbate glutathione cycle operating in order to detoxify the ROS, induced on exposure to Ni. Increase in GR activity in response to Ni has also been reported in a number of plants such as wheat, *Alyssum* species (Schickler and Caspi 1999) and *Cajanus cajan* (Rao and Sresty 2000). On contrary, in roots and shoots of *Crotalaria juncea*, GR activity was drastically reduced (Cardoso et al. 2005), whereas, in maize plants GR activity was not affected by Ni exposure (Baccouch et al. 2001). Ni induced several fold decrease in GR activity in Ni-resistant strain of *Scenedesmus acutus* while no significant changes was observed in the sensitive one (Randhawa et al. 2001).

Oxidative Stress and Antioxidant Systems

Ni is toxic only at higher concentrations. Several studies have been carried out with plants to evaluate the effect of Ni on ROS and activity of antioxidant enzymes. ROS such as superoxide anion radical ($O_2^{\cdot-}$) hydrogen peroxide (H_2O_2) and singlet oxygen ($^1O_2$) are continuously generated in plant tissues as byproducts of metabolic processes (Dat et al. 2000). In the respiratory electron transport chain, electrons depart from their normal route and reach to O_2. This leakage induces a univalent reduction of O_2 to $O_2^{\cdot-}$, where the main site of leakage is NADH-Co enzyme reductase complex I (Moller 2001). However, during photosynthetic electron transport, triplet chlorophyll facilitates the production of $O_2^{\cdot-}$ (Foyer et al. 1994). Anyhow, ROS are relatively more reactive as compared to O_2 and therefore, they are potentially toxic to the living system. These toxic ROS can cause damage to DNA, bring about the oxidation of proteins and lipids, and degradation of chlorophyll pigments (Schutzendubel and Polle 2002).

Transition metals, including Ni have ability to produce $OH^{\cdot}$ via a Fenton/Haber–Weiss reaction (Kehrer 2000). However, Ni does not seem to be an effective catalyst of this reaction due to its relatively high oxidation/reduction potential (Leonard et al. 2004). Moreover, direct catalysis of such a reaction by Ni has not been demonstrated yet, whereas, it has been shown that Ni-dependent reduction of H_2O_2 leading to $OH^{\cdot}$ formation may be increased by certain chelating agents. ROS may also originate from the reactions catalyzed by NADPH oxidases (Sagi and Fluhr 2006). These enzymes transfer electrons from cytoplasmic NADPH to O_2, which results in the formation of $O_2^{\cdot-}$. Moreover, pretreatment of wheat roots with NADPH oxidase inhibitors repressed Ni-induced increase in the production rate of $O_2^{\cdot-}$, which further confirmed the implication of NADPH oxidase in superoxide generation. Hao et al. (2006) revealed participation of Ca^{2+} in Ni-induced production of $O_2^{\cdot-}$ by NADPH oxidase. However, Torreilles and Guérin (1990) reported that when Ni chelates with peptides containing glycylgycyl-L-histidine sequence it could peroxidize lipids through hydroxyl radical production. Generation of $OH^{\cdot}$ via Haber–Weiss reaction catalyzed by chelated Ni could possibly occur in plant cells. The toxicity of ROS explains the evolution of complex arrays of non-enzymatic and enzymatic detoxification mechanisms (antioxidant systems) in plants capable of quenching ROS without itself undergoing conversion to a destructive radical, interrupting the cascades of uncontrolled oxidation (Gratão et al. 2005; Pitzschke et al. 2006). The ROS scavenging mechanisms of plants includes SOD acting against ROS, dismutating $O_2^{\cdot-}$ to H_2O_2. Afterwards, ascorbate peroxidase (APX, EC 1.11.1.11) or GPX subsequently detoxify H_2O_2 to H_2O (Gratão et al. 2005), particularly in apoplast (Zoller et al. 2003), and by CAT in peroxisomes (Igamberdiev and Lea 2002). Different isoforms of SOD and APX are specifically targeted to chloroplasts, mitochondria, peroxisomes, as well as to the cytosol and apoplast, whereas GPX is cytosolic and CAT is located mainly in peroxisomes (Apel and Hirt 2004). Detoxification of H_2O_2 by APX occurs by the oxidation of ascorbate to MDHA, which can be regenerated by MDHA using NAD(P)H as reducing equivalent. MDHA can be spontaneously dismutated into dehydroascorbate. Ascorbate regeneration is mediated by DHAR followed by oxidation of GSH and GSSH. Rapid disproportion to MDHA

radical means that some DHA is always produced when ascorbate is oxidized in leaves and other tissues. Finally, GR can regenerate GSH from GSSH using NAD(P)H as a reducing agent. The GPX cycle is closed by the regeneration of GSH from GSSG by GR (Apel and Hirt 2004).

