

Occurrence, physiological responses and toxicity of nickel in plants

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Abstract The focus of the review is on the specific aspects of nickel's effects on growth, morphology, photosynthesis, mineral nutrition and enzyme activity of plants. The mobility of nickel in the environment and the consequent contamination in soil and water is of great concern. Also, the detrimental effects of excessive nickel on plant growth have been well known for many years. Toxic effects of nickel on plants include alterations in the germination process as well as in the growth of roots, stems and leaves. Total dry matter production and yield was significantly affected by nickel and also causes deleterious effects on plant physiological processes, such as photosynthesis, water relations and mineral nutrition. Nickel strongly influences metabolic reactions in plants and has the ability to generate reactive oxygen species which may cause oxidative stress. More recent evidence indicates that nickel is required in small amounts for normal plant growth and development. Hence, with the increasing level of nickel pollution in the environment, it is essential to understand the functional roles and toxic effects of nickel in plants.

Keywords Higher plants · Micronutrient · Mechanisms of activity · Nickel · Oxidative stress

Introduction

In recent years, as a result of uncontrolled industrial development worldwide, many chemical substances have resulted in significant air, water and soil pollution, to such an extent that environmental pollution is now a serious worldwide problem. Nickel (Ni) is just one of a variety of ubiquitous trace metals emitted into the environment from both natural and anthropogenic sources (WHO 1991). Of particular concern is the increasing concentration of Ni deposited in agricultural soils by airborne Ni particles. The primary sources of Ni emissions into the ambient air are combustion of coal and oil for heat or power generation, Ni mining, steel manufacture, and other miscellaneous sources, such as cement manufacture. Chiefly found in pentlandite $[(\text{Ni},\text{Fe})_9\text{S}_8]$ and garnierite ores $(\text{Ni},\text{Mg})_3\text{Si}_2\text{O}_5(\text{OH})_4$, annual world production is over 1,300,000 tons where the primary mining areas are in Australia, Canada, Cuba, Indonesia, New Caledonia, Russia, South Africa and, the USA (Table 1). In polluted air, the predominant Ni compounds are nickel sulfates, oxides and sulfides, and to a lesser extent, metallic nickel (WHO 1991). Although generally established far from city centres, cement factories are one manufacturing industry commonly associated with particulate pollution and as a result significantly affect the local areas, where cement dusts can be spread over a large area via wind and rain, and are accumulated on plants and soils (Ayvaz 1992).

Nickel is the 28th element of the periodic table. It is a silver-white metal found in several oxidation states (ranging from -1 to $+4$); however, the $+2$ oxidation state $[\text{Ni}(\text{II})]$ is the most common one in biological systems (Denkhaus and Salnikow 2002). Nickel readily forms nickel-containing alloys, which over the last 100 years have found an ever increasing variety of uses in modern

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Table 1 World mine production of nickel (National Academy of Sciences 1975; International Agency for Research on Cancer 1976; Duke 1980; Kasprzak 1987; WHO 1991; Ronald Eisler 1998)

Years	Metric tons
1900	7,500
1925	42,700
1950	141,000
1970	694,100
1975	753,000 ^a
1980	84,100
1985	821,000 ^b
2000 (projected)	>2,000,000

^a About 32 % from Canada, 18 % from New Caledonia, 17 % from the former Soviet Union, 10 % from Australia, 5 % from Cuba, 4 % from the Dominican Republic, 3 % from the Republic of South Africa, 2 % each from Greece, Indonesia, and the United States, and 5 % from other countries

^b Mostly from Canada, the former Soviet Union, Australia, and Cuba, in that order. The United States produced 6,900 tons in 1985

technologies. Global input of Ni to the human environment is approximately 150,000 and 180,000 metric tonnes per year from natural and anthropogenic sources, respectively, including emissions from fossil fuel consumption, and industrial production, use, and disposal of Ni compounds and alloys (Kasprzak et al. 2003).

Plants require access to a variety of common minerals to attain good growth. The micronutrients which are needed only in trace amounts include boron (B), chloride (Cl), cobalt (Co), copper (Cu), iron (Fe), manganese (Mn), molybdenum (Mo), nickel (Ni), selenium (Se), silicon (Si), sodium (Na) and zinc (Zn). The term “essential” mineral elements (or mineral nutrients) were first proposed by Arnon and Stout (1939) who concluded that three criteria must be met for an element to be considered essential.

1. A plant must be unable to complete its life cycle in the absence of that mineral element,
2. The function of the element must not be replaceable by another mineral element and
3. The element must be directly involved in plant metabolism.

The importance of micro-elements

Although required in only minor quantities, micro-elements are incredibly important for plant nutrition. This importance was first understood in 1840 by the German chemist, Freiherr Justus von Liebig, who made major contributions to Agricultural Science and Biological Chemistry. Liebig’s Law of the Minimum, often simply called as Liebig’s Law, is an agricultural principle that

states that if one of the nutritive elements is deficient, plant growth will be restricted even when all the other elements are abundant. Thus, any deficiency of a nutrient, no matter how small will inhibit plant development. If the deficient element is supplied, growth will consequently increased up to the point where the supply of that element is no longer the limiting factor. Increasing the nutrient supply beyond the limiting point will not normally be helpful, because other elements would likely become in minimum supply and become the limiting factor.

