International Journal of Chemical Studies

P-ISSN: 2349–8528
E-ISSN: 2321–4902
IJCS 2019; 7(2): 1185-1191

Received: 15-01-2019
Accepted: 19-02-2019

Abhik Patra
Department of Soil Science and Agricultural Chemistry, Institute of Agricultural Sciences, Banaras Hindu University, Varanasi, Uttar Pradesh, India

Asik Dutta
Crop Production, Division, ICAR-Indian Institute of Pulse Research, Kanpur, Uttar Pradesh, India

Surendra Singh Jatav
Department of Soil Science and Agricultural Chemistry, Institute of Agricultural Sciences, Banaras Hindu University, Varanasi, Uttar Pradesh, India

Saroj Choudhary
Department of Soil Science and Agricultural Chemistry, Institute of Agricultural Sciences, Banaras Hindu University, Varanasi, Uttar Pradesh, India

Arghya Chattopadhyay
Department of Soil Science and Agricultural Chemistry, Institute of Agricultural Sciences, Banaras Hindu University, Varanasi, Uttar Pradesh, India

Correspondence
Asik Dutta
Crop Production, Division, ICAR-Indian Institute of Pulse Research, Kanpur, Uttar Pradesh, India

Horizon of nickel as essential to toxic element

Abhik Patra, Asik Dutta, Surendra Singh Jatav, Saroj Choudhary and Arghya Chattopadhyay

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: Environment; essential element; nickel; plant nutrition; soil; toxic to plant

1. 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. In recent years, Ni pollution has been reported from across the world, including Asia (Zhao *et al*., 2008) [90], Europe (Papadopoulos *et al*., 2007) [57] and North America (Kukier *et al*., 2004) [40]. Nickel is a trace metal added to the environment from both natural and anthropogenic sources (WHO, 1991) [87]. Nickel's natural source to the environment includes weathering of rocks, forest fires, volcanic emissions and wind-blown dust (Iyaka, 2011) [35]. 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) [4]. Nriagu (1990) [53] reported that industrial emissions of nickel amount to more than 100 times that from natural sources. Nickel concentrations may reach 26000 mg kg^{-1} in polluted soils (Alloway, 1995) [4] and 0.2 mg L^{-1} in polluted surface waters (Zwolsman and Bokhoven, 2007) [92].

The higher concentrations of Ni in plant cells results in alterations at the physiological, biochemical and cellular levels leading to the severe damage to plant (Singh *et al*., 2015) [77]. The phytotoxic city of various heavy metals differs and the order of toxicity in plants reveals $As^{5+} < As^{3+} < Cr^{6+} < Co^{2+} < Ni^{2+} < Cu^{2+} < Ti^{+} < Hg^{2+} < Cd^{2+} < Ag^{+}$. The most common symptoms of Ni^{2+} toxicity in plants are inhibition of photosynthesis (Chen *et al*., 2009) [16] and mitotic activities (Rao and Sresty, 2000) [63], inhibit sugar transport (Ali and Sajad, 2013) [3], reductions in plant growth (Molas, 2002) [49], adverse effects on fruit yield and quality (Gajewska *et al*., 2006) [30] and induction of chlorosis, necrosis and wilting (Pandey and Sharma, 2002) [56]. Extremely high soil Ni concentrations have left some farmland unsuitable for growing crops, fruits and vegetables (Duarte *et al*., 2007) [21].

Although many reports have focused on the toxic effects of Ni on plants, our knowledge of its toxicity is incomplete, and the detailed mechanisms involved are poorly understood. Keeping in view the increasing Ni^{2+} toxicity to crop plants and significant importance of cereals, oilseeds, grain legumes and vegetables as source of low cost food, the present article discusses various aspects of Ni toxicity to essentiality.

2. Nickel in the Environment

Nickel is the 24th most abundant metal in the earth's crust and 5th most abundant element by weight after iron, oxygen, magnesium and silicon, constituting about 3% of the earth composition. The core of the earth contains 8.5% nickel, deep-sea nodules 1.5% and meteorites have been found to contain 5–50% nickel (Fox *et al*., 1990) [25]. It has several oxidation states ranging from -1 to +4, but its bivalent (Ni^{2+}) form is the most common in biological systems. Nickel occurs either as a free metal in igneous rocks or in combination with irons (Sachan and Lal, 2017) [68]. In general, naturally occurring concentration of Ni in soil and surface waters is lower than 100 and 0.005 ppm, respectively (McGrath and Smith, 1995) [44]. Total Ni concentration commonly ranged from 5 to 500 mg kg^{-1}, with an average of 50 mg kg^{-1} in soils (Wilson and Benow, 1978) [86].

