Biological Forum – An International Journal **8**(1): 414-419(2016)

ISSN No. (Print): 0975-1130
ISSN No. (Online): 2249-3239

An Account of Nickel Requirement, Toxicity and Oxidative Stress in Plants

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(Received 30 March, 2016, Accepted 02 May 2016)
(Published by Research Trend, Website: www.researchtrend.net)

ABSTRACT: Nickel, heavy metal is an essential micronutrient for plants but its requirement is very low. Various anthropogenic activities have contributed to the toxic level of nickel in air, water and food. Through scientific studies, nickel toxicity now has been recognized as a primary growth-limiting factor in plants which results in reduced plant growth and development. Nickel hyperaccumulative plants have been reported presence of resistance against herbivores and pathogens but it cannot justify the overall environmental damage due to nickel toxicity. To overcome the nickel induced environmental pollution, its role in the environment and toxic effect are essential to understand. The objective of this paper is to cover the occurrence and sources of Nickel, its dimension from essential to toxic and its toxicity implications, leading to oxidative stress in plants.

Keywords: Heavy metal, Hyperaccumulative plants, Environmental damage, Oxidative stress

INTRODUCTION

Nickel (Ni) is a silver- white, hard, lustrous, malleable, ductile, ferromagnetic metal and the 24th most abundant element found in the earth's crust as well as the 5th most abundant element by weight after iron (Fe), oxygen (O), magnesium (Mg) and silicon (Si), constituting about 3% of the earth composition (Cempel and Nikel, 2006). It is present in all soil types and is also emitted from volcanoes. In the environment, it is primarily found combined with oxygen or sulfur as oxides or sulfides (Baralkiewicz and Siepak, 1999). It is also found in meteorites and on the ocean floor in lumps of minerals called sea floor nodules. The metal is released into the atmosphere during mining and by industries that make or use Ni, Ni alloys or Ni compounds (Nieminen & Shotyk, 2007). Ni is also released into the atmosphere by oil-burning power plants, coal-burning power plants and trash incinerators. According to EPA (Environment Protection Act), there are 882 of the 1,662 current or former NPL (National Priorities List) sites contaminated with Ni and are targeted for long-term federal clean-up activities. Chemical and physical forces such as erosion, leaching, precipitation constantly redistribute Ni between land, water and air (Davis *et al*, 2001). The average concentration of Ni in the earth's crust is 0.008%, 1.5% in deep-sea nodules and meteorites contain 5–50% (IARC, 1990). The natural background levels of Ni in water are relatively low, 0.228–0.693µg L^{-1} in open ocean water while in fresh water systems it is generally less than 2 µg L^{-1} (Nieminen *et al*, 2007). Agricultural soils contain Ni at levels of 3–1000 mg kg^{-1} (Nagajyoti *et al*, 2010). The Ni content is enriched in coal and crude oil. Ni in coals ranges up to 300 mg kg^{-1} while in crude oils, it is found in the range from 1 to 80 mg kg^{-1}. In urban areas, Ni levels in the ambient air range from 1-10 ng m^{-3} (Foxall K, 2009). Naturally occurring concentration of Ni in soil and surface waters is lower than 100 and 0.005 ppm respectively but in soils it varies widely and ranges from 3 to 1000 ppm (Iyaka, 2011). The hydrated form as Ni $(H_2O)^{+6}$, is the most common form of Ni found in the soil solution. It also exists in several other forms in soils such as adsorbed or complex on organic cation surfaces or on inorganic cation exchange surfaces, inorganic crystalline minerals or precipitates, water soluble, free-ion or chelated metal complexes in soil solution. Depending on the soil type and pH, nickel is highly mobile in soil (Lopez and Magnitskiy, 2011; Mellis *et al*, 2004). At pH >6.7, most nickel exists as insoluble hydroxides, whereas at pH<6.5, the compounds are relatively soluble.

At acidic pH, solubility and mobility of Ni increases, showing that soil pH is a major factor that controls the absorption of Ni, while other factors such as clay content, Fe-Mn mineral and soil organic matter are secondary (Gabbrielli *et al*., 1991). The present review gives an overview of nickel as a micronutrient, transporters, toxicity and implications and role of antioxidative system to overcome oxidative stress in the plant system.