Plant nutrient uptake

Plants can absorb nutrients only from the soil solution phase and cannot directly access nutrients from the soil solid phase. Thus, the problem with accessing micro-elements is their limited solubility of solid phase nutrients, which limits their presence in soil solution. Plant uptake from soil solution occurs in three major ways: root interception, mass flow and diffusion.

During root interception, as the roots proliferate through the soil, they move into spaces previously occupied by soil containing available nutrients, for example, absorbed by clay particles and the root surfaces intercept nutrients during this displacement process (Barber 1984). During mass flow, movement of water and dissolved nutrients into plants is driven by the plants transpiration gradient and simple diffusion allows movement of nutrients according to differential concentration gradients from high to low concentrations.

Sources

Nickel is a ubiquitous trace metal emitted into the environment from both natural and anthropogenic sources (Table 2) which are found in soil, water and air samples within the biosphere (Barrie 1981; WHO 1991). The compounds such as nickel acetate, nickel carbonate, nickel hydroxide and nickel oxide are used in a variety of industrial process (Cempel and Nickel 2006). These compounds ultimately accumulate in the soil and environment, and can be easily taken up by plants. Thus, they can enter the food chain and cause deleterious effects to animals and human (Nieboer and Nriagu 1992; Cempel and Nickel 2006). While the level of Ni in ambient air is generally small (about 6–20 ng m⁻³), levels up to 150 ng Ni m⁻³ could be present in air contaminated by anthropogenic sources. In water, Ni derives from biological cycles and solubilization of nickel compounds from soils, as well as from the sedimentation of Ni from the atmosphere. Uncontaminated water usually contains about 300 ng dm⁻³ Ni. Farm soils contain approximately 3–1,000 mg kg⁻¹ Ni soil, but the Ni

Table 2 Inventory of nickel in various global environmental compartments (modified from Nriagu 1980)

Compartment	Mean concentration (mg/kg)	Nickel in compartment (metric tons)
Lithosphere, down to 45 km	75	4,300,000,000,000,000
Sedimentary rocks	48	120,000,000,000,000
Soils, to 100 cm	16	5,300,000,000,000
Oil shale deposits	30	1,400,000,000,000
Dissolved oceanic	0.0006	840,000,000
Nickel ore reserves	>2,000	160,000,000
Coal deposits	15	150,000,000
Terrestrial litter	15	33,000,000
Terrestrial plants	6	14,000,000
Suspended oceanic particulates	95	6,600,000
Crude oil	10	2,300,000
Terrestrial animals	2.5	50,000
Swamps and marshes	7	42,000
Lakes and rivers, total	0.001	34,000
Consumers/reducers (biological)	3.5	11,000
Atmosphere	0.3	1,500
Oceanic plants	2.5	500
Lakes and rivers, plankton	4	230

concentration can reach up to 24,000 mg kg⁻¹ Ni in soil near metal refineries and 53,000 mg kg⁻¹ Ni in dried sludge. At pH < 6.5, Ni compounds present in soil are relatively soluble, whereas at pH > 6.7, most Ni exists as insoluble hydroxides.

Nickel toxicity to plants

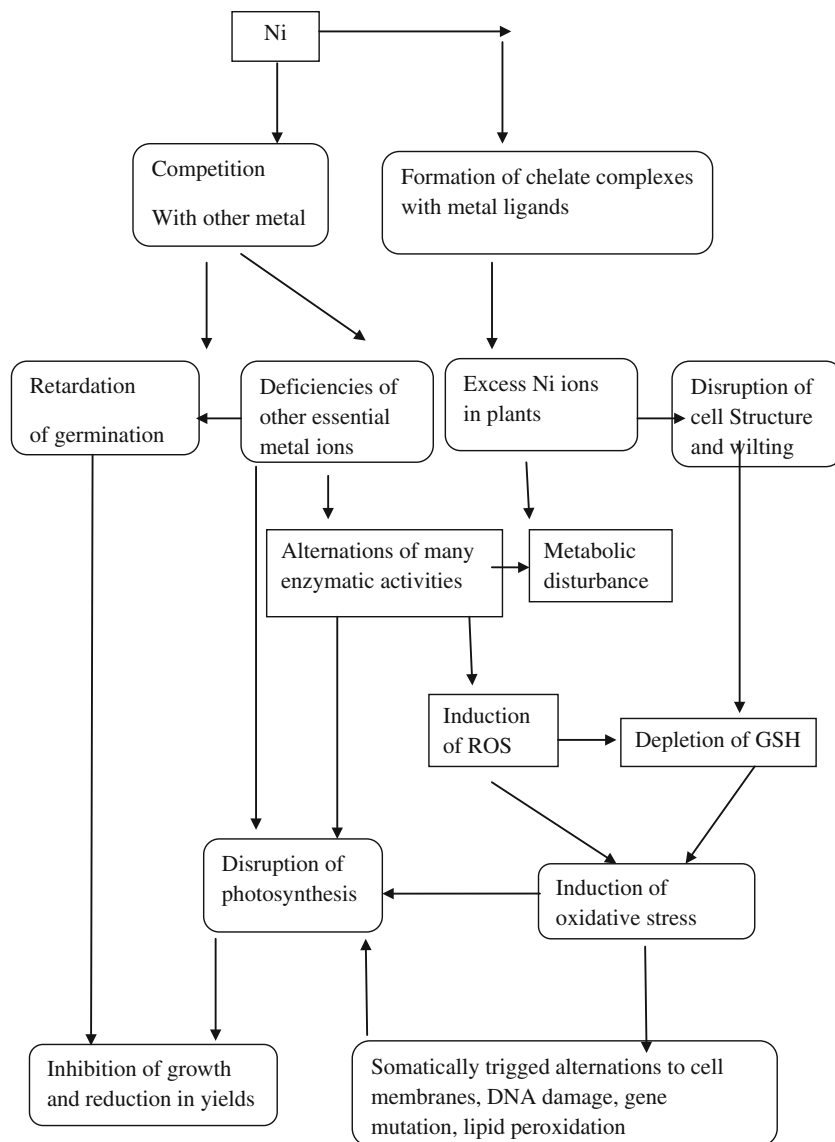
Nickel is a heavy metal and an essential microelement for plants, animals, and humans, but toxic at high concentrations, exceeding optimum intake values. As with other heavy metals, excess concentrations of Ni in plants cause chlorosis and necrosis, due to disruption of Fe uptake and metabolism (De kock 1956; Crooke 1958). Elevated concentrations of Ni can inhibit cell division at root meristems in non-tolerant plants (Robertson and Meakin 1980), and decrease plant growth (Foy et al. 1978). Previous studies have also shown that Ni has a negative effect on photosynthesis and respiration (Carlson et al. 1975). Nickel is an important metal for metabolism because Ni is a major component of plant enzymes (Fig. 1). For instance, Ni is a constituent of urease, and small quantities of Ni (0.01–5 µg/g dry wt) are essential for some plant species.