2.1 Air

Currently, almost 90% of the global anthropogenic Ni emissions originate from oil combustion (Pacyna and Pacyna, 2001) [55]. More specifically, sources of nickel emissions into the air include coal and oil burning for power and heat, waste and sewage sludge incineration, mining and steel production industries, and electroplating (WHO, 1991) [87]. Typical average levels of airborne nickel are: 0.00001-0.003 μg m^{-3} in remote areas; 0.003–0.03 μg m^{-3} in urban areas having no metallurgical industry; 0.07-0.77 μg m^{-3} in nickel processing areas. In Poland, the recommended nickel concentration in the atmospheric air is set as 0.025 μg m^{-3} (Bencko, 1983) [9]. In 1979, atmospheric nickel concentrations from fossil fuel combustion and automobiles were estimated to be about 120-170 ng m^{-3} in industrialized regions and large cities (Norseth and Piscator, 1979) [52]. Recent U.S. estimates of atmospheric nickel associated with particulate matter with a mean diameter of 2.5 μm (PM2.5) are in the range of 0.002-0.01 μg Ni m^{-3} (Chen and Lippmann, 2009) [17].

2.2 Water

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. Drinking water generally contains nickel at concentrations less than 10 μg L^{-1}. Average total nickel concentrations in drinking water ranged from 3-7 μg Ni L^{-1}, with concentrations up to 35 μg Ni L^{-1} occasionally encountered (Andersen *et al*., 1983) [5]. Nickel levels in natural waters have been found to range from 2 to 10 μg L^{-1} in fresh and tap water and from 0.2 to 0.7 μg L^{-1} in marine water (Rojas *et al*., 1999) [65]. In areas of nickel mining, however, up to 200 μg Ni L^{-1} in drinking water have been recorded (McNeely *et al*., 1972) [46]. The primary source of nickel in drinking water is leaching from metals which are in contact with drinking water, such as pipes and nickel may also be present in some ground water as a consequence of dissolution from nickel ore bearing rocks (Alloway, 1995; Salt, 1999) [4, 69]. The concentration of Ni in river water and in sediments of upper Ganges (India), has been estimated to be between 35–211 and 70,900–511,000 ppm, respectively (Israili, 1992) [34].

2.3 Soil

Nickel can exist in soils in several forms: inorganic crystalline minerals or precipitates, complexed or adsorbed on organic cation surfaces or on inorganic cation exchange surfaces, water soluble, and free-ion or chelated metal complexes in soil solution (Bennett, 1982) [10]. In soil, nickel has been found to vary widely in concentration, from 3 to 1000 ppm (Iyaka, 2011) [35], with the total abundance in the Earth's crust being about 84 mg g^{-1}. The Ni status of soils is highly dependent on its contents in parent material. However, the concentration of Ni in surface soils reflects the additional impact of both soil-forming processes and anthropogenic activities (Kabata and Pendias, 2001) [38]. The lowest contents are found in sedimentary rocks that comprise of clays, limestones, sandstones and shales, while the highest concentrations exist in basic igneous rocks (Kabata- Pendias and Mukherjee, 2007) [37]. Industrial waste materials, lime, fertilizer and sewage sludge constitute the major sources of nickel into soils (Mcllveen and Negusanti, 1994) [45]. Farm soils contain approximately 3–1,000 mg kg^{-1} Ni soil, but the Ni 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. Near to some nickel refineries or in dried sludge, soil levels of nickel have been found to be 24,000-53,000 ppm, up from the nonindustrial average level of 500 ppm (EPA, 1990) [22]. The mean content of nickel in soil affected by the Bolesław Mining and Metallurgical Plant was 19.62 mg kg^{-1} (Trafas *et al*., 2006) [81].

