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Short Communication

Nickel as an essential element and a toxicant

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ABSTRACT

Heavy metal contamination due to natural and anthropogenic sources is a global environmental concern. Among heavy metals, nickel is a potential pollutant that readily accumulates in soils, plants, animals, and aquatic systems. The nickel (Ni) concentration in soil ranges and drinking water ranges from 10 – 1000ppm and 6μg/l respectively. The concentration of nickel in natural vegetation ranges from 0.05 to 5mg/kg of dry weight. The amount of nickel entering the human respiratory tract ranges from 0.1 -0.7μg/day. 100-200 μg of nickel is consumed per day from nickel containing cooking vessels. Daily intake of nickel from food is about 170μg respectively. The level of nickel in seawater and river contains approximately 0.5 - 2ppb and 0.3ppb respectively. The main stream smoke of one cigarette contains about 0.04 - 0.58 μg of nickel. Nickel is found in both human and cow's milk at a concentration from 0.001 to 0.1 mg/l respectively. The sources of nickel includes weathering of rocks and soils, forest fires, fertilizers, industrial wastes, sewage and sludge which contains higher load of nickel. Although nickel is an essential element required for the healthy growth of plants, animals and soil microbes, its excess affects the photosynthetic functions of higher plants, causes acute and chronic diseases in humans and reduces the soil fertility. This article summarizes the properties, importance and toxicity of nickel.

Key words: Nickel, properties, biological importance, sources, toxicity

INTRODUCTION

Physical and chemical properties of nickel

Nickel is one of the essential elements found in abundance in the earth's crust occurring at an average concentration of about 75μg/g. It is a metallic element belonging to group VIII b of the periodic table. It is hard, silvery-white, lustrous and found in nature as a component of silicate, sulfide or arsenide ores. It's atomic number is 28, atomic weight is 58.71, specific gravity is 8.9 with the melting and boiling points of about 1.455°C and 2.732°C respectively. It has high electrical and thermal conductivity and is resistant to corrosion at ambient environmental temperatures between -20°C and +30°C and is therefore often electroplated as a protective coating (Chau and Cordeiro, 1995). Although it has oxidation state of -1, 0, +1, +2, +3 and +4, it exists principally in the divalent state (Ni^{2+}) and it is the stable form in the environment. In biological systems, nickel in its ionic state forms stable complex components with various ligands and bind to organic material (Fordsmand, 1997).

Biological importance of nickel

Nickel is considered to be an essential element in animals, microorganisms, plants and has been a constituent of enzymes and proteins. In acetogenic bacteria the reduction of carbon monoxide to acetate is dependent on nickel which is needed for activation and synthesis of carbon monoxide dehydrogenase (Drake, 1982). Nickel stimulates the growth of *Acetobacterium woodii* on the medium supplemented with fructose. Nickel is the core metal in the tetrapyrrole ring of methanogenic bacteria which is essential for the growth of these microbes. Nickel is essential for the active synthesis of urease in plant cells. In several species of higher plants such as jack beans (*Canavalia* sp.), soybeans (*Glycine max*), rice (*Oryza sativa*), and tobacco (*Nicotiana tabacum*), it is required for effective urea metabolism and urease synthesis (Kasprzak, 1987). Some terrestrial plants, such as *Alyssum* species accumulate nickel and require it for growth (Baker *et al*., 2000). Welch (1995) reported that nickel is required in low concentrations by legumes dependent on symbiotically fixed nitrogen either for the growth of rhizobia or for the utilization of fixed nitrogen or both.

Nickel is required for iron absorption, seed germination and its deficiency leads to the production of viable seeds in plants. Application of nickel to crops protects them from certain yield limiting diseases, thus potentially reducing pesticide usage and improving crop yield. Nickel also acts as a bio control agent for microbial pests. It is a key factor affecting the production of secondary plant metabolites and thus influencing plant resistance to disease (Wood and Reilly, 2007). High concentrations of nickel inhibited the formation of IAA, tryptophan and simultaneously promoted the formulation of phenolic and terpenoid inhibitors (Tikhomirov *et al.,* 1987). Khan and Moheman (2006) reported that nickel interacts with iron found in the hemoglobin and helps in oxygen transport, stimulates the metabolism and regarded as a key metal in several plants and animal enzyme systems. Nickel is involved in the transmission of genetic code (DNA, RNA) and it is also present in certain enzyme systems that metabolize sugars.

