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Review Article

NICKEL AND CADMIUM TOXICITY IN PLANTS

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ABSTRACT
Nickel (Ni) and Cadmium (Cd) considered as an essential nutrient where plants cannot complete their life cycle in its absence and cannot be substituted with any other element. Ni was first established as an essential nutrient for the completion of the life cycle and it was reported that Ni deficiency decreases the capacity of plants to develop viable seeds because of hindrance of embryo growth. The uptake of Ni in plants is mainly carried out through the root system via passive diffusion and active transport. The ratio of uptake between active and passive transport varies with the species. It has been reported that plant species and cultivars significantly differ in the uptake of Cd and its subsequent translocation from roots into shoots. Higher Cd accumulation in roots than leaves suggest that Cd transport to the xylem is restricted and Cd is not readily translocated in the phloem. Also, Cd accumulation in root and leaf also depends on binding to the extracellular matrix due to which a significant inhibition of root elongation may be observed. In a study, it was reported that Cd was translocated from lower leaves to upper leaves, and then from upper leaves to culms and ears. Lower leaves accumulated highest Cd content during the early growth stages. However, heavy metals accumulating in the food chain pose risk for health of living being as well as cytotoxicity in plants.
Keywords: Cadmium, heavy metals, nickel, toxicity.

INTRODUCTION

Heavy metals accumulating in the food chain, pose risk for health of living beings, which are less sensitive to metal toxicity than plants. Cytotoxicity of heavy metals in plants has been well documented. Nickel occurs abundantly in igneous rocks as a free metal or as a complex with iron. It stands at twenty second position amongst most abundant elements in the earth crust. The uptake of Ni in plants is mainly carried out through the root system via passive diffusion and active transport. The ratio of uptake between active and passive transport varies with the species, form of Ni and Cd concentration in the soil or nutrient solution.[1] Ni interferes with uptake, transport and utilization of essential nutrients including Ca, Mg, K, P, Cu, Fe, Mn[2]. The uptake of heavy metals from the soil solution is strongly affected by calcium ion. Ca lowered the absorption of Ni. The inhibitory effect of various metal ions on absorption and translocation of Ni from roots to shoots varied as Fe, Co ,Ca, Mg, K, Na. Besides being absorbed by roots, Ni and Cd can also enter into the plants via leaves. When a radioisotope of Ni was applied on the leaves of *Helianthus annus*, 37% of the total amount was translocated to other plant organs. Similar trend was also observed in oat, soybean and tomato leaves.

Toxicity of Nickel and Cadmium in Plants

Plants rely on a range of transition (heavy) metals as essential micronutrients for normal growth and development. These elements are essential for most redox reactions which in turn, are fundamental to cellular functions. However, these transition metals including Ni, Cd, above the permissible limit, interfere with the functions of many cellular components, thereby, altering the normal metabolism, causing cellular injuries, and in extreme cases cause death of plant. The current study is aimed mainly at determining the relationship between Cd and Ni in their effect on growth, photosynthesis and nutrient uptake. Three reasons that effect in generating toxicity by heavy metals including Ni, Cd are firstly displacement of essential components in the bio molecules by the metal, second blocking of essential biological functional group of the molecules and third is the modification of enzyme of plasma membrane. These enzymes and proteins contain several ligands close enough to chelating structures with the metal, and thereby lose their functional property. Heavy metal accumulation in crops is a function of complex interaction among soil, plant and environmental factors. It has been well documented that the contents of heavy metals in crop plants are closely associated with their levels in soil. Moreover, the uptake and accumulation of heavy metal by plants are largely dependent on the available rather than total level of a metal in soil. Plant growth and development are essential processes of life and propagation of species. They are continuous and mainly depend on external resources present in soil. Growth is primarily expressed as a function of genotype and environment, which consists of external and internal growth factors. Presence of excess Ni and Cd in the external environment leads to changes in the growth pattern and development of plants. Seed germination and growth are the initial events in the life of a plant, projecting the extent of future processes. Seed germination is the most resistant phase to heavy metals (Table 1)

