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ESSENTIALITY, TOXICITY AND THE MECHANISM OF TOXICITY OF NICKEL IN PLANT

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ABSTRACT

Nickel pollutant, one of the important widespread heavy metal pollutants starts from natural as well as anthropogenic sources. At present, it is a fact that nickel is an essential element vital for the healthy growth of plants. It plays a crucial responsibility for the activities of certain enzymes, for substantiating proper redox state of a cell and many other physiological, biochemical and growth responses. The use of nickel in modern technology is gradually increasing that result in hastening consumption of nickel-containing product. Nickel compounds arrive in the environment during its manufacturing as well as its use. Their rapid accumulation in different locations of the earth is responsible for widespread nickel pollution. This review presents an analysis of the existing situation related to essentiality, effect and mechanism of nickel toxicity in plants. The investigation of the current scenario is extremely crucial for the realization of the extent of the crisis linked to nickel as an environmental toxicant.

KEY WORDS : Niockel, Micronutrient, Environmental hazard, Heavy metal, Plant

INTRODUCTION

Nickel is a silver-white metal available in different oxidation states (from -1 to +4), though the primarily available form in the biological system is [Ni(II)], +2 oxidation state (Denkhaus and Salnikow, 2002). Easy availability of a variety of alloys from nickel results in its rapidly increasing use of modern technologies. For the production of stainless steel, alloys of nickel and nickel cast iron together with objects, such as coins, electrical equipment, tools, machinery, artillery, ornaments and domestic utensils, a lion share of nickel are taken up. The utility of the compounds of nickel is linked with electroplating, electroforming, batteries of nickel-cadmium alkaline, mordant of dye, catalysts electronic equipment and other varied sources together with cement manufacture as well (Duda-Chodak and Blaszczyk, 2008). The technological advancements assure improved living standard, but raise new challenges on environmental safety. Unrestricted industrialization, as well as urbanization without adequate controls of emission and pollution abatement, has put the lives of the human being at the threat. Annual inflow of nickel in the human environment from natural and anthropogenic sources globallyis around 150 000 and 180 000 metric tons respectively (Kasprzak *et al.*, 2003). The important sources are discharged from the burning of fossil fuel, volcanic eruption, mining, industrial production, use and dumping of nickel compounds and alloys (Kasprzak *et al.*, 2003). The developing countries normally depend on agricultural and industrial development for their economic growth. They try to bypass the environmental protection guidelines to a greater extent due to their underdeveloped economic state (Ikhuoria and Okieimen, 2000; Sahu and Arora, 2008).

Nickel is a micronutrient which is indispensable for the growth and development of the plant. It is also a component of the many enzymes including urease which is responsible for the metabolism of nitrogen in higher plants. Nevertheless, the paucity of nickel influences the metabolism of carbohydrate

(Anke *et al.,* 1984). It is a fact that nickel is toxic beyond the permission level to plants.

The greater part of the early works of literature focus on the toxicity and carcinogenic effects of particulate nickel compounds on ore miners. The area of investigation has restricted in toxicity of nickel carbonyl (used in cleansing) and immunological effects of dermatitis mediated by the nickel. Similarly, nickel toxicity to plants is also well documented (Yusuf *et al.,* 2011). Until recently, the toxicity of nickel in microorganisms thought as a problem restricted to cells exposed to industrial pollution or found in naturally occurring nickel-contaminated soils (Babich and Stotzky, 1983). It is the point of time to think about the way of the remedy of nickel toxicity. For this, we should have a lucid idea about the nature of toxicity and it magnetite in the living kingdom. This review article will help to conceive an in-depth conception on the effect of nickel in plant. From which creation of adequate awareness will be achieved and the way of its proper remedy will have come across.

Essentiality of Nickel in Plants

In 1987, nickel has recognized as a nutrient essential for the conclùsion of the life cycle of the plant (Brown *et al.*, 1987a). Now, the essentiality of this heavy metal for higher plants is well-established (Brown *et al.,* 1987a,b; Chen *et al.,* 2009; Harasim and Filipek, 2015). Nickel deficiency, also responsible for the hindrance of embryonic growth of barley plant leads to the declined the ability to produce viable seeds (Brown *et al.,* 1987b). Several embryonic anomalies, including poor or undeveloped root, impairment of endosperm development with declining dehydrogenase activities may result for deficiency of nickel (Seregin and Kozhevnikova 2006).