Ni²⁺ concentrations in polluted soil may reach levels 20- to 30-fold higher (200–26,000 mg/kg) than the range typically found in natural soils (10–1,000 mg/kg) (Izosimova 2005). Elevated levels of Ni in soil may cause various physiological alterations and diverse toxicity symptoms such as chlorosis and necrosis in a variety of plant species (Zornoza et al. 1999; Pandey and Sharma 2002), rice (Samantaray et al. 1997). Plants grown in high Ni²⁺ containing soil showed impairment of nutrient balance and resulted in disorder of cell membrane function. Thus, Ni²⁺ affected the lipid composition and H-ATPase activity of the plasma membrane as reported in *Oryza sativa* shoots (Ros et al. 1992). Exposure of wheat to high level of Ni²⁺ enhanced MDA concentration (Pandolfini et al. 1992). Moreover, Gonnelli et al. (2001) reported an increase in MDA concentration of Ni²⁺ sensitive plants compared to a Ni²⁺ tolerant *Silene paradox*. Such changes might disturb membrane functionality and ion balance in the cytoplasm, particularly of K⁺, the most mobile ion across plant cell membranes. Other symptoms observed in Ni²⁺ treated plants were related with changes in water balance. High uptake of Ni²⁺ induced a decline in water content of dicot and monocot plant species (Dimkpa et al. 2008). The decrease in water uptake was used as an indicator of Ni²⁺ toxicity in plants (Pandey and Sharma 2002; Gajewska et al. 2006).

Effect on plant growth

The toxic effects of Ni and other heavy metals are primarily manifested by the inhibition of plant growth (Seregin and Ivanov 2001; Seregin et al. 2003; Pandolfini et al. 1992; Nagajyoti et al. 2010), an index widely employed to assess environmental pollution (Wang 1987). Growth inhibition gains strength at higher metal concentration (Wang 1987; Samantaray et al. 1997; Yusuf et al. 2011). 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; Sresty and Madhava Rao 1999; Wong and Bradshaw 1982; Samantaray et al. 1997), and therefore the root test is widely used for evaluating the toxicity of various agents, including heavy metals (Seregin et al. 2003; Wong and Bradshaw 1982; Wang 1987). The tolerance index was determined between the root/shoot length of the heavy metal-stressed plant and that of the control plant (Samantaray et al. 1997), and LC50, the metal concentration that inhibits root growth by 50 %, are the indices of plant tolerance toward heavy metals (Wong and Bradshaw 1982; Wang, 1987). Using the latter index, Wong and Bradshaw arranged metals in decreasing toxicity to root growth order Cu > Ni > Mn > Pb > Cd > Zn > Al > Hg > Cr > Fe for *Lolium perenne* seedlings (Wong and Bradshaw 1982). This order will vary with different

Fig. 1 Mechanisms mediating toxic effects of excess Ni in plants (Chen et al. 2009)



plant species (Neiboer and Richardson 1980; Karataglis et al. 1986), since plant species differ in their innate metal tolerance. However, such orders do not always reflect genuine toxicity effects since some authors used the weight rather than molar concentrations of metals (Seregin and Ivanov 2001). Comparison of LC50 indices in diverse plant species allows plants to be classified into tolerant (*Cucumis sativus* and *Panicum miliaceum*) and less tolerant species (*Chloris gayana*, *Lactuca sativa*, *Lolium perenne*, *Panicum maximum*, and *Zea mays*) that have a LC50 value lower by an order of magnitude (Wong and Bradshaw 1982; Wang 1987). Unlike root growth, the process of lateral root initiation is very resistant to the most heavy metals (Seregin and Ivanov 2001; Ivanov 1994), due to the endodermal barrier and the characteristic structure of the central