A. Nickel as a micronutrient

Nickel has been considered as an essential element having a role in nitrogen metabolism that stimulates plant growth and seed germination (Sengar *et al*, 2008). More than 50% of plant nickel is retained in roots while Ni found in stems and leaves is mainly located in the vacuoles, cells walls and epidermal trichomes. Due to its requirement and its low concentration in plants (0.05-10 mg kg^{-1} dry weight) (Chen *et al*, 2009), it comes under the category of micronutrients (trace elements). Its metabolism is very critical for certain enzyme activities such as urease, glyoxalases (family I), peptide deformylases, methyl-CoM reductase,some superoxide dismutases and hydrogenases, in maintaining proper cellular redox state and various other biochemical, physiological and growth responses (Kutman *et al*, 2013).

B. Transporters for Nickel

Ni is transported from roots to shoots and leaves through the transpiration stream via the xylem. This essential element is supplied to meristematic parts of the plants by retranslocation from old to young leaves and to buds, fruits and seeds via the phloem. The transport of Ni is tightly regulated by Ni binding proteins and metal-ligand complexes (Chen *et al*, 2009). Metal ligands, such as nicotianamine (NA), histidine (His) and organic acids (citric acid and malate ions), act as intracellular chelators, which bind Ni in the cytosol or in subcellular compartments for transport, translocation and accumulation within plants (Hossain and Piyatida, 2011; Cataldo *et al*, 1978; Manara A, 2012).

Many factors affect the bioavailability of nickel in soil, the most important being the total metal concentration, pH, presence of organic matter, redox conditions, and the presence of clays and hydrous oxides (Reichman, 2002). It is reported that Cu^{2+} and Zn^{2+} inhibit Ni^{2+} uptake competitively, possibly sharing the same transport system (Cataldo *et al*, 1978). Ni compounds can also be absorbed by the Mg^{2+} transport system because of the same charge/size ratio of both the metal (Gospodarek & Socha, 2010). The uptake of Ni is higher in acidic soils as compared to the alkaline soils as in case of Lathyrus sativus, where Ni uptake was higher at pH 5, but was decreased at pH 8 (Chen *et al*, 2009). It can be explained as Ni is bound to ion-exchange sites or specifically absorbed or adsorbed or is co-precipitated with aluminium (Al) and iron (Fe) hydroxides in neutral or alkaline soils while in acidic organic rich soils where fulvic and humic acids are formed by the decomposition of organic material, nickel is reported to be quite mobile because of complex formation with these ligands and also due to higher solubility of these complexes (Nieminen *et al*, 2007). Secondary active transport of chelated Ni^{2+} has been reported with corresponding proteins that specifically bind Ni^{2+}, such as HoxN (high-affinity nickel transport protein, a permease), metallothionein and metallochaperones (Chen *et al*, 2009; Eitinger & Berthelot, 2000)

C. Nickel from essential to toxic

Ni deficiency can result in plant necrosis and its deficiency cannot be substituted by other metals such as Al, Tin (Sn), Cadmium (Cd) or Vanadium (V) (Eskew *et al*, 1984). Ni deficiency results in greatly reduced germination rates (i.e. 50% less than grain from Ni-adequate plants) and depressed seedling vigor of the viable grain. Grains containing less than 30ng per gram dry weight were non-viable. The essentiality of Ni was clearly demonstrated by Brown *et al* (1987) where barley plants failed to produce viable grain because of a disruption of the maternal plant's normal grain-filling and maturation processes that occur following formation of the grain embryo. The observations that barley plants fail to complete their life cycle in the absence of Ni and addition of Ni to the growth medium completely alleviates the deficiency symptoms in the maternal plants satisfies the essentiality criteria of Ni. Since it comes under the category of micronutrients, presence of more than its requirement causes toxicity as other heavy metals do. In general, naturally occurring concentration of Ni in soil and surface waters is lower than 100 and 0.005 ppm respectively but during the last decade its concentration has been reported up to 26,000 ppm in polluted soils and 0.2 mg/L in polluted surface waters i.e. 20–30 times higher than found in unpolluted areas (Yusuf M. *et al*, 2011).The range of nickel concentrations in plants averages 0.05-5 mg Ni/kg dry weight with concentrations above 50 mg Ni/kg dry weight being toxic. The critical toxicity level of Ni is more than 10 mg/kg dry mass (DM) in sensitive species, 50 mg/kg DM in moderately tolerant species and 1,000 mg/kg DM in Ni hyper-accumulator plants such as Alyssum and Thlaspi species (Kramer *et al*, 2000).