It is naturally present as an important component of metalloenzyme e.g., ureases (Aziz *et al.,* 2007). Reduced hydrogenase activity in symbiotic *Rhizobium leguminosarum* affects the symbiotic N_2 fixation is also reported due to nickeldeficiency (Ureta, 2005; Zobiole *et al.,* 2009). Nickel in stress amount augments the growth and yield of plants and vital for the biosynthesis of anthocyanins (Ragsdale, 1998; Lopez and Magnitskiy, 2011). It can persuade plant resistance to disease through the controlling of the creation of secondary plant metabolites resulting in increased productivity (Wood and Reilly, 2007). Through interacting with the iron of hemoglobin, nickel helps in oxygen transportation and stimulation of the metabolism and thus it may regard as a key metal in enzyme systems (Khan and Moheman, 2006).

Nickel does involve in the transmission of genetic code and presence in some of the enzyme systems for sugar metabolism (Khan and Moheman, 2006). For the transportation of nitrogen to seeds, nickel plays a considerable role (Brown *et al.,* 1987a,b). Nickel scarcity may inhibit the activity of urease. This inhibition may outcomes as a buildup of the concentration of urea at toxic levels which is expressed as necrosis of leaf tips or chlorosis of elderly leaves (Seregin and Kozhevnikova, 2006).

Insufficiency of nickel may lead to interference of the citric acid cycle resulting in declined the concentration of citrate. The distraction of carbon metabolism is pursued by a buildup of the concentration of lactic and oxalic acids consequences in mouse ear symptom (Bai *et al.,* 2006). From the above information, it may be concluded that nickel plays vital roles in various physiological processes, from the germination of seed followed by vegetative growth and culminating eventually to the seed maturation. Thus, for successful completion of a life cycle, adequate supply of this metal is inevitable for the plant. Consequently, nickel gets an opportunity to enlist itself in the list of essential micronutrients (Marschner, 2002).

Toxicity of NICKEL on plant system

Effect on the photosynthesis

Nickel at its elevated concentration considerably reduces the content of chlorophyll through protein inhibition (Lin and Kao, 2007; Zhou *et al.,* 2009). A report reveals that about 47% decrease in the content of chlorophyll at the 0.025 mM nickel concentration in comparison to the control plant (Wheeler *et al.,* 2001). Baccouch *et al.* (1998) have reported about the decreased content of chlorophyll-a, chlorophyll-b with 70% and 50% respectively, under the nickel stress of 100 ìM in maize. The amount of carotenoid is also decreased owing to nickel toxicity (Singh *et al.,* 2012). Toxicity of the heavy metal may also cause the demolition of the photosynthetic apparatus as it crumples the architect of grana of a chloroplast and also its size (Chen *et al.,* 2009). Toxic effect of nickel at a higher concentration on the photosynthetic apparatus and photosynthetic ability is well established. Ni^{2+} at elevated concentrations interrupt the function of effects light-harvesting complex II (LHCII) and impairs the synthesis of photosynthetic

pigments (Molas, 2013).

Nickel toxicity is responsible for the reduction of the numbers and size of the chloroplast, disorganization of ultrastructure with the slacken numbers of the grana and the thylakoids as well as altered membrane lipid composition. Ni^{2+} induced oxidative stress induces peroxidation of membrane lipids (Molas, 2013). A decline of chlorophyll level and disruption of electron transport in photosynthesis due to nickel toxicity was confirmed by the work of Sreekanth *et al.,* (2013). Increasing concentration of nickel impairs protein complexes of photosynthesis and gradual fall at the rate of Hill reaction in maize (Ghasemi *et al.,* 2012).

Reduced in stomatal conductance and photosynthetic activity due to nickel stress has observed in sunflower (Bazzaz *et al.,* 1974) which was confirmed on wheat by Ouzounidou *et al.,* (2006). The nickel stress of 200 µM to Poplar (*Populusnigra*) plants significantly decreases the stomatal conductance especially in emerging leaves from 0.40 to 0.03 mol $m^{-2}s^{-1}$ resulting a direct decrease in photosynthesis (Velikova *et al.,* 2011).

