Field Deficiency of Nickel in Trees: Symptoms and Causes

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Abstract

This communication reports that the mouse-ear or little-leaf disorder and the associated replant disease of pecan [Carya illinoinensis (Wangenh.) K. Koch] is a Ni deficiency and is cured by timely foliar application of Ni (at 100 mg L⁻¹); thus documenting the first known example of Ni deficiency in orchard crops. Deficiency is inducible on soils with adequate Ni content by a) excessively high soil Zn, Cu, Mn, Fe, Ca, or Mg; b) root damage by root-knot nematodes; or c) dry and or cool soils at time of bud break. Symptoms associated with Ni deficiency, but varying with severity of deficiency, include early-season leaf chlorosis, dwarfing of foliage, blunting of leaf/leaflet tips, necrosis of leaf or leaflet tips, curled leaf/leaflet margins, dwarfed internodes, distorted bud shape, brittle shoots, cold-injury-like death of over-wintering shoots, diminished root system with dead fibrous roots, failure of foliar lamina to develop, rosetting and loss of apical dominance, dwarfed trees, and tree death. In addition to pecan, Ni deficiency is exhibited by river birch; and based on symptoms and soil characteristics; it also appears to occur in other woody perennial crops (e.g. plum, peach and Pyracantha sp., and citrus). Its occurrence in two prominent ureide transporting hydrophiles, raises the possibility that such species are most likely to experience Ni associated disorders than are other species. Observations implicate excessive soil accumulation of light metals from long-term fertilizer usage as a primary cause of Ni deficiency. These results raise the possibility that Ni is a contributing factor in certain recalcitrant maladies and replant disorders of certain woody perennial crops and support the role of Ni as an essential nutrient element for higher plants. Ni deficiency in field situations appears to be far more common than generally recognized; thus, meriting greater attention in horticultural production strategies and greater awareness by horticulturalists.

INTRODUCTION

Plant Ni nutrition is most often considered within the context of toxicity rather than that of deficiency. For years, Ni stood out among the six metallic elements of the latter half of the first transition series (i.e., Mn, Fe, Co, Ni, Cu and Zn) as not possessing biological activity, except as a toxicant (Thomson, 1982). While Ni toxicity is a problem in metal enriched serpentine soils or when industrial sewage is used on soils, deficiency has received little attention. Nickel (Ni) was probably suspected of being an essential mineral nutrient in plants at least as far back as the early 20th Century when it was discovered that plants contained Ni. Field-level responses to Ni sprays were noted to increase yields of wheat, potatoes, and broad beans as early as 1946 (Roach and Barclay, 1946). With the discovery by Dixon et al., (1975) that urease [EC 3.5.1.5, urea amidohydrolase (Sirko and Brodzik, 2000)], an enzyme found in a very wide range of species, is activated by Ni, the notion of essentiality gained status. Additionally, it was noted that several plant species exhibited growth responses to Ni fertilization (Welch, 1981; Dobrolyubskii and Slavvo, 1957). During the 1980’s Welch and his USDA-ARS associates demonstrated that Ni was essential to legumes (Eskew et al., 1983, 1984). In 1987, the ARS group found that Ni was essential in temperate cereal crops and concluded that Ni is generally essential for all higher plants (Brown et al., 1987a, b and 1990). Ni was subsequently added in 1992, by USDA-ARS, to the list of essential plant nutrient
elements (Hull, 2003), the first since chlorine in 1954 (Eskew et al., 1983). Ni is now generally recognized as an essential mineral nutrient element to higher plants (Gerendas et al., 1999; Bloom, 2002; Marschner, 2002; Epstein and Bloom, 2005), yet field-level crop deficiencies have not been reported. Although, positive crop responses are beginning to be observed for certain crops (Atta-Aly, 1999).

Although Ni is generally regarded as an essential nutrient, at the time of this writing, Ni is not yet recognized as such by the American Association of Plant Food Control Officials (AAPFCO); although, it is under review and is tentatively approved for recognition. AAPFCO is an umbrella organization that functions to influence regulation, labeling, ingredients, and amounts of elements in U. S. fertilizer products (Terry, 2004). Thus, Ni fertilizers, or fertilizers containing Ni as a nutritional additive, are not presently sold or used in the U. S., except for pecan and river birch in the state of Georgia.