cylinder cells (Seregin and Ivanov 1997, 1998). However, Ni²⁺ considerably decreased the number of lateral roots in rice and maize (Seregin et al. 2003; Samantaray et al. 1997), apparently because Ni can cross the endodermal barrier and accumulate in the pericycle cells. Beside root growth, Ni²⁺ reportedly exerts considerable inhibitory effects on shoot growth and morphogenesis in *Phaseolus vulgaris* (Piccini and Malavolta 1992), *Digitaria sanguinalis*, *Cyperus difformis*, and *Chenopodium ambrosioides* (Ewais 1997). The total growth inhibition in *P. vulgaris* also affected seed formation (Piccini and Malavolta 1992). Seed germination is the most resistant indices to heavy metal exposure. The caryopses of rice (Das et al. 1978) and maize (Seregin and Kozhevnikova 2005) germinated at high concentrations of Ni salts (10⁻² M).



Effect on morphology

In addition to toxic effects on growth, heavy metals may induce changes in plant morphology and anatomy. Thus, exposure to 1 mM NiSO₄ solution decreased the mesophyll thickness, the size of vascular bundles, the vessel diameter in the main and lateral vascular bundles, and the width of epidermal cells in *Triticum aestivum* leaves (Seregin and Kozhevnikova 2006a, b), whereas in the leaves of *Brassica oleracea* plants grown in agar in the presence of NiSO₄·7H₂O (10–20 g/m³), the volumes of intercellular spaces and palisade and sponge mesophyll decreased relative to control plants (Molas 1997). In addition to general metabolic disorder, heavy metals are known to decrease the plasticity of cell walls, probably by direct binding to pectines and by promoting peroxidase activity in the cell walls and intercellular space. These peroxidases are essential for lignification and linkage between extensin and polysaccharides containing ferulic acid (Pandolfini et al. 1992). Another way to inhibit growth is by hampering cell division (Robertson and Meakin 1980; L’Huillier et al. 1996; Knasmüller et al. 1998; Amosova et al. 2003). The incubation on 1.5–5 mM NiCl₂ solution decreased the mitotic index in *Vicia faba* roots (Knasmüller et al. 1998), and at a concentration of 60 mM, in *Z. mays* roots (L’Huillier et al. 1996). At a concentration of 0.1 mM, NiSO₄ cell divisions were blocked in the rhizoderm, exoderm, middle cortex, except in the distal cells of these tissues, and in the peripheral cells of caliptrogen in the embryonic root of *T. aestivum* (Seregin and Kozhevnikova 2006a, b). The inhibition of cell division was frequently accompanied by disorganization of nuclear structures. Thus, in the root tips of *Cajanus cajan* plants grown in the presence of 1.5 mM NiSO₄·6H₂O, two nucleoli developed in the nucleus, the chromatin became exceedingly condensed, and the nuclear membrane was disrupted (Sresty and Madhava Rao 1999).

Heavy metals may cause mitosis disorder and chromosome aberrations. For example, in the meristematic cells of *Allium cepa* root, Ni²⁺ (10–100 μM) produced various chromosome aberrations: C-metaphases, sticky chromosomes, and chromosome bridges, while the interphase cells contained micronuclei. At high Ni²⁺ concentrations (1–10 mM), the nuclear material was found in the cytoplasm, whereas the nuclei contained nucleoli of irregular form (oval, oblong, and dumbbell like). Similar changes were observed in plant cells exposed to other heavy metals; however, the extent of damage depended on concentration and became the basis for ordering the metals by decreased mutagenic effect (the minimum toxic concentration is listed in parentheses): Hg²⁺ and Cd²⁺ (10⁻⁷–10⁻⁵ M) > Zn²⁺, Pb²⁺, Cu²⁺, Ni²⁺, Co²⁺, Al³⁺ and Cr³⁺ (10⁻⁴–10⁻³ M) > Mn²⁺ and Mg²⁺ (10⁻² M) (Liu et al. 1995). Plant growth inhibition by Ni and other heavy metals

results from general metabolic disorder and immediate inhibition of cell divisions. However, it is not clear whether Ni enters cell nuclei at high concentrations and, if it does, how important is immediate Ni interaction with DNA and nuclear proteins. The possible effect of Ni²⁺ on fragment formation is also unknown. By elucidating these issues, we will better understand the toxic effects of Ni on plant growth and morphogenesis.

