How Do Turfgrasses Use Nickel?

After all, it's the second least-abundant micronutrient

By Richard J. Hull

Nickel (Ni) is the latest element to be added to the list of nutrients that are essential for plants.

While suspected to be essential since the 1970s, Ni was experimentally demonstrated to be required for legumes in 1983 by Dr. Ross Welch and his associates of the USDA Plant, Soil and Nutrition Laboratory at Cornell University (Eskew et al., 1983). Four years later, the same investigators reported Ni to be generally essential for all plants (Brown et al. 1987). In 1992, the Agricultural Research Service officially added Ni to the list of plant-essential elements.

Although Ni is the last essential nutrient element to be discovered, it is not really the least abundant in plants. Plant leaves require approximately 1 mg Ni/kg dry tissue in parts per million (ppm), a little more than the amount of molybdenum (Mo) required.

To put this in relative terms, for every atom of Mo required by plants, two atoms of Ni and one million atoms of nitrogen are needed (Hull, 2003). Clearly Ni is a micronutrient.

I am aware of no Ni analyses having been conducted for turfgrass leaves but like Mo, they would likely average about 2.5 ppm. In most plants, the Ni concentration is in the range of 1 ppm to 10 ppm of dry tissue (Marschner, 1995). Given those values, the Ni content would be sufficiently high to preclude any possibility of deficiency symptoms in field-grown plants.

The low probability of Ni ever being of concern to the turfgrass manager may explain the virtual absence of published research on Ni in the turf literature. Since Ni is an essential part of an enzyme required for the proper metabolic cycling of nitrogen (N) in all plants, it should spark at least indirect interest among turfgrass professionals.

**Ni: essential component of urease**

The only known function of Ni in higher plants is its role in the enzyme urease. There it is built into a complex protein structure by becoming coordinated between three N and three oxygen (O) atoms. During enzymatic activity, one of the oxygen atoms is displaced by a water molecule which is then inserted into a urea molecule, hydrolyzing it into two ammonium ions ($NH_4^+$) and a carbon dioxide ($CO_2$).

$$H_2N-C-NH_2 + H_2O \rightarrow 2 NH_4^+ + CO_2$$

The primary purpose of the urease enzyme is to recycle the N in urea that is a waste product of N metabolism. Urea is a byproduct of several biochemical pathways through which N is cycled during biosynthetic and degradation processes (Figure 1). These include the ornithine cycle by which the essential amino acid arginine is made and metabolized into the greatest source of metabolic urea.

Metabolism of the ureides, allantoin and allantoic acid, that in many plants are transported from roots to leaves through the xylem, produces urea when their N is assimilated into proteins. Ureides can also be derived from the recycling of nucleic acids when purines are metabolized.

In short, urea is an inevitable product of normal nitrogen metabolism that if allowed to accumulate in plant tissue will cause substantial damage. To prevent such damage and reutilize its N, urea is hydrolyzed by urease and the resulting $NH_4^+$ is reassimilated in the same way as that derived from nitrate metabolism (Hull, 1996).

Since plants lack the elimination system of animals by which urea can be released in urine and its N wasted, plants have evolved a more conservative approach to urea removal and N recycling. To do this, Ni is essential.

Ni deficiency symptoms can be linked directly to the failure of urease to eliminate urea. The initial deficiency symptom is leaf tip burn resulting from urea accumulation in this oldest portion of a grass leaf.

Injury probably occurs as a salt-like effect as urea accumulates to high levels. Root and shoot growth is suppressed, and leaves lose their green

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TABLE 1

Sources of Urea in Plants and $N_4H$ Assimilation

<table>
<thead>
<tr>
<th>Sources</th>
<th>Urea Cycle</th>
</tr>
</thead>
<tbody>
<tr>
<td>PURINES</td>
<td>H$_2$PO$_4$</td>
</tr>
<tr>
<td>XANTHINE (C$_7$H$_6$O$_3$)</td>
<td>CARBAMOYL-PO$_4$ (C$_7$N$_6$O$_3$P$_2$)</td>
</tr>
<tr>
<td>URIC ACID</td>
<td>2 GLUTAMINE (C$_7$N$_4$O$_3$H$_2$)</td>
</tr>
<tr>
<td>ALLANTOIN</td>
<td>2 GLUTAMATE (C$_7$N$_4$O$_3$H$_2$)</td>
</tr>
<tr>
<td>ALLANTOATE (C$_7$N$_4$O$_3$H$_2$)</td>
<td>H$_2$O</td>
</tr>
<tr>
<td>GLYOXALATE (C$_6$O$_3$H$_4$)</td>
<td>Urea</td>
</tr>
</tbody>
</table>

