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Unraveled Role of Nickel in Plants

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Nickel is the seventeenth element recognized as essential for plant growth and development. Nickel is unique among plant nutrients because its functions in plant growth and development. Nickel is a key component of selected enzymes involved in N metabolism and biological N fixation. Though the importance of Nickel was discusses at less range, the vital role played by Ni in plant growth and development was enormous. Its uptake mechanism, deficiency, symptoms and role in physiological process of plants makes its essential under the criteria of eligibility.

INTRODUCTION

Nickel is considered to be an essential micronutrient in plants because of its role in the metalloenzyme urease. Nickel is a key component of enzymes involved in N metabolism and biological N fixation. The important constituent and essential part of enzyme – UREASE is nickel. Plant level N fixing micro organisms requires Nickel for effective symbiotic nitrogen fixation

Urea -------urease (Nickel) ----------> $CO_2 + NH_4$

UPTAKE IN PLANTS

Ni has been identified as a component of a number of enzymes, including glyoxalases (family), peptide deformylases, methyl-CoM reductase and ureases, and a few superoxide dismutases and hydrogenases. Therefore, Ni plays a role in various important metabolic processes, including ureolysis, hydrogen metabolism, methane biogenesis and acitogenesis. Ni may also have other functions that have yet to be discovered in plants. Since Ni is essential for plant metabolism, its uptake and transport in plants is involved in some important physiological processes.

The uptake of Ni in plants is carried out mainly by root systems via passive diffusion and active transport. The ratio of uptake between active and passive transport varies with species, Ni form and concentration in the soil or nutrient solution. Soluble Ni compounds can be absorbed via the cation transport system. Since Cu^{2+} and Zn^{2+} inhibit Ni^{2+} uptake competitively, these three soluble metal ions seem to be absorbed by the same transport system. The uptake of Ni by plants depends on Ni^{2+} concentrations, plant metabolism, the acidity of soil or solution, the presence of other metals and organic matter composition.

TRANSPORT AND DISTRIBUTION OF NI IN PLANTS

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. This transport is tightly regulated

by metalligand complexes and proteins that specifically bind Ni. Metal ligands, such as nicotianamine (NA), histidine (His) and organic acids (citric acid and malate ions), can act as intracellular chelators, which bind Ni in the cytosol or in subcellular compartments for transport, translocation and accumulation within plants.

Organic acids, such as citric and malic acids, provide both a source of protons for solubilization and anions for Ni chelation. Three distinct Ni^{2+} metallochaperones, including HypB, CooJ and UreE proteins, have been identified in bacteria. It is likely that similar Ni binding proteins will be found in plants.

DEFICIENCY

Severe Ni deficiency in woody perennials can trigger growth disorders i.e., “mouse-ear” or “little-leaf” and an orchard replant disorder. Deficiency also disrupts nitrogen (N) and carbon (C) metabolism in expanding foliage; although, the influence of Ni deficiency on N-forms translocated in spring xylem sap to canopysinks is unknown. Such relationships are potentially important to management decisions pertaining to pollution, cost, and productivity of agricultural ecosystems Ni deficiency also influenced urea cycle intermediates in that the concentration of asparagine declined and citrulline increased Ni deficiency also reduced the xylem sap concentration of phenylethylamine.

Ni deficiency quantitatively altered xylem sap reduced-N composition, but had no qualitative influence; thus, being similar to that observed in active growing Ni deficient pecan foliage.The decline of xanthine, and rise in allantoic acid, in sap of Ni deficient trees is evidence that Ni directly or indirectly affects ureide catabolism at one or more metabolic points, as was similarly found for ureide catabolizes in actively growing spring foliage. This raises the possibility that Ni ions can potentially influence the activity of certain ureide catabolic enzymes. Nickel deficiency also appears to disrupt the urea cycle. This is based on the observed increase in citrulline concentration and reducing asparagine concentration of asparagine in spring xylem sap .This metabolic shift indicates that Ni deficiency likely reduces either the presence or activity of argininosuccinate syntheses, which catalyzes citrulline to argininosuccinate.

MANAGEMENT

Nickel deficiency appears as leaflet-tip necrosis, or "mouse-3 mear" leaves. The easiest and most effective strategy to correct acute Ni deficiency is foliar spraying with a dilute solution of $NiSO_4$ or other water-soluble Ni fertilizer. Municipal biosolids can be effectively used as a Ni fertilizer.

TOXIC EFFECTS OF NI ON PLANTS

Although Ni is an essential metal and plays important roles in plant metabolism, Ni toxicity has become a particular concern,due to its increased industrial use. Under Ni stress conditions, many common Ni-detoxification responses appear in plants. These responses include the formation of Ni^{2+}-organic acid and Ni^{2+}–NA complexes, the overproduction of NA and its synthase, and high levels of free histidine. Other responses include the induction of MTs and thiol glutathione, and high concentrations of glutathione, Cys and O-acetyl-L-serine (OAS). In addition, some enzyme activities may be enhanced, such as serine acetyltransferase (SAT) and glutathione reductase. However, under excess Ni conditions, toxicity symptoms in plants will develop.

DISRUPTION OF PHOTOSYNTHESIS

The influence of Ni on photosynthesis is pervasive, occurring both in isolated chloroplasts and whole plants. Ni damages the photosynthetic apparatus at almost every level of its organization, including destroying cells of mesophyll and epidermal tissue and decreasing chlorophyll content (chlorophyll a, b, total chlorophyll and chlorophyll a/b ratio). Nickel also damages the thylakoid membrane and chloroplast grana structure, reducing the size of grana and increasing the number of non-appressed lamellae. At the biochemical level, Ni affects light-harvesting complex II (LHCII) and the amounts of xanthophylls and carotenoids. It also interferes with the photosynthetic electron transport chain and its intermediates (such as cytochromes b6f and b559) in leaves.

The inhibition of electron transport is mainly on the donor side of photosystem II (PSII) and the binding site for QB, the secondary quinine acceptor of PSII. Further studies on photosynthetic protein complexes have suggested that Ni mainly inactivates photosystem I (PSI) in vivo, whereas it primarily targets PSII in vitro. Ni in stems and leaves are mainly located in the vacuoles, cell walls and epidermal trichomes associated with chelators, such as nicotianamine(NA), histidine (His), citrate, organic acids and proteins with various important functions, including permeases, metallothionein (MT), metallochaperones and YS1-like proteins (YSLs).

CONCLUSION

Nickel is the most recently discovered micronutrient, it is required in small amounts by plants. Leguminous crops like bean and cowpea require more Ni than other crops because Ni plays an important role in nodulation and N fixation. If Ni deficiency occurs, it will likely be associated with soils having pH > 6.7 or soils that have received excessive applications of Zn, Cu, Mn, Fe, Ca, or Mg. Nickel is a challenging plant nutrient with which to work because it readily oxidizes to unavailable forms in the soil.