In today's scenario, much more concern about the toxicity of Ni is there than its deficiency. General signs of Ni phytotoxicity include reduced growth of roots and shoots, poor branching, deformation of various plant parts, decreased yield, leaf spotting, abnormal flower shape, mitotic root tip disturbance, germination inhibition, and chlorosis (Canadian Environmental Quality Guidelines, 1999). Ni toxicity basically originates in the soils due to anthropogenic activities such as smelting, burning of fossil fuel, vehicle emissions, disposal of household, municipal and industrial wastes, metal mining, fertilizer application, and organic manures. 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 (Yusuf *et al*, 2011)**.** Earlier, Ni toxicity was not of major concern because of its dual role in plant as a micronutrient and also due to its complex electronic chemistry. Excess supply of nickel produces phytotoxic effects and the problem of nickel toxicity acquires a serious concern because of agricultural use of sewage sludge that is rich in nickel and the industrial use of nickel production of Ni-Cd batteries which leads to discharge of nickel rich effluents (Coman *et al*, 2013). Excess Ni was reported to affect a number of biological and physiological processes resulting in an inhibition of plant growth. In addition, it can cause indirect toxic effects by replacing essential nutrients at cation exchange sites in plant (Crooke, 1958). Toxic effects of Ni have also been illustrated in Khan *et al* (2012), Di toro *et al* (1992), Yang *et al* (1996).

D. Nickel- ROS and Antioxidative system

Nickel is toxic only at higher concentrations. It's a known haematotoxic, immunotoxic, neurotoxic, genotoxic, reproductive toxic, pulmonary toxic, nephrotoxic, hepatotoxic and carcinogenic agent (Das *et al*, 2008). Being a heavy metal, it causes abiotic stresses leading to hazardous effects. A common consequence of heavy metal's toxicity is excessive accumulation of reactive oxygen species (ROS) which causes peroxidation of lipids, oxidation of protein, inactivation of enzymes, DNA damage and interaction with other vital constituents of plant cells. The major ROS are singlet oxygen ($^1O_2$), superoxide radicals (O_2^-), hydrogen peroxide (H_2O_2), and hydroxyl radicals ($OH^\cdot$). As Ni is a redox-active metal, it can directly generate ROS. However, it has been reported that Ni has the ability to produce O_2^- and also $OH^\cdot$ via Fenton/Haber–Weiss reaction. The Fenton/Haber–Weiss reaction follows the mechanism given in reaction 1, 2 and 3 respectively.

$$MX + O_2^- \longrightarrow M(X-1) + O_2 \quad \ldots (1)$$

$$2\,O_2^{\bullet-} + 2H^+ \longrightarrow H_2O_2 + O_2 \quad \ldots (2)$$

$$M(X-1) + H_2O_2 \longrightarrow MX + OH^\cdot + OH^- \quad \ldots (3)$$

The role of nickel (II) in causing oxidative DNA damage via Fenton reaction has been investigated (Valko *et al*, 2005) where experiments confirmed the formation of putative intrastrand cross-links, 8-hydroxydeoxyguanosine (8-OHdG) and single- and double-strand breaks.

In plant cells, ROS are generated during respiration and photosynthesis (Foyer & Harbinson, 1994). Usually ROS are generated when electrons get transferred from reduced electron's sources to oxygen as a sink. In respiration, complex I, II and III get oxidized by donating its electrons to complex IV. In between, electrons may deviate from their normal routes and cause a univalent reduction of O_2 to $O_2^{\bullet-}$, leading to generation of more ROS. The main site of leakage in respiratory electron transport chain is complex I which is NADH –co-enzyme Q (Sweetlove and Foyer, 2004). During photosynthesis, triplet chlorophyll facilitates the production of ROS mainly $O_2^{\bullet-}$. ROS may also originate from the reactions catalyzed by NADPH oxidases (Gapper and Dolan, 2006). These enzymes transfer electrons from cytoplasmic NADPH to O_2, which results in the formation of $O_2^{\bullet-}$. Moreover, pretreatment of wheat roots with NADPH oxidase inhibitors repressed Ni-induced increase in the production rate of $O_2^{\bullet-}$, confirming the implication of NADPH oxidase in O_2^- generation (Yusuf M *et al*, 2011).

Nickel does not induce ROS production to the extent as other heavy metals do, but its reactivity with ROS can be modulated by certain histidine and cysteine containing ligands (Das & Buchner, 2007). It has been reported that Ni^{2+} incubated with cysteine in an aerobic environment generates the $OH^\cdot$ radical, which then reacts with cysteine to generate a carbon-centered alkyl radical. It has also been observed that when Ni chelates with peptides containing glycylgycyl-L-histidine sequence it can peroxidize lipids through hydroxyl radical production (Yusuf M *et al*, 2011). The production of ROS by chelated Ni can be a possible mechanism of ROS in plant cells. Hence, free radical generation from the reaction of Ni (II)-thiol complexes and molecular oxygen or lipid peroxides plays an important role in the mechanism of Ni (II) induced toxicity (Das *et al*, 2008).