3. Essentiality of Ni in plants

The response of Ni application to field crops (potato, wheat, beans) was first evident in 1945, but its essentiality was not conclusively demonstrated until 1987 (Brown *et al*., 1987a) [13]. Subsequently other researchers demonstrated the positive responses of several crops, viz. cowpea, tomato, barley and oats to Ni application under controlled conditions (Walker *et al*., 1985; Brown *et al*., 1987b) [85, 14]. Eskew *et al*. (1983) [23] reported that Ni-deficient soybean accumulates toxic levels of urea in its leaflet tips because of depression in urease activity in leaves. The discovery in 1975 that nickel (Ni) is a component of the enzyme urease (Dixon *et al*., 1975) [20], which is present in a wide range of plant species led to bring of a new era of research about the role of Ni in higher plants. The establishment of nickel as an essential element, however, highlights the limitations of the current definition of essentiality of nutrients as applied to plants (Arnon and Stout, 1939) [6]. Essentiality of nickel was subsequently established in 1987, when Brown *et al*. (1987a) [13] demonstrated that barley (*Hordeum vulgare* L. cv. 'Onda') could not complete its life cycle in the absence of added nickel, even when plants were supplied with a nonurea source of nitrogen. Nickel is the 17th element recognized as essential for plant growth and development (Liu, 2001) [42]. Nickel is now generally accepted as an essential ultra-micronutrient as its requirement is the lowest of all essential elements at < 0.5 mg kg^{-1} of dry weight (Marchner, 1995) [43]. It plays important role in various metabolic processes including ureolysis, hydrogen metabolism, methane biogenesis and acetogenesis (Mulrooney and Hausinger, 2003) [51]. Nickel deficiency is also found associated with the reduced symbiotic hydrogenase activity in *Rhizobium leguminosarum* that may directly affect the symbiotic N_2 fixation (Zobiole *et al*., 2010) [91]. Nickel is a constituent of eight metalloenzymes, e.g., glyoxylase (EC 4.4.1.5), acireductone dioxygenase (EC 1.13.11.54), Ni-superoxide dismutase (EC 1.15.1.1.), hydrogenase (EC 1.12.98.2), methyl reductase (EC 2.8.4.1), carbon-monoxide dehydrogenase (EC 1.2.99.2), acetyl coenzyme-A synthase (EC 2.3.1.169) and urease (EC 3.5.1.5) (Harasim and Filipek, 2015) [33]. A few reports demonstrate that Ni supply increases the yield of crop plants (Sabir *et al*., 2011; Kumar *et al*., 2018) [67, 41].

4. Nickel toxicity to plants

Nickel toxicity has become a particular concern, due to its increased industrial use. Its concentrations in polluted soil may reach levels 20 to 30 fold higher (200–26,000 mg kg^{-1}) than the range typically found in natural soils (10–1,000 mg kg^{-1}) (Izosimova, 2005) [36]. Nickel toxicity levels vary widely between 25 to 50 ppm (Mishra and Kar, 1974) [47]. According to Ochiai (1977) [54] 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. The toxic symptoms generated by Ni include chlorosis, necrosis (Pandey and Sharma, 2002) [56], inhibition of shoot and root growth and decrease in leaf area (Shaw *et al*., 2004) [74]. Elevated concentrations of Ni can inhibit cell division at root meristems in non-tolerant plants (Robertson and Meakin, 1980) [64], and decrease plant growth (Foy *et al*., 1978) [26]. Excess Ni^{2+} also affects nutrient absorption by roots (Rahman, 2005) [62] and inhibits photosynthesis, transpiration and transport of photo assimilates from leaves (Seregin and Kozhevnikova, 2006; Shi and Cai, 2008) [73, 76]. High uptake of Ni^{2+} induced a decline in water content of dicot and monocot plant species (Dimkpa *et al*., 2008) [19]. The decrease in water uptake was used as an indicator of Ni^{2+} toxicity in plants (Gajewska *et al*., 2006) [30].