Inhibition of photosynthesis

Heavy metals are known to cause non-specific inhibition of photosynthesis, by several direct and indirect means. The diminished rate of photosynthesis is related to disrupted chloroplast structure, blocked chlorophyll synthesis, disordered electron transport, inhibited activities of the Calvin cycle enzymes, and CO₂ deficiency caused by stomatal closure (Seregin and Ivanov 2001). The decrease in chloroplast size and numbers and the disorganization of chloroplast ultra structure were reported in *B. oleracea* plants grown in agar in the presence of NiSO₄·7H₂O at 10–20 g/m³. Other changes such as diminished numbers of grana and thylakoids, their deformation, the formation of plastoglobuli, and the changes in the membrane lipid composition were also observed in the *Brassica* plants. Such changes arose from either the Ni-induced decline in cell moisture content or from an oxidative stress resulting in peroxidation of membrane lipids (Molas 1997). Several authors reported diminished chlorophyll content in the leaves of Ni-treated plants, where such chlorosis could result from both Fe and Mg deficiency and the inhibition of chlorophyll synthesis (Ewais 1997; Piccini and Malavolta 1992). The disruption of electron transport exemplifies another mechanism that hampers photosynthesis. Numerous experiments demonstrated that Ni²⁺, as with other heavy metals, primarily affects PSII (Seregin and Ivanov 2001; Veeranjanyulu and Das 1982; Mohanty et al. 1989; Krupa and Baszynski 1995; Maksymiec 1997); this evidence is in line with the predominant Ni accumulation in the PSII-containing lamella regions (Veeranjanyulu and Das 1982). When inspected in more detail, Ni was shown to inhibit electron transport from pheophytin via plastoquinone QA and Fe to plastoquinone QB by changing the structure of carriers, such as plastoquinone QB, or the reaction center proteins (Mohanty et al. 1989; Krupa et al. 1993). In the thylakoids, Ni ions also decreased the contents of cytochromes b6f and b559, as well as ferredoxin and plastocyanin and as a result, the efficiency of electron transport decreased (Veeranjanyulu and Das 1982). By inhibiting key enzyme activities of the Calvin cycle, such as Rubisco, 3-phosphoglycerate kinase, fructose-1,6-bisphosphatase, aldolase, and NAD- and NADP-dependent phosphoglyceraldehyde dehydrogenases, heavy metals can



inhibit photosynthesis. Such effects were demonstrated in *C. cajan* leaves following several days of incubation with 1 mM NiCl₂ solution (Sheoran et al. 1990). The inhibition of Calvin cycle reactions would lead to the accumulation of ATP and NADPH produced by photosynthetic reactions; the latter, in their turn, develop a high pH gradient across the thylakoid membrane that blocks PSII activity (Krupa and Baszynski 1995). Another mechanism inhibiting photosynthesis stems from stomatal closure in Ni-stressed plants that limits plant CO₂ uptake (Sheoran et al. 1990; Bishnoi et al. 1993). The toxic effects of heavy metals on many other metabolic processes would amplify the direct inhibition of photosynthesis.

Effect on mineral nutrition

One of the non-specific mechanisms of heavy metal toxicity is the reduction of cation and anion absorption by plant roots (Pallavi and Ram Shankar 2005). The published literature on the effects of Ni on plant mineral nutrition is rather contradictory. In the presence of Ni, the contents of mineral nutrients in plant organs may increase, decrease, or be unaffected. One of the probable mechanisms for decreasing the uptake of macro- and micro-nutrients by Ni relies on the competition for common binding sites due to comparable ionic radii of Ni²⁺ and other cations. Such mechanisms may have operated when the uptake of Mg²⁺ (78 pm), Fe²⁺ (82 pm), and Zn²⁺ (83 pm) decreased in the presence of Ni²⁺ (78 pm) where the ionic radii in parentheses (Emsley, 1991). The reduced uptake of Mg and Fe is one of the prime causes of chlorosis induced by excess of environmental Ni (Piccini and Malavolta 1992; Khalid and Tinsley 1980). The decline in nutrient uptake may also result from Ni-induced metabolic disorders that affect the structure and enzyme activities of cell membranes (Seregin and Ivanov 2001). Thus, Ni²⁺ affected the sterol and phospholipid composition of the plasma membrane in *O. sativa* shoots, with concomitant changes in the ATPase activity (Ros et al. 1990). Apparently, these changes affected the membrane permeability and in this way changed the ion balance in the cytoplasm. The effects of Ni on nutrient uptake depend on the Ni concentration in the environment. Experiments with ryegrass demonstrated that Fe content in the shoots increased at low Ni concentrations and decreased at higher concentrations (Khalid and Tinsley 1980). An increase in soil Ni content from 50 to 200 mg/kg soil decreased the contents of Cu and Mg in the caryopses and Mg and Ca in the shoots of *T. aestivum* (Barsukova and Gamzikova 1999). Rubio and Pandolfini concluded that at high Ni concentrations (about 0.1–1 mM), the contents of macro and micro-nutrients in plant tissues are usually lowered because of disordered absorption and transport (Rubio et al. 1994; Pandolfini et al. 1992). At the same

time, at low Ni concentrations in the environment (10–1 μM), the contents of nutrients did not change and in some cases even increased (Piccini and Malavolta 1992; Barsukova and Gamzikova 1999). Such a phenomenon was described as a “concentrating effect”, because they were observed as a result of growth inhibition (dry biomass decreases) in the plants that were grown in nutrient solutions low in Ni. While the rate of metal absorption stayed the same in the control plants, consequently, the contents of heavy metals increased per unit of dry matter (Piccini and Malavolta 1992). Identical Ni concentrations would have different effects on different plant species. For example, when *T. aestivum* and *T. durum* were exposed to 67 μM Ni, Ca and Mg concentrations increased in both species while Zn concentrations decreased in the leaves of *T. aestivum* but remained unchanged in *T. durum* (Barsukova and Gamzikova, 1999). Toxic Ni concentrations specifically affect the ionic balance in various plant organs. The contents of Fe, Mn, and Zn decreased in *T. aestivum* leaves at the tillering stage, while only Mn content declined in the roots (Barsukova and Gamzikova 1999). Plant species which are tolerant to Ni may differ in their changes in mineral contents as affected by Ni than plant species susceptible to Ni. This susceptible genotype also manifested Mn and Mg deficiency in the leaves, resulting in chlorosis (Barsukova and Gamzikova 1999).