Effect on mineral nutrition

Mineral nutrient contents in plant organs may increase, decrease, or stay even in the presence of nickel. Decrease uptake of macro- and micronutrients may have resulted from the competition of Ni^{2+} with other cations for the common binding sites. Comparable ionic radii of Ni^{2+} (78 pm) with other cations such as Mg^{2+} (78 pm), Fe^{2+} (82 pm), and Zn^{2+} (83 pm) is liable for such competitive operation (Emsley, 1991; Barsukova and Gamzikova, 1999). Elevation of concentration of nickel 50 to 200 mg/kg of soilcan decrease Mg and Cu in the caryopses and Mg and Ca in the shoots of *T. aestivum* (Barsukova and Gamzikova, 1999). At the nickel overloaded environment, decrease uptake of Mg and Fe may responsible for chlorosis (Khalid and Tinsley, 1980; Piccini and Malavolta, 1992).

Metabolic disorders mediated by nickel may be linked with the declined uptake of nutrient that perturbs the structure and enzyme activities of cell membranes (Seregin and Ivanov, 2001). Ros *et al.* (1990) find its reflection in the shoot of *Oryza sativa* where nickel affects the composition of sterol and phospholipid of the plasma membrane and also the activity of ATPase. Thus, the membrane permeability is affected causing impairment of ion stability within the cytoplasm.

Water relation

Equilibrium between transpiration and uptake of water determines the water constancy in plants. Nickel treated plants are related to changes in water balance. The water content of a plant, both in dicot and monocot decline at the uptake of nickel beyond the permissible concentration (Yadav, 2010). Fall in the absorption of water is a parameter for the progress of nickel toxicity in plants (Pandey and Sharma, 2002; Gajewska *et al.,* 2006). Various parameters such as water uptake, movement of water along the apoplast and symplast and functioning of stomata etc. at multiple levels are the toxic effects of heavy metals (Barcelo and Poschenrieder, 1990).

Nickel toxicity is responsible for the reduction in the area of transpiration plane (leafs blades) of plants (Chen *et al.,* 2009). The reduction in leaf surface of *Cajanuscajan* under the nickel stress of 1 mM in nutrient solution has found as 40% (Sheoran *et al.,* 1990). This finding corroborates with the reports of Molas (1997) in *Brassica oleracea* grown in agar medium under 5.2 gm^{-3} $NiSO_4,7H_2O$. Reduction of transpiration rate and shutting of the aperture of stomata is the primary impact of toxicity of heavy metals (Molas, 1997; Seregin and Ivanov, 2001; Roy and Saha, 2019).

Molas (1997) opines that the increase of the concentration of abscisic acid (ABA) level is accountable for stomata closing in the leaf tissues of *P. vulgaris* under the nickel stress. Rao and Sresty (2000) have revealed that the nickel toxicity increases the creation of reactive oxygen species (ROS) causing peroxidative damage in membrane lipids. Over accumulation of lipid peroxidation resulted in HMs toxicity and oxidative damage (Pandolfini *et al.,* 1992; Luna *et al.,* 1994; Chaoui *et al.,*1997). Gajewska and Sklodowska (2007) have observed that nickel stress is responsible for the significant increase of H_2O_2 quantity in the leaf of wheat.

Effect on the seed germination

Germination of seed is the first physiological process of plant life. It is regulated by hormonal interactions which are dependent on the environmental factors and become effective only at favorable conditions which may harshly be affected by heavy metals (Iglesias and Babiano, 1997). Nickel beyond permissible levels may affect amylase, protease and ribonuclease activity that impairs the digestion and

mobilization of food reserves in germinating seeds, thus causing hindering its germination (Ahmad and Ashraf, 2011). Nickel in a solution of 1.5 mM reduce the germination rate of pigeon pea (*Cajanuscajan*) seed by 20% and the inhibition rate of germination is in magnitude to the concentration of nickel (Rao and Sresty, 2000).

On the other hand, in germinating seeds of *Brassica nigra*, it is reported that nickel in combination with NaCl conspicuously impairs pigments, photosynthetic machinery, leaf water, and potential growth by increased leakage of electrolyte, peroxidation of lipid, H_2O_2 content, the action of anti-oxidative enzymes and the concentration of proline. Decrease stability of membrane, effectiveness of the nitrate reductase as well as carbonic anhydrase due to nickel toxicity is also documented (Yusuf *et al.*, 2012).

Mechanism of toxicity of NICKEL ON plant

In spite of works of literature about the phototoxicity of nickel is widely available the detailed mechanisms are still not clear (Gajewska and Sklodowska, 2007). Nickel is not an active or redox metal thus its toxicity is likely to be based on indirect mechanisms. Toxicity of nickel induces through interfering with other essential metal ions and creation of oxidative stress (Fig. 1).