Some roots are primary target of metal anions; their growth is more affected than the other parts. In some species which accumulate Ni and Cd in their roots, root growth is inhibited more. Root test is widely used for measure the toxicity of many toxicants including heavy metals .Unlike root growth, lateral root initiation is very resistant to heavy metals, due to the internal barrier and the structure of central cylinder. The number of lateral roots decreased considerably in rice and maize and apparently Ni and Cd could cross the endodermal barrier and got accumulated in the cells of pericycle. Treatment of wheat seed with 100 and 200 mM Ni reduced root growth by 35 and 55%, respectively, over the control. Exposure of the wheat seedling to excess Ni caused a rapid accumulation of this metal in roots. The Cd contents depend on translocation from both upper and lower leaf blades and sheaths, and on accumulation in the culm.Cd accumulation in wheat grain is the product of translocation from leaves and stems into maturing grain. Root length was determined as the distance between the root-shoot junction and tip of the main root. After Cd exposure, plants were harvested. In the apical stem region, the procambium, leaf primordia, and the ground parenchyma, whereas in Cd exposed poplars the signal was low and mainly detected in sub apical leaf primordial. Each plant was separated into stem, leaves, coarse and fine roots. Much research had been conducted on the interaction of Cd and Zn in uptake and accumulation in plant because of their normally accompanying pollution in soil.[3] Some reports concluded that Cd addition decreased the Zn concentration in corn and barley. However, there were also contrasting reports on the relationship between Cd and Zn. For instance, reported that Ni and Cd concentration in plants was synergistic.[4] It has been reported that plant species and cultivars significantly differ in the uptake of Cd and its subsequent translocation from roots into shoots. Higher Cd accumulation in roots than leaves suggest that Cd transport to the xylem is restricted, therefore less in above ground parts, that Cd is not readily translocated in the phloem, Cd accumulation in the root and leaf also depends on binding to the extracellular matrix., a significant inhibition of root elongation was observed.[4] Plant roots are the first entry point for Cd uptake from soil solutions, and the transport processes of Cd into the roots have been well reviewed from the viewpoints of physiological and genetic studies. The amounts of Cd were significantly higher in the high-Cd cultivars than in the low Cd cultivars up to 36 h. After10 h, the amounts of Cd reached plateaus for all cultivars, but slight decreases were found in the high-Cd cultivars. Approximately 90% of the Cd absorbed by the rice cultivars accumulated in their roots, whereas only 60-70% of the Cd in the rice cultivars was distributed in their roots.

Effect on growth of stem

It is noticed from available data that heavy metals particularly Ni affects plant growth at cellular, organ and the organism level. Stem growth inhibited by Ni and Cd results from general disorders and immediate inhibition of cell division. However, it is not clear whether Ni enters cell nuclei at high concentrations and if it does, how important is immediate interference of Ni with DNA and nuclear proteins.[5] By discussing these issues, we will be able to give some insight of Ni toxicity and its effects on plant growth and morphogenesis. Leaf growth, leaf area and total leaf number decisively determines the yield of crops. Exposure to 0.085-0.255 mM (5-15 ppm) Ni and Cd, for a week ,developed chlorosis and necrosis along the veins in newly developed leaves of water spinach. Ni at a concentration of 0.5 mM produced dark-brown necrotic spots along the leaf margins also observed a significant decrease in leaf area even at lower doses (50 and 0.1 mM) of Ni and Cd. Possibly delay in seed germination is caused by the inhibitory action of Ni ions on the processes governing its metabolism and elongation of the resulting embryonic axis, as Ni and Cd is known to inhibit cell division and their elongation.[6] Similarly, deformation of leaves is apparently an expression of the irregular cell elongation. It may be suggested that leaf growth traits might serve as suitable bioindicators of heavy metal pollution and in the selection of resistant plant species. Toxicity symptoms in the form of Ni-induced leaf necrosis were also pronounced in the wild type and empty-vector control lines but not in the transgenic lines. The primary functional mechanism of heavy metal toxicity is the displacement of essential ions e.g. Ni displaces Mg ion. The excess quantity of Ni declined the level of nitrogen in the leaves and roots of mungbean. However, the toxicity was more where Ni was supplemented in combination with other heavy metals namely Cd, Co, Pb. The toxic effect of Ni and Cd on plant growth decreased the area of leaf blades, the major transpiring surface.[6] Nickel hyper accumulation in *T. goesingense* is primarily determined by its high degree of Ni and Cd tolerance achieved through an efficient system to pump and store Ni in the central vacuole of shoot cells (Phytoextraction of Cd from polluted soil using hyper accumulator plants with high Cd tolerance (e.g. *Thlaspi caerulescens*) is one of the major engineering-based methods for environmental restoration [5] reported that in 4 day old plants of *Triticum aestivum* in sand culture, with 10 mM Ni added to the nutrient solution, leaf water potential, stomatal conductance, transpiration rate. *Arabidopsis lyrata,* over expression was found to co- segregates with Cd tolerance[5]. The biochemical and molecular basis of Ni/Cd tolerance in *Thlaspi* and other hyper accumulator.[7] However, the molecular signaling pathways that control these mechanisms are not understood. Salicylic acid (SA) is a potent signaling molecule in plants and is well established to be involved in eliciting specific responses to biotic stresses.