Effect of nickel on enzyme activity

As with many other heavy metals, Ni affects various physiological processes in plants, including enzyme activities (Van Assche and Glijsters 1990). However, it is not always easy to differentiate between the direct and indirect effects of heavy metals on enzyme activities. Indirect effects arise from ion-induced imbalances due to competitive inhibition of absorption and transport of nutrients such as Cu, Fe and Zn. Direct effects of heavy metals inhibit enzymes by interacting with the SH groups of proteins, altering protein conformation and thereby causing inactivation of the enzymes. Presently, about a hundred enzymes are known to be inhibited by SH-group binding resulting in concomitant metabolic disorders (Muhammad Sajid and Muhammad 2012). When *Z. mays* seedlings were incubated in solutions of Ag, Cd, Co, Cu, Hg, Pb, Tl and Zn salts (0.001–3 g/l), the affinity of metals for SH-groups was found to significantly correlated with the molar concentrations that inhibited growth by 50 % (Ivanov et al. 2003). Binding of SH groups by Ni²⁺ is one of the mechanisms proposed for the in vitro Ni toxicity towards Mg²⁺-dependent ATPases of the plasmalemma; however, Ni²⁺ may also directly bind to ATP and in this way deplete the substrate pool of ATPase (Ros et al. 1992). While the affinity of Ni²⁺ for sulphhydryl groups is lower

than that of other heavy metals, the affinity of Ni for histidine exceeds that of both Cd and Pb, and therefore, inhibition of enzyme activity by Ni may result from the interaction with histidine. The toxic effects of metals on enzyme activity in vitro do not always agree with the in vivo effects at the same salt concentration. Such disagreement may stem from the presence of efficient cellular mechanisms for detoxification and the physiological barriers that curb metal translocation into the cytoplasm. To illustrate, Ni^{2+} was shown to promote in vivo Mg^{2+} -dependent ATPases in the plasmalemma of *O. sativa* shoots (Ros et al. 1990). Total decline of enzyme activities is sometimes observed due to decreased enzyme contents. Thus, the decrease in nitrate reductase activity in soil-grown *Beta vulgaris* plants following the addition of 1 mM NiSO_4 resulted from the diminished rates of nitrate uptake and translocation into the shoots wherein nitrate is reduced. Nitrate in the cytoplasm induces the expression of the nitrate reductase gene, and hence it is the shortage of nitrate in the cells that would primarily decrease the enzyme concentration. Besides, glutamine synthetase and alanine aminotransferase activities were also lowered and in this case, both activities depended significantly on the cytoplasmic levels of nitrate and their substrates (Kevresan et al. 1998; El-Shintinawy and El-Ansary 2000). Similar mechanism of indirect influence on nitrate reductase activity was established for other heavy metals (Pallavi and Ram Shankar 2005). Depending on its concentration, Ni ions can both stimulate and inhibit enzyme activities in plant tissues. Thus, the activities of IAA oxidase, ascorbate oxidase, catalase, and peroxidase in *O. sativa* seedlings were at their highest when exposed to 50 μM NiCl_2 , but the enzyme activities considerably declined at a higher Ni concentration and were promoted at lower Ni concentrations. In addition, it was shown that Ni did not affect these enzymes directly: under in vitro conditions, the same Ni concentrations did not produce any visible inhibition of enzyme activities (Das et al. 1978). Enzyme in vivo resistance to Ni varies with plant development stage. Thus, in *C. cajan* leaves collected from young plants (30 days after sowing), Ni inhibited the activities of the Calvin cycle enzymes. Meanwhile, the inhibitory effect was insignificant when Ni salt was added at later developmental stages (70 days after sowing) (Sheoran et al. 1990). However, the mechanism of this phenomenon is unknown. The production of reactive oxygen species in plant cells is another universal mechanism of heavy metal toxicity. Plants respond to oxidative stress by elevating the activity of the antioxidant enzymes of the ascorbate–glutathione cycle, such as catalase, peroxidase, superoxide dismutase, glutathione reductase, and ascorbate oxidase, which protect plant cells against free radicals (Das et al. 1978; Seregin and Ivanov 2001; Pandolfini et al. 1992). The effect of

Ni^{2+} on antioxidant enzyme activities may differ for accumulator and non-accumulator plant species. When the non-accumulator, *A. maritimum* were grown in nutrient solution containing Ni, the activities of superoxide dismutase, ascorbate peroxidase, and glutathione reductase were enhanced, whereas these activities were diminished in the hyper accumulator *A. argenteum*, and activity of superoxide dismutase was completely inhibited (Schickler and Caspi 1999). The tolerance to Ni in the hyper accumulators seems to employ other detoxification mechanisms for Ni so that Ni content is diminished in the cytoplasm, and the demand for antioxidant enzyme activities is alleviated. We conclude that, at high Ni concentrations, most of enzyme activities were diminished, whereas some activities, especially those of the antioxidant enzymes, increased. In most cases, we do not know whether these changes in enzyme activities stem directly from Ni^{2+} effects, such as binding to SH-groups or histidine or displacing the metals from metal–enzyme active centers, or indirectly, when mediated by the chain of reactions that affect the expression of the corresponding genes or exhaust their substrate pools. The inhibition of enzyme activities by heavy metals is one of the causes of declining cell metabolism (Table 3).