Suitable mechanisms are present in plant cells to maintain the steady state concentration of potentially toxic oxygen derived free radicals under normal physiological condition by cell's intrinsic antioxidant defense system.

However, enhanced generation of these reactive oxygen species (ROS) can impede cell's intrinsic antioxidant defenses. Ni toxicity affects not only the production of ROS but it also alters the antioxidant level in plant and animal cells (Hassan & Barakat, 2008). It interferes indirectly with a number of antioxidant enzymes for example, superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GSH-Px), glutathione reductase (GR), peroxidase (POD), guaiacol peroxidase (GOPX), and Ascorbate peroxidase (APX) (Madhava and Sresty, 2000). At low concentration of Ni, enhanced activity of antioxidants has been reported but at higher concentrations, reduced activities of many cellular antioxidant enzymes, both in vitro and in vivo has been reported resulting in altered capability of the plant to scavenge ROS, leading to ROS accumulation and finally oxidative stress (Yan *et al*, 2008; Maheshwari and Dubey, 2009). The induction of antioxidant system via Ni is well established. An in vitro study using Jatropha curcas L. embryos under nickel concentrations of 100, 200, 400 and 800 µM was done to observe the effects of high nickel concentrations on seedling growth. Activities of SOD, POD, CAT and phenylalanine ammonia-lyase (PAL) in the cotyledons were also examined. SOD activity was found to be increased significantly up to 400 µM and then decreased at 800 µM Ni. POD activities were induced remarkably at 100 and 200 µM, but the activity decreased with increasing nickel concentrations. Similarly, a negative link between CAT activity and nickel concentrations was observed. PAL activity had a positive correlation to nickel concentrations, and the highest activity was found at 400 µM nickel (Yan *et al*, 2008). Another study on effect of Ni on antioxidative enzymes was done where seven days old seedlings of black gram were subjected to different concentrations of nickel chloride ($NiCl_{2)}$ (20 µM, 40 µM, 60 µM , 80 µM and 100 µM) (Dubey and Pandey, 2011), indicating nickel induced oxidative damage in Black gram. Results suggested that treatment with different levels of Ni may enhance the antioxidative activities in leaves. Selvaraj *et al* (2010) also did a study in which morphogenic, biochemical and enzyme characters of Vigna radiata treated with different concentration of $NiCl_2$ were examined. Results demonstrated a decrease in biochemical parameters such as chlorophyll, carotenoids, soluble sugar and protein content with the increasing concentration of $NiCl_2$. The activities of antioxidative enzymes such as CAT and POD were found to be increased with increasing concentration of $NiCl_2$. A study was done on Radish (Raphanus sativus) to examine Ni toxicity and its recovery by boron and copper (Yadav *et al*, 2009) which included the evaluation of biometric parameters, chlorophyll, carotenoids, pheophytin, amylase, protein, sugar as well as activity of CAT and POD. The CAT and POD activities were found to be increased under the influence of Ni while pigmentation and protein level was decreased. But when copper was applied in combination with nickel, antioxidative activities were reduced while the opposite effect was observed with boron. The effect of Ni toxicity on antioxidative system was also reviewed in an another study where activities of SOD, CAT, APX, GPX and GR along with H_2O_2 content, lipid peroxides, proline content and protein thiolation were studied in the roots and leaves of barley plants (Kumar *et al*, 2012). A significant increase in GPX, APX, SOD and GR activities in leaves and roots were observed under 200 µM and 400 µM nickel treatments. Significant change in lipid peroxide content was also observed. Ni induced depletion of low molecular weight proteins, such as GSH, may contribute to the induction of oxidative stress in plants. It has been observed that exposure of maize and pigeonpea to Ni provoked activation of antioxidant systems with a severe depletion in glutathione level (Yusuf M. *et al*, 2011).

CONCLUSION

Scientific studies have observed that nickel mediate toxicity in plant by competition with other metal ions or form chelate complexes with metal ligands. Consequences of nickel toxicity include deficiency of other metal ions and retarded seed germination, disruption of cell structure and wilting, ROS induction, metabolic disruption and ultimately growth inhibition and reduction in yields. To overcome nickel induced environmental damage, its sequestration from soil and water is required. More research in the area of nickel hyperaccumulative plants is needed so that nickel can be removed phytoremedically from the polluted sites.

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