Effect on mineral nutrient on plasma membrane

Ni along with other nutrients such as K, Na, Ca, Mg, Fe, Cu, Zn and Mn is necessary for plant growth[8]and therefore it has been placed in the list of micronutrients. However, the interaction between the toxic heavy metals and other essential plant nutrients determining their availability and uptake needs. Ni has some similar characteristics to Ca, Mg, Mn, Fe, Cu and Zn. Therefore, Ni may compete with these minerals in absorption, uptake and their utilization in the plant system.[9] Varietal differences in Cd accumulation were reported in several crops, including wheat the largest negative translocation from vegetation stage to pre-heading, compared with the other plant parts. The concentration has not changed or has slightly increased. The presence of Ni in *Phaseolus vulgaris* leaf tissues was shown to elevate the level of ABA, which is known to bring about stomatal closure, this may lead to perturbation in physiological and biochemical processes, and ultimately results in toxic effects.[10] For example, Ni in addition to other toxic heavy metals (Cd, Cr, Co, Zn and Pb) is reported to cause Fe deficiency either by retarding its uptake or by causing its immobilization in roots.

The excess quantity of Ni and Cd declined the level of nitrogen in the leaves and roots of chickpea and mungbean. However, the toxicity was more severe in cases where Ni and Cd was supplemented in combination with other heavy metals namely Cd, Co Cu. Lower leaves had the highest Cd contents during the early growth stages, but by the fully-ripe stage contained almost no Cd. The phosphorus content in *Helianthus annus* and *Hyptis suaveolens* also exhibited a significant decrease in response to Ni treatment; it was attributed to an increase in the activity of acid phosphatase and ATPase. Since excess Ni and Cd has been shown to decrease the contents of Fe, it can therefore be speculated that Ni might have reduced the biosynthesis of these metalloenzymes by causing deficiencies of these essential metals[10] as heavy metals are recognized to alter the membrane structure and its functions. In soybean seedlings, Ni (200 mM) induced the accumulation of all free amino acids in roots in association with a decrease in alanine aminotransferase and aspartate aminotransferase, reflecting the accumulation of both alanine and aspartic acid. Accumulation of proline in response to Ni treatment has been found in cabbage soybean, wheat and in rice plants. The Ni and Cd treatment decreased the level of proteins and carbohydrates in sunflower and, maize and soya bean .In legumes, Ni is reported to affect the symbiotic association with bacteria Rhizobium [6] which consequently decreased the rate of nitrogen fixation and hence nitrogen content in plants. Plasma membrane is first functional part of the plant cell that comes in contact with toxic transition metals where these ions alter the membrane fluidity and structural conformation of membrane bound enzymes (e.g. ATPase) and their activity[7]. Ni has also been reported to decline the activity of membrane bound ATPase) thus affecting the solute mobility across the membrane. These modifications are believed to be the consequence of conformational changes in ATPase, brought about by the metal directly or thoroughly by the changes in the lipid composition, associated with the membrane. Changes in the plasma membrane structure resulting from alterations in lipid composition have been observed in rice plants, exposed to Cd and Ni.Cd does not contribute to the greater photo oxidation yield, as is clear from the independence of the rate constant on Cd concentration. In this regard, the function of Cd in photo assembly differs completely from the role of bicarbonate which accelerates by direct thermo-dynamic stabilization of the photo oxidized high-affinity Ni. In addition to plasma membrane, the heavy metals also adversely affect thylakoid membrane, both structurally and functionally.