The induction of antioxidant system by heavy metals, including Ni is well established. Exposure of maize (Baccouch et al. 2001) and pigeonpea (Rao and Sresty 2000) to Ni provoked positive response to antioxidant systems. The exposure to metal initially resulted in a severe depletion of glutathione (Rao and Sresty 2000). However, Ni (>200 ppm) suppressed the activity of CAT in sunflower and *Hyptis*. Moreover, the activity of the CAT enzyme in sunflower decreased by more than 5 times (Pillay et al. 1996). They also observed an increase in the activity of POX (2 times) and polyphenol oxidase (8 times) at 200 ppm Ni. Depression in CAT activity and an increase in POX activity in mustard leaves (DeKock et al. 1960) and in sunflower (Agarwala and Kumar 1962) have also been reported. However, in *Crotalaria juncea*, the activity of CAT did not exhibit a clear cut response to Ni exposure, whereas, GR activity decreased significantly, both in root and shoot (Cardoso et al. 2005). Majority of studies demonstrating the response of GR to metal exposure have shown that its activity increased as a part of defense system against the metal induced stress, a change that is found to be dependent on the dose of the metal and time of application (Gratão et al. 2005). Gajewska et al. (2006) also reported an inhibition in the activity of CAT and that of SOD in wheat shoots, subjected to Ni (200 μM) stress. The respective decrease in the activity of these two enzymes, compared to the control, was of the order 31 and 24%. A decrease in the activity of these enzymes in *Alyssus bestolonii* and *Nicotiana tabacum*, subjected to Ni stress was also reported (Boominathan and Doran 2002). However, Baccouch et al. (2001) showed that Ni enhanced the activity of SOD in *Zea mays*. Likewise, the activity of POX and glutathione transferase exhibited a significant increase in response to Ni in wheat (Gajewska et al. 2006). The stimulation in the activity of POX also occurred in the seedlings of wheat (Pandolfini et al. 1992), pepper (Diaz et al. 2001) and barley (Simonovicova et al. 2004). Similarly, Alam et al. (2007), Sharma and Bhardwaj (2008) also observed an increase in the activities of CAT, POX and SOD in *Brassica juncea* plants, exposed to Ni, both in the presence and absence of 28-homobrassinolide.

Concluding Remarks

This review article provides quick access to aspects related to the essentiality of Ni in proper growth and development of the plants. Ni in adequate quantities has vital roles in a wide range of physiological processes, starting from seed germination to the productivity. Moreover, plants cannot complete their life cycle without adequate supply of this metal. Therefore, Ni has been enrolled in the list of essential micronutrients. Besides this, at elevated level it alters all the metabolic activities of the plant such as water relation and mineral nutrition, causes enzyme inhibition, disrupts stomatal functioning, photosynthetic electron transport and degrades chlorophyll molecules, consequently minimizes the photosynthetic rate, and biological yield of plants.

Excess Ni-concentration triggers oxidative damage in the plants which relates to the observed diverse toxic effects of the metal. Therefore, larger quantities of ROS/RNS and lipid peroxides damage many cellular organelles and DNA, oxidise proteins and lipids and also degrade chlorophyll pigments. However, plants are well equipped with an organized 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, metallothiones and nicotianamide, followed by sequestration into the vacuole, making it less toxic for the plants.

Future Perspectives

- Ni should get registered as a mobile trace metal which is warranted by a number of studies. However, there are certain contradictory reports as well. This aspect, therefore requires additional attention and should be addressed in future.
- It is very illustrious that Ni is an essential component of the enzyme urease, which metabolizes urea in plants. It is also reported that Ni favors seed germination/viability and seedling vigor but the reasons are yet to be explored.
- The information available on the interaction of Ni with other mineral nutrients and metabolism is restricted to only a few elements and processes, which is insufficient to make out concrete insight. The mechanism to generate Ni toxicity in photosynthetic machinery is also not very clearly understood.
- Ni, at concentrations above the tolerable limit, generates oxidative stress. However, the mechanisms operating both at protein and molecular levels that result in the generation of toxicity symptoms are yet to be explored in details.
- Ni pollution in the environment has led to researches on the emerging fields, like phytoremediation (i.e. the use of hyper-accumulators or wetland plants to remove and/or sequester Ni from soil and water). However, many such plants have limited utility for phytoremediation,

because of their slow growth, difficult propagation, seasonal growth and low biomass. Solutions to this problem are important and need further research.

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