Nickel can substitute for calcium in excitation process and in the binding with membrane ligands such as the phosphate groups of phospholipids in the process of nerve

transmission, muscle excitation and contraction (Howard, 2003).

Nickel exists in human and rabbit serum as three forms namely, nickel bound to ultrafilterable ligands, albumin bound nickel and macroglobulin bound nickel. Albumin is the main transport protein for nickel in human, rat and bovine sera. A metalloprotein termed nickeloplasmin has been isolated from the sera of rabbits (α - 2 macroglobulin) and human (α-glycoprotein) (USPHS, 1993).

Ultrafilterable nickel binding ligands play an important role in extracellular transport and in the elimination of nickel in urine. L - histidine was identified as a low molecular mass nickel binding constituent of human serum which has greater affinity for nickel than serum albumin. It has been found that L - histidine nickel complex has smaller molecular size than the albumin nickel complex which mediates the transport through a biological membrane by virtue of the equilibrium between these two molecular species of nickel. The exchange and transfer of nickel between L - histidine and albumin appears to be mediated by a ternary complex in the form of albumin nickel L - histidine (Sigel and Sigel, 1988).

Nickel is an essential nutrient in 17 animal species, including chicken, cow, goat, pig, rat and sheep (IPCS, 1991). At very low levels, nickel is also an essential element to humans (Wintz *et al.*, 2002). Schnegg and Kirchgessner (1980) reported that nickel deficiency in rats led to reduced iron content in organs, reduced haemoglobin and hematocrit values and anaemia. King *et al.* (1985) suggested that nickel might serve as a cofactor for the activation of calcineurin, a calmodulin dependent phosphoprotein phosphatase.

Nickel has an essential function in the action or formation of cGMP, a signaling agent that regulates various physiological processes such as blood pressure control, sperm physiology, sodium metabolism and cardiovascular health. Nickel is consistently present in RNA and is bound to several biological substances such as proteins (keratin, insulin), amino acids and serum albumins. It also activates enzymes like arginase, trypsin, acetyl coenzyme A, carboxylase and synthetase (Yokoi *et al.*, 2002).

SOURCES AND OCCURRENCE OF NICKEL

Nickel is a naturally occurring element that is present in soil, water, air and biological materials. It is a natural component of earth's crust and is present in igneous rocks (Chauhan *et al*., 2008). Natural sources of nickel include dusts from volcanic emissions and the weathering of rocks and soils (Kasprzak *et al*., 2003). Inorganic fertilizers particularly phosphate fertilizers have variable levels of nickel depending on their resources (Sharma and Agarwal, 2005).

Atmospheric nickel is considered to exist mainly in the form of aerosols with different concentrations of nickel particles depending on the type of sources. A part of atmospheric nickel entering into the environment originates from meteoric dusts, smoke particles from forest fires, volcanic ash, windblown soil dusts and aerosols from oceanic dusts (Ross, 1994).

Although nickel occurs naturally, concentrations found in the environment may also be caused by anthropogenic input such as depositions from the burning of fossil fuels (IARC, 1990), energy supplying power stations (coal burning power plants, petroleum combustion, nuclear power stations and high tension lines) (Verkleji, 1993), chemical industries (pigment manufacturing, plating/metal finishing, cement manufacturing) and metallurgical industries (steel manufacturing, ore refining and alloy manufacturing). The lubricants which are antiwear protectants for vehicles emit nickel from inefficient engines during the transportation (Sharma and Agarwal, 2005). Nickel is also used as a catalyst in oil refining process, in cryogenic containers, in pollution abatement equipments and also as a component of some plumbing materials. When pipes and other materials corrode, nickel can be released to drinking water and may cause damage to human health, but the releases from this source are small. Oats, chocolate, soy beans, nuts and other whole grains are excellent sources of nickel (Salniko *et al*., 2003).

Nickel occurs in aquatic system as soluble salts adsorbed on clay particles or organic matter (detritus, algae, bacteria) or associated with organic particles such as humic acid, fulvic acid and proteins. Nickel may enter surface water as a particulate matter in rain water, through the dissolution of primary bedrock minerals and from anthropogenic sources or from secondary soil phases (Boyel, 1981).

Nickel toxicity and health hazards

Although nickel is omnipresent and is vital for the function of many organisms, concentrations in some areas from both anthropogenic release and naturally varying levels may be toxic to living organisms.

Waste water discharged from electroplating, electronics and metal cleaning industries often contains high concentration of nickel ions and causes various types of acute and chronic disorders (Akhtar *et al.*, 2004).