Toxicity mechanisms of Ni in plants

Although Ni toxicity in plants has been extensively reported, the detailed mechanisms involved are still poorly understood (Gajewska and Sklodowska 2007). The toxicity of Ni is likely to be caused by indirect mechanisms, because it is not an active or redox metal. Based on analyses of the available data, we propose two mechanisms of Ni toxicity in plants: interference with other essential metal ions and induction of oxidative stress.

Interference with other essential metal ions

It is well known that Ni and a range of other metals such as K, Na, Ca, Mg, Fe, Cu, Zn, and Mn are essential for plant growth (Taiz and Zeiger 2002). Ni has some similar characteristics to Ca, Mg, Mn, Fe, Cu, and Zn. Therefore, Ni may compete with these metals in the absorption and transpiration processes (Kochian 1991; Kupper et al. 1996). As a result of competition, Ni at high concentrations may inhibit the absorption of these metals, decreasing their concentration and even leading to their deficiency in plants (Ahmad et al. 2007; Van Assche and Glijsters 1990; Rubio et al. 1994). Subsequently, this may affect important physiological processes, and ultimately result in toxic effects (Gajewska et al. 2006; Gon_alves 2007). For example, Ni can decrease Mg (or Fe) uptake and its supply



Table 3 Nickel effects on enzymatic activity (Seregin and Kozhevnikova 2006b)

Enzyme	Process	Concentration Ni, mM	Enzyme activity	Plant species	Source
Rubisco	CO ₂ fixation	0.5; 1	Decrease	<i>C. cajan</i>	Sheoran et al. (1990)
Glyceraldehyde 3-phosphate dehydrogenase	Calvin cycle	0.5; 1	Decrease	<i>C. cajan</i>	Sheoran et al. (1990)
3-Phosphoglycerate kinase	Calvin cycle	0.5; 1	Decrease	<i>C. cajan</i>	Sheoran et al. (1990)
Aldolase	Calvin cycle	0.5; 1	Decrease	<i>C. cajan</i>	Sheoran et al. (1990)
Fructose 1,6-bisphosphatase	Calvin cycle	0.5; 1	Decrease	<i>C. cajan</i>	Sheoran et al. (1990)
NADP- and NAD-dependent phosphoglyceraldehyde dehydrogenases	Calvin cycle	0.5; 1	Decrease	<i>C. cajan</i>	Sheoran et al. (1990)
Nitrate reductase	Nitrate reduction	1	Decrease	<i>B. vulgaris</i>	Kevresan et al. (1998)
H ⁺ -ATPase	Ion transport	0.5	Increase	<i>O. sativa</i>	Ros et al. (1990)
Glutamine synthetase	Glutamine synthesis	1	Decrease	<i>B. vulgaris</i>	Kevresan et al. (1998)
Alanine aminotransferase	Transformation of alanine into pyruvate	0.2	Decrease	<i>Glycine max</i>	El-Shintinawy and El-Ansary (2000)
IAA oxidase	IAA oxidation	<0.05 >0.05	Increase Decrease	<i>O. sativa</i>	Das et al. (1978)
Glutathione reductase	Glutathione reduction	0.01–1	Increase	<i>Allysum maritimum</i> , <i>A. argenteum</i>	Schickler and Caspi (1999)
Ascorbate oxidase	Ascorbate oxidation	<0.05 >0.05	Increase Decrease	<i>O. sativa</i>	Das et al. (1978)
Superoxide dismutase	O ₂ ⁻ deactivation	0.01 0.1	Decrease Increase	<i>Allysum maritimum</i>	Schickler and Caspi (1999)
Catalase	H ₂ O ₂ degradation	<0.05 >0.05	Increase Increase	<i>O. sativa</i>	Das et al. (1978)
Peroxidase	Polyphenolic oxidation	1–40	Increase	<i>T. aestivum</i>	Pandolfini et al. (1992)

to aerial parts via competition, and then induce deficiencies of these elements in plants. This can result in the retardation of germination, growth suppression, and reductions in yields (Madhava Rao and Sresty 2000; Seregin and Kozhevnikova 2006a, b). These inhibitory effects of Ni on the growth of plants can be reduced by supplying additional Mg (or Fe) ions (Genrich et al. 1998; Gon_alves 2007; Ouzounidou et al. 2006). Therefore, Ni toxicity in plants is partly due to interference with other essential metal ions. In addition, Ca²⁺ has been shown to reduce the toxic effects of Ni²⁺ on root development in *Alyssum bertolonii* Desv. (Gabbrielli and Pandolfini 1984), while Cu seemed to increase Ni toxicity in terms of reduced vitality and growth of Scots pine (Nieminen 2004). Many enzymes, such as superoxide dismutase (SOD) and catalase (CAT), are metalloenzymes containing Fe, Cu, Zn, or Mn in their prosthetic groups. Since excess Ni has been shown to decrease the contents of Fe (Pandey and Sharma 2002), Cu and Zn (Parida et al. 2003) in plant tissues, it is speculated that Ni may reduce the biosynthesis of these metalloenzymes by causing deficiencies of these essential metals

(Gajewska et al. 2006). Further studies on photosynthesis in plant leaves suggest that Ni can competitively remove Ca ions from the Ca-binding site in the oxygen evolution complex (Boisvert 2007) and replace the Mg ion of chlorophyll (Kupper et al. 1996; Souza and Rauser 2003; Solymosi 2004) which may eventually inhibit the PSII electron transport chain.