[6] In turn, Ni did not induce immediate effects on membrane permeability but the K+ loss continued with time. The roots of plants grown in Ni-supplemented medium for more than two days, while no differences with respect to controls were detected in plants treated with Cd. Thus the membrane dysfunction induced by metals, including Ni, could be due to changes in the level of membrane lipids and/or membrane lipid per oxidation. One of the membrane bound enzymes which is altered in metal stressed plants seems to be H-ATPase. This is the only proton pump operating in plasma membrane, playing a crucial role in the regulation of ion homeostasis. It was observed that the hydrolytic activity of H-ATPase in roots of different plants was inhibited by Cd as well as by Cu. Treatment of cucumber seedlings with heavy metals (Cd, Cu and Ni) changed the hydrolytic and transporting activities of plasma membrane bound H-ATPase. Higher concentrations of Cd, Cu and Ni (100 mM) in nutrient solution caused a distinct inhibition of H-ATPase activity. Cd rapidly and continuously altered expression of numerous metal transport genes, indicating that it disturbs metal homeostasis. The greater inhibition (about 60%) of ATP hydrolysis was observed in plasma membrane isolated from cucumber roots, treated with Cd, while in the case of Cu and Ni it was 45 and 20%, respectively.[3] Inhibition of the plasma membrane proton pump in root cells under heavy metal stress could also result from the alteration at the transcriptional as well as the translational levels of the cells. Cd measured in vitro is not longer present in plants treated with the metal for 5-10 days. Thus, under our experimental conditions, a detoxifying mechanism seems to be induced in rice root cells soon after Cd uptake, reverting the immediate negative effects caused by this metal. In contrast, Ni an essential nutrient does not elicit immediate responses, even at higher external concentration, but its continuous influx leads to disturbances in K^+ content and water balance, along with other disorders of cell metabolism. The presently reported occurrence of two temporal phases in the responses to Cd and H_2O_2, with a stress-specific timing, emphasizes on the value of kinetic analyses of stress responses especially when they are to be compared. Indeed, one cannot assume that two stresses of equal duration and toxicity have the same biological effects, in term of gene regulation. This important role in the regulation of the plasma membrane bound H-ATPase has its auto inhibitory domain in the C-terminal region of the enzyme.

Effect on enzyme

The values for nitrate reductase (NR) activity in leaves decreased significantly over the control in the presence of different heavy metals such as Cd and Ni. Ni concentrations up to 100 mM resulted in significant inhibition of NR activity in *Brassica juncea*. Wheat seedling treated with 100 mM Ni showed sharp decline in NR activity.[4] Ni also inhibited the activity of NR in soybean which supplies the organic nitrogen to the plants. The above reports revealed that at high Ni and Cd level (100 mM), a significant decrease in NR activity was observed that was more significant in leaves than roots. Here the Ni might be hindering the mechanism of nitrogen uptake by plant roots. Both the uptake and transport of nitrates into the cells depend on the availability of metabolic energy, utilized for cell membrane polarization. The main role in this process is played by H-ATPase proton pump. An inhibition of NO_3 uptake can result from the action of Ni on H-ATPase pump, symport. Moreover, proteins of the NO_{3-} uptake system contain -SH groups, and due to that they are sensitive to heavy metals including Ni and Cd. It is also suggested that NR inhibition is likely to occur because of reduced supply of NADH. This might result from disorganization of chloroplasts, reduced rate of photosynthesis and respiration. NADH oxidation or reduction in NO_{3-} supply to the site of the enzyme as a consequence of water stress induced by the heavy metal, or direct effect of heavy metals on protein synthesis as they have a strong affinity for functional -SH group of enzyme.[6] Nitrogen-fixing organisms of the soil may be free living or form a symbiotic association between the bacteria and the host plant. The invading rhizobia induce the formation of root nodules, where leghaemoglobin ensures a low oxygen environment in which the enzyme dinitrogenase can function.