Ni accumulated in seeds

Although absorbed from the soil solution as the divalent nickel ion (Ni$^{2+}$), within plant cells Ni readily combines with organic anions such as citrate and cysteine. It is likely that Ni is transported within plants as its citrate chelate. Unlike some cationic micronutrients (e.g. iron or manganese) that exhibit substantial xylem mobility but are virtually immobile in the phloem, Ni$^{2+}$ is readily transported in both xylem and phloem (Kochian, 1991). As a result, Ni often accumulates in developing seeds to concentrations greater than most other plant organs. This is one reason why Ni deficiency symptoms were not demonstrated until recently. There can be sufficient Ni within the seeds of some annual plants to meet their needs for one or more generations. This contributes to the low probability of observing a Ni deficiency under anything but carefully controlled laboratory conditions.

Toxicity greater concern than deficiency

In many situations, Ni toxicity is a reasonable possibility especially where soils have been supplemented with sewage or industrial sludges contaminated with heavy metals. Plants vary in their sensitivity to Ni with some capable of tolerating tissue concentrations of more than 1,000 ppm while many plants exhibit toxic symptoms when their tissues exceed 10 ppm Ni. Most soils contain less than 100 ppm Ni but only a small amount of that is exchangeable and available to plants (about 1 ppm). Much Ni in soils is present as insoluble mineral fractions and the remainder is bound into organic complexes and is sparingly available to plant roots. Thus, Ni toxicity is not normally a problem for the turf manager.

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color due to interveinal chlorosis and eventually necrosis (tissue death). In younger leaves, the tip region fails to unfold properly. Because seed germination is accompanied by rapid nitrogen metabolism, the seeds from Ni-deprived plants often fail to germinate likely because of urea accumulation. These observations have been made on laboratory-grown plants since no Ni deficiency has ever been reported in the field.

Urease key to N fertilizer

Urease allows urea to be used as N fertilizer. Urea contains two N atoms per molecule (40 percent N), is easily and inexpensively manufactured and is safe to handle, making it a popular source of fertilizer N. However, when applied to soil or plant leaves, urea is not absorbed directly but rather hydrolyzed to NH$_4^+$ and CO$_2$ by the urea hydrolysis. The NH$_4^+$, then absorbed by plant cells and assimilated into amino acids.

It is urease derived from the plant or soil micro-organisms that releases NH$_4^+$ from urea making its N available to the plant. Urease, regardless of its source, makes urea-N available to plants, and it depends on Ni for its hydrolytic function. Thus, urea is an effective N fertilizer due to the action of Ni.

Urea probably can be absorbed by plant cells because Ni-starved plants exhibit greater deficiency symptoms when urea is provided as their N source. Since urease is less active in Ni-deficient plant, any urea absorbed will accumulate within cells and reach toxic concentrations. However, urea is so abundant in the plant-soil environ-
In the coastal mountains of California, serpentine soils can be found that are enriched in several heavy metals including iron, magnesium, chromium (Cr), cobalt and Ni, the latter being present at concentrations as high as 250 ppm exchangeable metal (Mengel and Kirkby, 1982). Such soils are inhospitable to most plants largely because of the magnesium content being greater than calcium (Ca/Mg about .4) but also because of Ni and Cr being present in toxic concentrations. Serpentine soils are not suitable for turfgrass growth but the Ni toxicity can be alleviated by lime applications that neutralize the soil acidity. At higher pH values, both Ni and Cr become less soluble, making them less available for root absorption. Lime also increases the Ca/Mg ratio and may stabilize membrane transport proteins which increase the selectivity of ion uptake by roots. Those plants that can tolerate serpentine soils absorb Ni less rapidly and concentrate it to levels in excess of 200 ppm but as citrate and malate chelates that are sequestered within vacuoles and rendered less toxic. Some hyperaccumulators can achieve tissue Ni concentrations of more than 10,000 ppm again mostly as a chelated form. Other defensive measures likely are also employed by Ni-tolerant plants.

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REFERENCES