Interference with other essential metal ions

The characteristic of nickel has resemblance with a variety of other metals (such as K, Na, Ca, Mg). Accordingly, nickel may compete with these metals in the absorption and transportation (Kochian, 1991; Kupper *et al.*, 1996). Nickel at its elevated concentrations may hinder the absorption of these metals through competition leading to decreasing their concentration leading to their insufficiency in plants (Van Assche and Clijsters, 1990; Rubio *et al.*, 1994; Ahmad *et al.*, 2007). Consequently, this may impair the vital physiological processes and finally causes toxic effects (Gajewska *et al.*, 2006; Gon_alves 2007). For instance, through competition nickel can reduces Mg (or Fe) uptake and its availability to aerial parts and then bring insufficiencies of these elements in plants.

Nickel is capable to form stronger complexes with many ligand-forming elements. Thus, capable to displace the metal that forms weaker complex than that of nickel (Irving and Williams, 1984). Consequentially ultimately resulting in the abnormalities in germination, growth suppression and reductions in yields (Rao and Sresty, 2000; Seregin and Kozhevnikova, 2006). It is possible to diminish these inhibitions of nickel through supplementing of Mg (or Fe) ions to the concerned

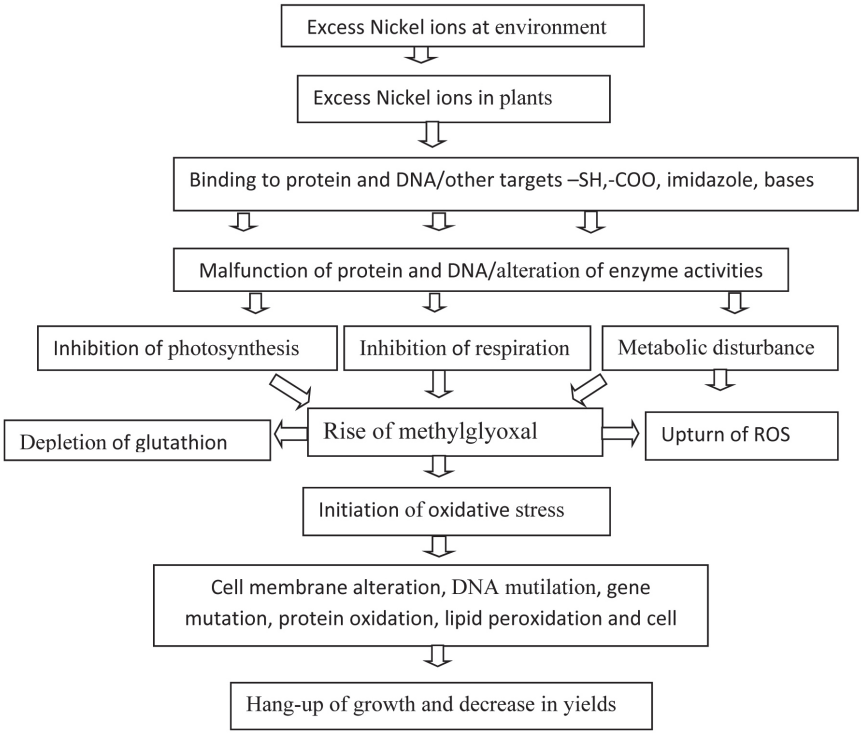

Fig. 1. Molecular mechanism of nickel toxicity in plant

plant (Ouzounidou *et al.,* 2006; Gon_alves, 2007). Therefore, nickel toxicity in plants is partly due to the interference of the nickel with other essential metal ions.

In addition, the toxicity of this heavy metal on the development of root in *Alyssum bertoloni*are timed down due to Ca^{2+} (Gabbrielli and Pandolfini, 1984). On the contrary, Cu seems to make stronger the toxicity of nickel in terms of reduced vigor and growth of Scots pine (Nieminen, 2004). A number of metalloenzymes, e.g., SOD and CAT contain Fe, Cu, Zn, or Mn in their prosthetic groups. Nickel at its surplus concentration is capable to diminish the contents of Fe (Pandey and Sharma, 2002) as well as that of Cu and Zn (Parida *et al.,* 2003) in plant tissues. From this, the idea may be conceived that this heavy metal may trim down the biosynthesis of these metalloenzymes by causing a shortfall of these crucial metals (Gajewska *et al.,* 2006). Experimental studies on the plant photosynthesis advocate that nickel can competitively wipe out the Ca ions from its binding site in the oxygen evolving complex (Boisvert, 2007) and displace the Mg ion from chlorophyll (Kupper *et al.,* 1996; Souza and Rauser, 2003; Solymosi, 2004) which may ultimately impede the PSII electron transport chain.