4.1 Growth and development

Nickel is an essential element which plays a vital role in the plant growth but, an elevated dose is deleterious for crop health. Zhang *et al*. (2007) [89] reported *Alyssum murale*, a common hyperaccumulator shed their high Ni content leaves which inhibit the seed germination of the adjacent plant. Germination percentage of pigeonpea subsided by 20% in 1.5 mM Ni solution and the effect can be multiplied with proportionate increase in Ni concentration in solution (Rao and Sresty, 2000) [63]. Similarly, in case of wheat the germination percentage tail off by 6% by increasing Ni concentration from 100 to 200 mM (Gajewska and Sklodowska, 2008) [29]. The justification for this deleterious impact of Ni on seed germination and seedling growth is disruption in the cellular elasticity and cellular expansion also it arrests the enzymatic activity (Seregin and Kozhevnikova, 2006) [73]. The negative impact of heavy metals is paramount at the roots due to their direct contact with the soil solution than any other above ground part (Panday and Sharma, 2002) [56] and this harmful impact can be very much prominent in excluder plant species (Seregin *et al*., 2006) [73]. Nickel content up to (10 µM) had no significant impact on root growth but, upon elevating the dose by 20 times we can see a significant inhibition on root growth and development in wheat (Gajewska *et al*., 2006) [30]. Due to lack of scientific evidences very poor information is available showing the impact of Ni on shoot growth and development. Gajewska and Sklodowska (2007) [28] showed that at 100 µM Ni^{2+} chlorotic and later necrotic symptom's appeared in the shoot causing growth reduction. Application of Ni in 0.1 mM dose for 2 weeks caused distinct chlorosis and necrosis in leafs (Rahman *et al*., 2005) [62]. The feasible reason may be uneven cell elongation. Higher biomass allocation is the key point to achieve higher yield and proper source to sink relation is the basis of this. Alam *et al*. (2007) [2] showed 100 µM of nickel reduced the root: shoot ratio and biomass production in *Brassica juncea*. So, as an end note we can say that inhibitory role of Ni or any other heavy metal is due to blocking of general metabolic disorder in plants and direct role in cell division. But, the molecular level interaction of Ni is still to be resolved.

4.2 Mineral nutrition

As mentioned in the previous section Ni holds last but not the least position from plant nutrition point of view. Nickel plays quite similar role like other secondary and micro elements like Ca, Mg, Mn, Fe, Cu and Zn and for this reason it can modify the uptake of others (Yusuf *et al*., 2011) [88]. As the ionic radii of Ni (78 pm) is nearly equal to other above mentioned nutrients like Mg, Fe, Zn, it can compete for the adsorption sites in the soil and following utilisation by the crops (Seregin and Kozhevnikova, 2006) [73]. So, it can be speculated that chlorosis in the leaves may be due to Ni induced Fe or Mg deficiency. So, to alleviate the Ni toxicity need based supplementation of Fe or any other should be done (Goncalves *et al*., 2007) [32]. Apart from this the detrimental impact is may be due to disruption in the cell membrane structure specifically membrane permeability, ceasing regular enzymatic activity and changing the ionic balance of the cytoplasm (Seregin and Ivanov, 2001) [72]. Ros *et al*. (1992) [66] in rice reported the adverse effect of Ni as it kindle the sterol and phospholipid composition of cell membrane collaterally altering ATPase activity. Cationic micronutrients like Fe, Cu, Zn, Mn acts as a prosthetic group in many enzymes like peroxidase, superoxide dismutase (SOD), catalase (CAT), alcohol dehydrogenase etc. (Panday and Sharma, 2002) [56]. Nickel can compete with many cationic micro nutrients and lessening their content in the plant tissue, thus it can be expected that the biosynthesis of metalloenzymes will be hampered (Gajewska *et al*., 2006) [30]. Sometimes, even low Ni concentration (1-10 µM), the mineral nutrient uptake remains same even increased, this is due to concentrating effect; repercussion is lowering of plant biomass in plants grown in solutions devoid of Ni, while metal absorption remains same in the control plots (Barsukova and Gamzikova, 1999) [8]. Even within the plant species, the deficiency of Ni can differ. Barsukova and Gamzikova (1999) [8] from their experiment reported that Ni concentration of 67 µM, in both *Triticum aestivum* and *Triticum durum*, the interveinal chlorosis due to Zn deficiency only appeared in *T. aestivum*, but not in *T. durum*. By and large it may be conclude that alteration in the cellular structure is the foremost reason of Ni toxicity hindering translocation of other essential nutrients.