The dearth of agricultural interest in Ni is probably attributable to the absence of diagnosed Ni deficiencies in orchard, vine, field or nursery crops. The combination of relatively low presumed Ni sufficiency levels (>0.05-0.2 μg g⁻¹ DW, depending on species (Welch, 1995]) in plants, and the relatively high amount of Ni available in almost all agricultural soils (> 5 kg ha⁻¹), has contributed to the notion that concerns about Ni deficiency are irrelevant to real-world agricultural enterprises. This notion appears to have been largely based on Ni needs of non-woody plants (especially that of agronomic grass and legume crops). While Ni is an essential plant mineral nutrient element, it has traditionally been ignored by U. S. agricultural practitioners as a nutritional supplement and appears to receive only slightly more attention in very few other countries.

The traditional relative insignificance of Ni to production horticulture is now changing with the discovery of field-level Ni deficiency. The initial discovery was that "Mouse-ear or Little-leaf" (ME-LL) of pecan was in fact a Ni deficiency (Wood et al., 2004a, b), rather than a Cu deficiency as was previously suspected and implicated (Wood et al., 2004a, b). The ME-LL disorder (i.e., Ni deficiency) was first reported in 1918 by Marz (1918) and was initially observed in yard-trees located within certain regions of the Gulf Coast Coastal Plain of the southeastern U. S. (Demaree, 1926). The disorder was initially attributed to spring cold injury prior to bud break, but later attributed to disease pathogens (Demaree, 1926). It became increasing common in pecan orchards through the 1930's and is now a malady of rapidly increasing frequency and severity.

Gammon and Sharpe (1956) concluded in the 1950's that the ME-LL problem was a manganese deficiency, based on occasionally reducing certain symptoms using impure sulfate salts of Mn; however, in recent years soil or foliar application of Mn to affected trees failed to exhibit efficacy. It is postulated that this is because of the usage of higher purity Mn sources depleted of trace Ni contamination.

The linkage of Ni deficiency to morphological symptoms, as the result of the pecan discovery by Wood et al. (2004a, b), led to the recognition by Wood that a similar little-leaf disorder on river birch (Betula nigra) is also a Ni deficiency. Subsequent research on this postulate by Reuter (University of Georgia, USA) showed that foliar Ni sprays also corrects and prevents ME-LL of river birch in containerized nurseries and in landscapes.

Based on the symptoms of Ni deficiency exhibited by pecan, river birch, and other woody crops, here morphological symptoms (i.e., acute deficiency symptoms) of Ni deficiency likely to be exhibited by woody perennial crops are reported; circumstances conducive to occurrence of Ni deficiency; woody perennial crops in which Ni deficiency symptoms have been noted; and raise the notion that poor Ni nutrition merits consideration as a contributing factor to replant and recalcitrant disorders of many woody perennial crops.
MATERIALS AND METHODS

Symptoms of Ni Deficiency
Wood et al. (2004a, b) showed the existence of Ni deficiency in pecan and described associated symptoms. In the present paper symptoms noted previously are expanded (Wood et al., 2004a, b) and for the first time provide a listing of symptoms according to degree of apparent Ni deficiency. Morphological and growth associated symptoms displayed by Ni deficient pecan, and also supported by observations on river birch and other woody perennial species, are compiled to provide descriptors to aid field diagnosis of Ni deficiency in woody perennial crops (see Fig. 1 and 2). Symptoms are based on evaluation of hundreds of affected trees exhibiting various stages of acute Ni deficiency in commercial orchards throughout the Gulf Coast Coastal Plain of Georgia and Florida, U.S.A., and in greenhouse seedlings growing in soils producing various degrees of Ni deficiency.

Crops Exhibiting Ni Deficiency Symptoms
The identification of Ni deficiency descriptors, including the key descriptor of ME-LL-type foliage morphology, enabled a means for putative identification of Ni deficiency in field planting of other woody crops. A small-scale survey was conducted in certain orchards of a few tree crops exhibiting growth abnormalities that had defied corrective measures by orchard managers. An examination of affected specimens identified trees and orchards exhibiting several Ni deficiency symptoms identified from the above mentioned study of pecan and river birch, and also included the key indicator symptoms of ME-LL-type blunted foliage. Orchard managers were interviewed regarding contemporary and historical orchard practices and practices noted possessing potential for substantially limiting Ni availability to the crop.