Induction of oxidative stress

Another means of nickel toxicity is linked to oxidative stress (Rao and Sresty, 2000; Gonnelli *et al.,* 2001; Boominathan and Doran, 2002; Gajewska *et al.,* 2006). Nickel beyond its permissive level leads to the considerable rise in the concentration of hydroxyl radicals, superoxide anions, nitric oxide and hydrogen peroxide (Stohs *et al.,* 2001; Boominathan and Doran, 2002; Hao *et al.,* 2006). As nickel is non-redox-active metal, thus it is incapable to create ROS directly. However, it impedes indirectly with many antioxidant enzymes. Among which SOD, CAT, GSH-Px, glutathione reductase (GR), guaiacol peroxidase (GOPX), peroxidase (POD), and ascorbate peroxidase (APX) are noteworthy (Pandolfini *et al.,* 1992; Baccouch *et al.,* 2001; Pandey and Sharma, 2002; Gajewska and Sklodowska, 2005). Increase performance of SOD, POD, GR, and GOPX augment the activation of supplementary antioxidant defenses has been found in plants due to exposure to nickel at low concentrations (0.05 mM). To that of short time exposure, it leads to the scavenging of ROS (Freeman 2004; Gomes-Juniora, 2006).

In the contrary, excess nickel involves in decreasing the functionality of many cellular antioxidant enzymes, *in vitro* as well as *in vivo*. Eventually, the plant's capability to scavenge ROS, leading to ROS accumulation and finally oxidative stress in plants is slowed down (Del Carmen *et al.,* 2002; Zhao *et al.,* 2008). The performance of antioxidant enzymes is coupled with the duration and type of stress treatment, plant species as well as parts of plant involved. Nickel has also been made known to enhance the plasma membrane (PM) NADPH oxidase, which has shown to be concerned in nickel-induced ROS creation in young roots of wheat seedlings (Hao *et al.,* 2006). ROS is involved with the mutilation of cell membrane, proteins, lipids as well as DNA (causing, inter alia, oxidation of bases DNA, DNA and protein cross-links, creation of DNA gaps and breaks), resulting in lipid peroxidation (Baccouch *et al.,* 2001; Boominathan and Doran, 2002), developmental imperfections and genetic unsteadiness in plant species (Oller *et al.,* 1997; Bal and Kasprzak, 2002; Papazoglou *et al.,* 2005).

CONCLUSION

Even though nickel is a heavy metal, it is necessary for plants to lead a successful life. Nickel in acceptable quantities has crucial roles in morphological and physiological functions, starting from germination to the yield of the plant. Nickel has drawn much attention as an intoxicating pollutant for the growing anthropogenic stress on the environment. This heavy metal at the higher concentration causes enzymatic inhibition of a good number of enzymes, resulting in a decrease in their activity. Due to these enzymatic inhibitions, the normal biogeochemical cycle is badly affected. Nickel intoxication in plants causes stunted growth, necrosis of leaves, mouse ear disorders and reduction of total plant yields. Nickel beyond it permissible level may show the way to develop deficiencies of other essential minerals. It may come about by means of competition and/or creating complexes with metal ligands results in reduced germination, undersized plant growth, initiation of chlorosis and wilting of leaves. Moreover, nickel toxicity may alter in enzymatic activities, metabolic perturbations through oxidative stress, interrupt photosynthesis and eventually incite growth inhibition and yield reductions. From the existing works of literature, some justifications about the interaction of nickel with other metal ions, its

uptake, transportation, translocation and accumulation within the plant bodies have been unearthed. Still, it desires to look-for on the molecular level. Excess nickel brings on oxidative stress however mechanism generating nickel toxicity at the protein and molecular level may be explored in detail. Nickel pollution in the environment has led to researches on the emerging fields, like phytoremediation (i.e. the employing hyper-accumulators or wetland plants to eliminate and/or sequester nickel from soil and water). However, a number of such plants have partialefficacy for phytoremediation, as their seasonal growth, slow growth rate, difficult propagation, and low biomass. Solutions to this problem are important and need further research.

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