The product after nitrogen fixation is ammonium, which is rapidly incorporated to form amino acids through the activity of enzyme complex GOGAT, before being exported out of the nodule. P-II proteins sense cellular carbon/nitrogen balance and regulate GOGAT activity. Plants that do not form nitrogen-fixing associations with bacteria generally take up nitrogen in the form of nitrate from the soil. Nitrate must first be reduced to ammonia in the plant before it can be incorporated into organic molecules.[10] It is reported that treatment of wheat seedlings with 100mM Ni led to a decrease in NR activity, without altering its the activation state. Decline in Ni activity was more pronounced than that of NR after the application of 100mM Ni. Moreover, the activities of GS and NADH-GOGAT also showed substantial decrease in response to Ni stress with the latter being more susceptible to the metal. In another study, exposure of wheat seedlings to Ni led to the alteration in nitrogen metabolism in the shoots resulting in the accumulation of ammonium and proline in this organ. The membrane bound enzymes which is altered in metal stressed plants seems to be H-ATPase. This is the only proton pump operating in plasma membrane, playing a crucial role in the regulation of ion homeostasis. It was observed that the hydrolytic activity of H-ATPase in roots of different plants was inhibited by Cd. Higher concentrations of Cd, Cu and Ni (100 mM) in nutrient solution caused a distinct inhibition of H-ATPase activity. The greater inhibition (about 60%) of ATP hydrolysis was observed in plasma membrane isolated from cucumber roots, treated with Cd, while in the case of Cu and Ni it was 45 and 20%, respectively. Inhibition of the plasma membrane proton pump in root cells under heavy metal stress could also result from the alteration at the transcriptional as well as the translational levels of the cells.[9] In rice, it was reported that Cd grain concentration in rice grain was largely governed by the transport of Cd from shoot to grain. Additionally, the high accumulation of Cd in durum wheat grain at maturity reflects redistribution of Cd via the phloem pathway .Therefore, the interaction between qcd4-1 and qcd4-2 may affect phloem-mediated translocation from leaves to brown rice after heading.NILqcd4-2 showed higher Cd accumulation and translocation into ears after heading than "Nippon bare", but had same Cd concentration in brown rice. Decline in NiR activity was more pronounced than that of NR after the application of 100mM Ni. Moreover, the activities of GS and NADH-GOGAT also showed substantial decrease in response to Ni stress with the latter being more susceptible to the metal. In another study, exposure of wheat seedlings to Ni led to the alteration in nitrogen metabolism in the shoots resulting in the accumulation of ammonium and proline in these organs. Induction of NADH-GOGAT and NADH-GDH activities and glutamate producing amino transferase activities might compensate for the decreased Fd-GOGAT activity, serving as alternative means of glutamate synthesis. Chloroplast, cytoplasm and mitochondria of higher plants are the base for glutathione reductase (GR) where it catalyzes the NADPH-dependent reduction of disulphide bond of oxidized glutathione resulting in the generation of GSH. It is involved in defense against oxidative stress, where it plays an important role within the cell system, which includes participation in the ascorbate-glutathione cycle in the breakdown of H_2O_2 maintenance of the sulfhydryl groups of cysteine in a reduced form, storage of reduced sulphur and a substrate for glutathione-S-transferases. Some transition (heavy) metals at higher concentration could induce transient depletion of GSH and an inhibition of antioxidative enzymes [4], especially GR.GR activity in response to Ni stress is often found to be dose dependent and variable over time and this increased activity helps in maintaining glutathione in the reduced form, prior to its incorporation into phytochelatins, and/or the activation of the ascorbate glutathione cycle operating in order to detoxify the ROS, induced on exposure to Ni. Increase in GR activity in response to Ni has also been reported in a number of plants such as wheat, *Alyssum* species and *Cajanus cajanus*. On contrary, in roots and shoots of *Crotalaria juncea*, GR activity was drastically reduced whereas, in maize plants GR activity was not affected by Ni exposure. Several studies have been carried out with plants to evaluate the effect of Ni on ROS and activity of antioxidant enzymes. ROS such as superoxide anion radical (O_2-) hydrogen peroxide (H_2O_2) and singlet oxygen (O_2) are continuously generated in plant tissues as byproducts of metabolic processes. In the respiratory electron transport chain, electrons depart from their normal route and reach to O_2. ROS are relatively more reactive as compared to O_2 and therefore, they are potentially toxic to the living system. These toxic ROS can cause damage to DNA, bring about the oxidation of proteins and lipids, and degradation of chlorophyll pigments. Ni does not seem to be an effective catalyst of this reaction due to its relatively high oxidation and reduction potential. Moreover, direct catalysis of such a reaction by Ni has not been demonstrated yet, whereas, it has been shown that Ni-dependent reduction of hydrogen peroxide may be increased by certain chelating agents. ROS may also originate from the reactions catalyzed by NADPH oxidases.[12] These enzymes transfer electrons from cytoplasmic NADPH to oxygen, which results in the formation of oxygen. Moreover, pretreatment of wheat roots with NADPH oxidase inhibitors repressed Ni-induced increase in the production rate of oxygen which further confirmed the implication of NADPH oxidase superoxide generation. Participation of Ca in Ni-induced production of oxygen by NADPH oxidase reported that when Ni chelates with peptides containing glycylgycyl-L-histidine sequence it could per oxidize lipids through hydroxyl radical production. The toxicity of ROS explains the evolution of complex arrays of non-enzymatic and enzymatic detoxification mechanisms (antioxidant systems) in plants capable of quenching ROS without itself undergoing conversion to a destructive radical, interrupting the cascades of uncontrolled oxidation.[13] The ROS scavenging mechanisms of plants includes SOD acting against ROS Afterwards, ascorbate peroxidase particularly in apoplast[9] and by CAT in peroxisomes. Different forms are specifically targeted to chloroplasts, mitochondria, peroxisomes, as well as to the cytosol and apoplast. The induction of antioxidant system by heavy metals, including Ni is well established. Exposure of maize and pigeon pea to Ni provoked positive response to antioxidant systems. The exposure to metal initially resulted in a severe depletion of glutathione.[12] However, Ni suppressed the activity of CAT in sunflower and *Hyptis*. Moreover, the activity of the CAT enzyme in sunflower decreased by more than 5 times.[14] Depression in CAT activity and an increase in POX activity in mustard leaves and in sunflower[9]have also been reported. However, in *Crotalaria juncea*, the activity of CAT did not exhibit a clear cut response to Ni exposure, whereas, GR activity decreased significantly, both in root and shoot. Majority of studies demonstrating the response of GR to metal exposure have shown that its activity increased [15] as a