Replant Disorders
Ni deficiency in pecan is most typically, although not always, exhibited by young replant trees in old orchards. Thus, the ME-LL problem has recently been associated with a replant disorder that is increasingly common and severe in pecan orchards. The nature of this replant relationship was investigated in a commercial pecan orchard with the incidence of ME-LL rated on 5-year-old ‘Desirable’ trees in a drip irrigated orchard planted on a site that previously supported 80-year-old ‘Success’ trees. The previous ‘Success’ orchard had been commercially managed for several decades, receiving annual fertilizer applications according to Georgia Extension Service guidelines. These ‘Success’ trees, planted on a 18.3 m x 18.3 m spacing, were removed and replaced by ‘Desirable’ trees (rootstock unknown) at a higher planting density that resulted in transplants being established at the exact position of the pre-existing trees and also midway between pre-existing trees (giving a 18.3 m x 9.15 m orchard spacing). Many of the transplanted ‘Desirable’ trees began to exhibit ME-LL (i.e., Ni deficiency) symptoms the second and third year after planting transplanting, with symptoms becoming increasingly severe as trees aged. All trees were visually rated for ME-LL symptoms (blunted foliage, rosetting, dwarfing, etc.) during the sixth growing season and then mapped in relation to pre-existing trees (see Table 1).

As populations of pathogenic nematodes, fungi, and bacteria typically increase at sites of older trees and in older orchards, soils supporting ME-LL trees (i.e., replant disorder trees) were evaluated under controlled conditions regarding the role of biological agents in induction of the replant disorder. Briefly, potted ‘Desirable’ pecan seedlings were grown in a ME-LL inducing soil. The study had three treatments consisting of pasteurized soil, non-pasteurized soil, and pasteurized soil plus Meliodogyne partityla. Treatments were structured in a RCB possessing nine blocks. Trees were grown in a micro-plot situation with pots being placed within pots and separated by 1 m of course gravel so as to prevent nematode migration. Plots were drip irrigated and scored for
severity ME-LL (i.e., Ni deficiency) symptoms the following spring in 2003 and again in 2004 (see Table 3).

**Circumstances Associated with Induction of Ni Deficiency**

Standard soil characteristics were determined for sites supporting Ni deficient trees adjacent to non-deficient trees. First, differences in soil characteristics were documented between sites in the same second generation 'Desirable' orchard described above that exhibited a clear association between Ni deficiency symptoms and pre-existing trees (i.e., Table 1). It is known that the malady was Ni deficiency because subsequent study (using a variety of high purity Ni sources) corrected symptoms when used as a timely foliar spray. Second, standard soil characteristics were also determined for regional orchards exhibiting severe Ni deficiency. Briefly, soils were analyzed for standard characteristics by the Georgia Extension Service's Plant and Soil Analysis Laboratory or by a private soil testing laboratory. Elements in samples were subsequently classified (i.e., very low = VL, low = L, sufficient = S, high = H, and very high = VH) by the testing labs as to status for pecan (see Table 2).

**RESULTS AND DISCUSSION**

**Symptoms of Ni Deficiency**

Previously published reports describing the symptoms of Ni deficiency (i.e., ME-LL) have noted the mouse-ear-like distortion of foliage and dwarfed leaflets and leaves, but not other morphological symptoms exhibited by severely affected trees (Gallaher and Jones, 1976; Worley, 1979; Grauke et al., 1983). Several distinct symptoms of the severe Ni deficiency are illustrated (Fig. 1, 2, and 3) and are derived from Wood et al. (2004a, b).

**Foliage**

1. **Chlorosis.** Ni deficiency is usually first associated with abnormally pale, or yellowish-green, foliage during shoot, leaf or canopy expansion. This chlorosis tends to be uniform throughout the leaf. Such foliage typically becomes the normal shade of green by mid-season. This symptom appears to be the earliest visual indicator acute deficiency; although, it is not definitive because other nutrient deficiencies (e.g., Fe and S) also produce foliage of a similar type of chlorosis.

2. **Reduced Size and Altered Shape.** The first reasonably clear indicator of acute Ni deficiency is reduced leaf or leaflet size and blunting of apical tips (Fig. 1A and 1B). The degree of blunting varies with apparent degree of Ni deficiency. Leaf apexes exhibit obtuse features. In pecan, this leaf blunting is such that the normally acute apex becomes obtuse, producing a shape similar to the ear of a mouse, thus the disorder is often termed mouse-ear (Fig. 1B). This blunting can be isolated to certain leaves on a shoot (usually the physiologically oldest leaves and leaflets), or to certain branches, or to large limbs, or can be relatively uniform throughout the canopy. In clear cases of severe Ni deficiency, the mouse-ear-like deformation is most prominent at the top of the tree canopy.