In humans, nickel is known to cause liver, kidney, spleen, brain and tissue damage, vesicular eczema, lung and nasal cancer on acute exposure (IPCS, 1992). Nickel induces embryotoxic and nephrotoxic effects, allergic reactions and contact dermatitis (EPA, 2002). Nickel sensitization also occurs in general population from exposure to coins, jewellery, watchcases, and clothing. It causes conjunctivitis, eosinophilic pneumonites, asthma and local or system reactions to nickel containing prostheses such as joint replacement, cardiac valve replacements, cardiac pacemaker wires and dental inlays (Hostynek and Maibach, 2002).

Nickel compounds are carcinogenic to humans and they accumulate in the nucleus, especially in the nucleolar fraction

(NAS, 1975). Intracellular binding of nickel to nuclear proteins, RNA and DNA may cause strand breakage, induce chromosomal aberrations, sister chromatid exchange, diminished RNA synthesis, mitotic activity and gene expression in both mammalian and human cultured cells (Zienolddiny *et al*., 2000). Matlock *et al*. (2002) observed the transformation of tumorous cells which involves DNA damage resulting from mutation caused by hydroxy radical or other oxidizing species.

Acute exposure of human lungs to nickel results in pathological pulmonary lesions, hemorrhage, edema, deranged alveolar cells, degeneration of bronchial epithelium and pulmonary fibrosis. When the skin surface is exposed to nickel ions, it diffuses through the epidermis and binds to the carrier proteins to form allergen which causes skin disorders and allergies (Grimsrud *et al*., 2003). Nickel penetration into the skin is also found to be enhanced by sweat, blood, detergents and other body fluids (USEPA, 1980). Nickel compounds have been found to penetrate the mammalian placental barrier and affect the foetus in relevance to the presence of female workers in industry (Sunderman *et al*., 2001).

Nickel is a potent animal teratogen. Inhalation and exposure of nickel carbonyl compounds to rats and hamsters were found to cause fetal death, decreased weight gain and eye malformations (Sevin, 1980). Nickel was also proved to be embryolethal and teratogenic to white leghorn strains of the domestic chicken (*Gallus* sp.), possibly due to the mitosis - inhibiting activity of nickel compounds. Malformations might include poorly developed or missing brain and eyes, everted viscera, short and twisted neck and limbs, hemorrhaging and reduction in body size (Gilani and Marano, 1980). Rodents exposed to nickel during gestation showed a decline in the frequency of implantation of fertilized eggs, enhanced resorption of fertilized eggs and foetus, an increased frequency of stillbirths and growth abnormalities in live - born young (Hausinger, 1993).

Release of nickel effluents into the water bodies was reported to cause stress to aquatic animals and affect their metabolic and physiological activities, biochemical composition and histology of tissues (More *et al*., 2003). Nickel toxicity in plants cause patchy discolourations, premature senescence, yellowing of old leaves, stunting of the roots, deformation of various plant organs, necrosis of leaves, wilting, growth reduction, wrinkled and cupped leaves, shortened internodes resulting in stunted plants and witches broom appearance referred to as “mouse ear” disorder (Fordsmand, 1997). It was also found to affect the photosynthesis, cell divisions and act as a mutagen to plants. Some species of plants accumulate higher doses of nickel above ground tissues. These hyperaccumulators tolerate high levels of nickel due to free histidine in the xylem sap, which also provides a defense mechanism against herbivory (Kramer *et al*., 1996).

In microorganisms, nickel was seen to bind mainly to the phosphate, carboxylic and hydroxycarboxylic groups of the cell walls. From this site, an active transport mechanism designed for magnesium was observed to transport nickel. In microorganisms and higher plants, magnesium is considered as the usual competitor for nickel in the biological ion-exchange reactions (Kasprzak, 1987).

CONCLUSION

Nickel has gained considerable attention as a potent heavy metal pollutant due to the growing anthropogenic pressure on the environment. Even though it is a heavy metal it is essential for plants, animals and human beings to lead a successful life. Nickel toxicity in plants causes stunted growth, necrosis of leaves and mouse ear disorders. In humans it causes lung and nasal disorders, dermatitis and vesicular eczema. It is a potent animal teratogen. Thus with the development of science and technology in the recent past, nickel containing effluents can be treated using biological materials and instead of its disposal it can be recycled for the benefit of mankind in fish farming and agriculture.

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