4.3 Enzymatic activity and oxidative stress

Nickel plays a pivotal role both directly and indirectly in altering enzymatic activity in plants like the other important physiological processes (Van Assche and Clijsters, 1990) [82]. In rice shoots (*Oryza sativa* L.), under in-vitro condition, the Ni^{2+} induced Mg^{2+} deficiency caused poor functioning of ATPase in the cell membrane (Ros *et al*., 1992) [66]. Addition of 1mM nickel sulphate ($NiSO_4$) in beetroot (*Beta vulgaris* L.) pots abate the nitrate uptake as a consequence the genetic expression of NR genes hence enzymatic activity dwindled (Ros *et al*., 1992) [66]. Nickel concentration of 100 µM can significantly brought down the NR activity without modifying the activation state and it is very astounding that deleterious impact is more marked on nitrite reductive (NiR) than its counterpart (NR) (Gajewska *et al*., 2009) [31]. Nickel is having oxidative property only at higher concentration and numerous studies have been conducted to visualise the oxidative property of Ni in crops. As a by-product of various metabolic processes reactive oxygen species (ROS) such as, superoxide anion radical (O_2^-), hydrogen peroxide (H_2O_2) and singlet oxygen ($^1O_2$) are repeated producing in the plant tissue (Dat *et al*., 2000) [18]. Due to leakage in the electron transport system (ETS) of respiratory cycle, electrons generated reacts with oxygen (O_2) leads to O_2^- and within this cycle NADH-Co enzyme reductase complex I is the focal point (Møller, 2001)[50]. Another, source for the production of ROS is triplet chlorophyll facility (Foyer *et al*., 1994) [27]. Howbeit, ROS are more potent danger causing agent in compared to oxygen (O_2) for the living entity by causing damage to DNA, photosynthetic pigments like chlorophyll and desaturating the normal structure of biomolecules (Schutzendubel and Polle, 2002)[71]. Similar to other transitional metals, Ni have the propensity to generate -OH via Fenton/Haber–Weiss reaction but, as it is having a high redox potential ($Li^+ + e = Li$, $E_o = -3.04$ Volts) compared to others, it failed to be a good catalyst (Vanýsek, 2011) [83].

The Ni^{2+} effect on antioxidant enzymes of accumulator and non-accumulator plant species has a marked difference. Antioxidant enzymes like SOD, *Ascorbate peroxidase* and GR escalated in non-accumulator (*A. maritimum*) in lieu, the activity became nil in hyper-accumulator plant (*A. argenteum*) (Schickler and Caspi, 1999) [70]. So, passive we can conclude that resistant or so called hyper accumulator plants can detoxify the Ni^{2+} in their cytoplasm and remain safe even in the absence of antioxidant enzymes. As, previously discussed that Ni^{2+} chelates with histidine very strongly, it could peroxidise lipids through –OH radical production via Haber–Weiss reaction which is much observable in the plant cells (Torreilles and Guerin, 1990) [80]. A complex enzymatic and non-enzymatic antioxidant systems such as CAT in peroxisome, ascorbate peroxidase in apoplast, SOD in the plants slake the harmful effect of ROS like conversion of O_2^- or H_2O_2 to water etc. (Pitzschke *et al*., 2006) [60]. The induction of anti-oxidant system plants due to Ni^{2+} is well documented especially on the crops like corn and pigeonpea (Baccouch *et al*., 2001) [7]. An exception over this like activity suppression of CAT in 200 ppm is seen in sunflower (*Helianthus annus* L.) by 5 fold while the activity of polyphenol oxidase surged by 8 times (Pillay *et al*., 1996) [59]. Based on these details we can conclude that elevated dose of Ni can increase the anti-oxidant enzymatic activity while the other enzymatic activity reduced. The root cause of this event is still not clear as many believes that substitution of metals from the binding site or concomitantly some series of events mediated a particular genetic expression or modifying the substrate pool. Future research with graded Ni dose over a set of crops can unfold the mystery behind the role of Ni on plant metabolic and enzymatic activity.