Induction of oxidative stress

Increasing evidence suggests that Ni toxicity in plants is also associated with oxidative stress (Madhava Rao and Sresty 2000; Gajewska et al. 2006; Boominathan and Doran 2002; Gonnelli et al. 2001). Excessive Ni leads to significant increases in the concentration of hydroxyl radicals, superoxide anions, nitric oxide and hydrogen peroxide (Boominathan and Doran 2002; Stohs et al. 2001; Hao et al. 2006). Since Ni is not a redox-active metal, it cannot directly generate these reactive oxygen species (ROS). However, it interferes indirectly with a number of antioxidant enzymes (Pandey and Sharma, 2002;

Pandolfini et al. 1992; Baccouch et al. 2001; Gajewska and Sklodowska 2005), for example, SOD, CAT, glutathione peroxidase (GSH-Px), glutathione reductase (GR), peroxidase (POD), guaiacol peroxidase (GOPX), and ascorbate peroxidase (APX). Exposure of plants to Ni at low concentrations ($=0.05$ mM) 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 defences and hence lead to the removal (or scavenging) of ROS (Freeman 2004; Gomes-Juniora 2006). However, excess Ni 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 (Zhao et al. 2008; Del Carmen et al. 2002).

The activity of antioxidant enzymes may vary with the duration and type of stress treatment, and between plant species (and plant parts). For instance, in experiments by Gajewska and Sklodowska (2007), SOD and CAT activities decreased significantly in the leaves of wheat plants in response to 100 (1 M) Ni treatment for 3, 6 and 9 days, whereas GSH-Px, GOPX and APX activities were increased. However, the same authors (Gajewska and Sklodowska 2005) found that exposure of 14-day-old pea plants to Ni (10, 100, 200 ml for 1, 3, 6 and 9 days) resulted in reductions in SOD activities in both leaves and roots, and APX activity in roots, together with increases in APX activity in leaves, increases in glutathione S-transferase (GST) activities in both leaves and roots (most pronounced in roots), while CAT activity generally remained unchanged. Ni at 0.5 mM concentration increased the activities of SOD, GR and POD and decreased the activity of CAT in 6-day-old seedlings of pigeonpea (*C. cajan* L. Millspaugh) (Madhava Rao and Sresty 2000). CAT and POD activities in leaves decreased significantly after cabbage was treated with 0.5 mM Ni for 8 days (Pandey and Sharma 2002). The same tendency was found for SOD, CAT and POD activities in leaves of *Hydrocharis dubia* in response to 0.5, 1, 2, 3, 4 mM Ni treatments for 3 days (Papadopoulos 2007). Ni has also been shown to increase the plasma membrane (PM) NADPH oxidase, which was shown to be involved in Ni-induced ROS generation in roots of 5-day-old wheat seedlings (*Triticum durum*) (Hao et al. 2006). ROS have been shown to damage cell membrane, proteins, lipids and DNA (causing, inter alia, DNA base oxidation, DNA protein cross-links, DNA gaps and breaks), resulting in lipid peroxidation (Boominathan and Doran 2002; Baccouch et al. 2001), developmental defects and genetic instability in plant species (Oller et al. 1997; Bal and Kasprzak 2002; Papazoglou et al. 2005). For example, malondialdehyde (MDA, a lipid peroxidation product) content in roots and

shoots increased, when pigeon pea plants were treated with 0.5 to 1.5 mM Ni (Madhava Rao and Sresty 2000). Similar results have also been reported in corn, wheat and *Alyssum* species (Baccouch et al. 2001; Schickler and Caspi 1999; Dietz et al. 1999). In addition, Ni-induced depletion of low molecular weight proteins, such as GSH, may contribute to the induction of oxidative stress in plants (Madhava Rao and Sresty 2000; Kukkola et al. 2000).

Concluding remarks

This review 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 morphological and physiological functions, starting from germination to the productivity. Moreover, plants cannot complete their life cycle without adequate supply of this metal. Excess Ni toxicity is illustrated by the inhibition of lateral root development, photosynthesis, mineral nutrition and enzymatic activity and it is in this aspect where Ni toxicity differs from that of other heavy metals such as Ag, Cd, Pb, Zn, Cu, Co, and Hg. Thus, one of our future challenges to understand Ni role in plants would be to unravel the complete picture of translocation, partitioning and required amounts at different stages of plant development.

Future perspectives

1. Although many studies focused on the toxic effects of Ni on plants, the detailed mechanisms involved at both protein and molecular levels are poorly understood and require further indepth research.
2. Nickel is an essential element to plants which involves urea metabolization process. Ni presence also enhances seed germination and seedling vigor and the reasons yet to be explored.
3. The interaction of Ni with the other plant nutrients in plants is very little known and some of the studies must be devoted in this direction to understand fully the role of Ni in plants.
4. Ni pollution is a serious environmental concern which led to research on phytoremediation. However, studies are needed to know the details at both biochemical and molecular levels to understand the Ni tolerance of Ni hyperaccumulators.

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