The surface area of leaves or leaflets is diminished in proportion to the degree and timing of Ni deficiency. The most typical reduction in area for older trees is in the 10-75% range. This reduction in area led to the term "little-leaf". Thus, in river birch, the disorder is termed "little-leaf". As with foliage shape, the degree of reduction in area tends to increase with canopy height.

3. **Transitory Dark Green Zone at Leaf Tip.** The apical tips of affected spring foliage exhibit a small dark green zone just below the apical tip (Fig. 1C). This color distinction disappears as the leaf ages. The green zone is most noticeable during the first few weeks after bud break. This characteristic appears to possess great diagnostic power as a clear indicator of Ni deficiency as it appears that this characteristic is unique to a Ni deficiency and is not produced by other elements.

4. **Tip Necrosis.** As the severity of Ni deficiency increases, the tips of affected leaves or
leaflets exhibit cell death (Fig. 1C). This dead zone expands with aging and degree of severity. Death is most likely due to a toxic accumulation of urea associated with little or no urease activity in affected cells. Ni deficiency is most pronounced in the most rapidly growing portions of the leaf. This characteristic possesses great diagnostic power for identification of Ni deficiency as it appears to be unique for Ni.

5. Cupping and Wrinkling of Leaves. Cell expansion in the margins of leaves or leaflets, especially toward the apex, is reduced to the point that there is cupping and wrinkling of the lamina (Fig. 1C). Affected foliage also feels thicker and less pliable, tending to be brittle. Foliage during this stage exhibits a Bonsai-like appearance; i.e., miniature. This characteristic also possesses great diagnostic power for Ni deficiency.

6. Absence of Laminar Development. Severely affected foliage can be such that leaf lamina development is completely arrested, displaying a miniature vascular array (Fig. 1D). Thus, in extreme cases, leaf area can be reduced to nothing, which is most likely to occur on young transplant trees about 3-5 years after planting. This state is accompanied by an elongation and sharpening of buds. Both characteristics exhibit great diagnostic power.

7. Winged Petiole. The degree of malformation of the wings on leaf petioles increases with degree of severity. Severe Ni deficiency can produce pronounced protrusion of petiole wings, with each of the two wings becoming as wide as the petiole itself.

Shoot, Root and Tree Characteristics

1. Apical Dominance and Rosetting. Severe Ni deficiency results in a loss of apical dominance, producing rosetted growth (Fig. 2A) similar to that of Zn or Cu deficiency. The resulting shoot growth can vary from a witches broom type of growth to that of a tightly packed shoot with several dwarfed shoots and foliage. This symptom is almost always associated with dwarfed, blunted, necrotic-tipped foliage or foliage with little or no leaf lamina.

2. Buds and Bud Break. Bud shape tends to become increasingly distorted as severity of Ni deficiency increases (Fig. 1D and Fig. 2C). In the case of pecan, buds become elongated and pointed. Bud break tends to be delayed, potentially up to as much as a week or more on Ni deficient shoots (Fig. 2B). However, in the case of young seedlings, Ni deficiency can instead advance bud break. Buds on severely affected trees often die during the dormant season, giving the appearance of winter cold injury.

3. Internodes. Length of shoots is increasingly diminished as severity of Ni deficiency increases. Reduction is due to reduced length of internodes (Fig. 2C). Shoot length can be reduced > 90%, but is most usually in the 10-50% range.

4. Brittleness. The shoots and limbs of trees with moderate to severe Ni deficiency are noticeable brittle. Limbs, branches, and shoots are easily broken when bent by hand or by strong winds.

5. Tree Size. Tree size is increasingly diminished as the severity of Ni deficiency and duration of deficiency increases (Fig. 2D). Young orchard transplants can appear stunted, with little or no canopy or caliper growth. Extreme deficiency produces dwarfed trees, often less than 10% normal size for their unaffected peers.

6. Roots. The root systems of severely Ni deficient trees are reduced, with fibrous roots being dead or decayed. If such trees live, they are very slow to come into even meager fruit production.

7. Death. Severe deficiency produces very weak shoots and trees. Affected shoots often die during the dormant season; thus, giving the impression of death from winter cold. Severely affected trees usually die within a few years of transplanting.

Reproduction

1. Staminate Flowers. Catkin development is retarded, being shortened in excess of 50%. As with foliage, the apical tips of catkins often exhibit necrosis. This necrosis is assumed to be due to urea toxicity associated with the excessive accumulation of metabolic urea.
2. Pistillate Flowers. Development of female flowers can be suppressed to the point that they either fail to become visible or are stunted. A certain small percentage of flowers will develop to ripening. Trees with acute Ni deficiency exhibit reduced crop yield.