part of defense system against the metal induced stress, a change that is found to be dependent on the dose of the metal and time of application [10] also reported an inhibition in the activity of CAT in wheat shoots, subjected to Ni (200mM) stress. Cd to the soil increased the inhibitory effects on biological parameters. The addition of lower Cd could stimulate soil microbial biomass and its metabolic activity [16]. The first time addition of Cd to soil can obviously inhibit soil microbial biomass and its metabolic activity, then favoring a selection of Cd resistant microorganisms with prolongation of addition time. The respective decrease in the activity of these two enzymes, compared to the control, was of the order 31 and 24%. A decrease in the activity of these enzymes in *Alyssus bertolonii* and *Nicotiana tabacum*, subjected to Ni stress was also reported.[10]

CONCLUSION

This review article provides effect of essentiality of Ni and Cd in growth and development of the plants. Ni and Cd in proper quantities has major roles in physiological processes, like seed germination, plasma membrane permeability, plant productivity etc. Plants cannot complete their life cycle without sufficient supply of this metal. Therefore, Ni has place in major list of essential micronutrients. Besides this, Ni and Cd at excess level it affect the metabolic activities of the plant like water relation and mineral nutrition causes enzyme inhibition, stomatal functioning, photosynthetic electron transport and degrades chlorophyll molecules, minimizes the photosynthetic rate, and biological yield of plants. Excess Ni and Cd concentration cause oxidative damage in the plants which relates the diverse toxic effects of the metal. Therefore, damage many cellular organelles and DNA, oxidize proteins and lipids and also degrade chlorophyll pigments. Plants have well organized defense system to tolerate the toxic effects that includes restriction of entry of the metal into the cell through plasma membrane and chelation of the metal by phytochelatins, metallothiones and nicotianamide, making it less toxic for the plants.

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Table 1 Effect of nickel on plant

Nickel concentration	Plant	Site of action	References
200 mM	Barley	Nuclei attained irregular shape	Liu et al.[17]
400 mM	Onion	Root biomass severely affected	Brune and Dietz[18]

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