4.4 Effect on Photosynthesis

Photosynthesis is defined as the process by which green plants produce their food by using CO_2, water in the presence of sunlight. So, the chloroplasts present in the green leaves serves as kitchen in higher plants which may be affected by heavy metal toxicity by both directly and indirectly. The fatalistic impact of Ni can be perceived in the intact leaves or in isolated condition (Boisvert *et al*., 2007) [12]. The damage caused by Ni is listed here under: 1. Vandalizing main photosynthetic reaction sites like mesophyll cells and epidermal cells impeding the chlorophyll structure and reduction in the total chlorophyll (Ahmad *et al*., 2007) [1] 2. Tapering the size of grana and at the same time the number of non-appressed lamellae builds up (Molas, 2002) [49] 3. Disrupts the normal electron flow and activities of the C3 cycle enzymes (Seregin and Kozhevnikova, 2006)[73] 4. Ni induced water stress due to oxidative stress (Seregin and Kozhevnikova, 2006) [73] 5. CO_2 deficit caused by stomatal closure (Seregin and Ivanov, 2001) [72]. In cabbage (*Brassica oleracea* L.), addition of Nickel sulphate ($NiSO_4$) at 10-20 g cm^{-3} slashed down the chloroplast number and the number of dis organised chloroplast increased. Concurrently, the number of deformed grana, thylakoids, genesis of plastoglobuli and the plasma membrane lipid composition also altered in cabbage (Sreekanth *et al*., 2013) [79]. Piccini and Malavolta (1992) [58] and Ewais (1997) [24] also conceptualise that due to substitution of Fe and Mg by Ni from the chlorophyll ultrastructure, the normal production of chlorophyll hampers. As the Ni concentrates in the PS-II containing lamellar region, it obstruct the normal electron flow from pheophytion via plastoquinone QA and Fe to plastoquinone QB while it modifies the structure of the electron carrier for example plastoquinone QB or proteins presents in the reaction sites (Krupa *et al*., 1993) [39]. Veeranjaneyulu and Das (1982) [84] also emphasized the fact that reduced amount of cytochromes b6f and b559, along with ferredoxin and plastocyanin decelerate the electron transport cycle. Besides, the dual nature of Ni makes its harmful impact more unpredictable where *in vivo*, it slows down the PS-I, but *in vitro* situation, it targets mainly PS-II (Singh *et al*., 1989) [78]. Supplementation of Ni by 1mM boost up the activity of two proteins (16 and 24 kD polypeptide) analogous to the oxygen evolving complex of PS-II in the spinach leaves (Rao and Sresty, 2000) [63]. Secondarily, it curbs the enzymatic activity in the Calvin cycle like Rubisco, 3-phosphoglycerate kinase, fructose-1,6-bisphosphatase, aldolase, and NAD- and NADP-dependent phosphoglyceraldehyde dehydrogenases there by deposition of ATP and NADH successively increase the pH along the thylakoid membrane and forestall PS-II activity (Sheoran *et al*., 1990) [75]. So, all of this toxic effect singularly and conjointly undermine the plant metabolism resulting curtailed photosynthetic rate.

4.5 Effect on water regime

Key goal to get healthy plant is to maintain a uniform soil-plant-atmosphere continuum of water. Water is required in every single step from keeping the plants cool, turgid to translocation of nutrients throughout the plant body. Particularly in the arid arears to sustain the photosynthetic process, it is ubiquitous to maintain uniform water content in the plants (Yusuf *et al*., 2011) [88]. Heavy metals choke the water circulation from below to above ground parts at numerous ends such as water translocation through symplast and apoplast, stomatal water balance causing dehydration (Prasad, 1997; Chen *et al*., 2004) [61, 15]. Several experiments disclosed the negative effect of Ni on water uptake and balance (Schickler and Caspi, 1999) [70]. Less than a week old wheat seedlings in the sand with added 10mM of Ni in the nutrient culture showed reduce leaf water potential (MPa), stomatal conductance (mol H_2O m^{-2} s^{-1}), transpiration rate (mmol H_2O m^{-2} s^{-1}) and total moisture content and even the $1/10^{th}$ of the aforementioned Ni concentration can reduce the leaf area of pigeonpea by 40% (Bishnoi *et al*., 1993) [11]. Molas (1998) [48] reported that 5.20 gm^{-3} $NiSO_4$-$7H_2O$ in cabbage edge off leaf area by 40% over control plots. 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. Stomatal number may vary due to depletion of leaf area and expansion of epidermal cells but, stomatal operations like transpiration rate scaled down due to toxic effect of Ni (Seregin and Ivanov, 2001) [72]. The lethal impact of Ni exalted the concentration of Abscisic acid (ABA)-a growth retardant hormone which amplify the stomatal closure event uniquely in non-nodulating legumes like *Phaseolus vulgaris* (Molas, 1998) [48]. The collective aftermath of shortfall in transpiration rate, closing of stomata and lastly buoy up ABA level under excess Ni^{2+} misbalance the idiosyncratic water relation in plants.

5. Conclusions

Nickel 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.

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