Physiological Influence of Ni Deficiency

The above described acute Ni deficiency symptoms appear to be either direct or indirect consequences of the role of Ni in the activity of plant urease, as Ni is the only known activator of urease (Sirko and Brodzik, 2000) and urease is the only known enzyme that recycles N from urea (Polacco and Holland, 1993). Urease allows plants to use external or internally generated urea as a nitrogen source (Mobley and Hausinger, 1989; Mobley et al., 1995), and a substantial amount of plant N flows through urea (Sirko and Brodzik, 2000). Metabolic urea is a natural product of cellular N metabolism; thus, urease is cytosolic and is generally ubiquitous in cells of plant tissues and organs (Hogan et al., 1983); perhaps explaining the manifestation of Ni deficiency by essentially all plant tissues and organs (as described above). Additionally, urea accumulates in rapidly growing cells and tissues; thus, likely explaining Ni deficiency symptoms expressed at the apical tips of leaves, leaflets, and catkins.

While plants utilize many forms of N for growth, it is only N reduced to ammonia that is incorporated directly into organic substances [i.e., largely by aminotransferases, glutamine synthetase, and asparagine synthetase (Bryan, 1976)]. Because plants cannot excrete urea, as do animals, urea waste accumulates if Ni is insufficiently available to activate urease for the hydrolysis of urea to ammonia and carbon dioxide. Thus, the supply of ammonia for synthesis of certain amino acids and various classes of proteins is severely limited. This disruption not only leads to urea toxicity, but also appears to lead to other disruptions in N metabolism [especially that of glutamine synthetase (Gerendas and Sattelmacher, 1997; Gerendas et al., 1998)], apparently producing a multitude of side-effects on plant physiological and growth processes. The symptoms above hint of possible direct or indirect effects on endogenous growth regulators, protein synthesis, lignification, catalysis, electron transfer and gene regulation.

Because Ni deficiency is typically first expressed in early spring, when the plant’s N pool is being processed via various N pathways to satisfy growth needs, there appears to be an associated toxic accumulation of urea in zones of rapid growth, such as leaf and catkin tips, and perhaps in other tissues and organs (Polacco and Holland, 1993). Krogmeier et al. (1989) similarly concluded that in the case of urea fertilization, leaf tip necrosis occurred due to accumulation of toxic amounts of urea rather than ammonia (as a product of urease action). Nicoulaud and Bloom (1998) concluded that in tomato, Ni improves the growth of plants receiving foliar urea. The discovery of field-level Ni deficiency therefore raises the possibility of greater attention to Ni nutrition when urea is used as a foliar fertilizer in fruit crops.

The impressive breath of Ni-related growth abnormalities associated with the above described symptoms exhibited by various plant organs indicates that either urea buildup in cells and tissues a) indirectly triggers a multitude of adverse effects on physiological processes, or b) that Ni is involved either as a structural component or activator of enzymes in addition to urease. To date, Ni has not been found to be an unique activator of any other enzyme in higher plants, although several enzyme systems in microorganisms are activated by Ni [i.e., urease, NiFe-hydrogenase, carbon monoxide dehydrogenase, acetyl-CoA decarboxylase/synthase, methyl coenzyme M reductase, certain superoxide dismutases, some glyoxylases, aci-reductone dioxygenase, and methylenediurease (Mulrooney and Hausinger, 2003)]. It is not obvious that an exhaustive effort has been made to discover all Ni associated enzymes in higher plants, especially tree crops.

The key identifying symptom for recognition of acute Ni deficiency on deciduous woody perennial crops is the “mouse-ear” or “little-leaf” morphological symptom when it is associated with pronounced leaf tip necrosis and dwarfed foliage. As discussed above, this is due to toxic accumulation of urea associated with reduced urease activity.
It is noteworthy that Ni deficiency not only occurs at the acute level (i.e., expression of morphological symptoms), but also likely occurs at the chronic level (i.e., expression of only physiological symptoms, but no morphological symptoms). While the existence of acute Ni deficiency is likely to be relative rare in crops, the existence of chronic deficiency may be far more common. The characterization of chronic symptoms and impact on yields merits further investigation, especially considering that N is typically the most limiting and most commonly applied element in world agriculture and that urea is an increasingly common and widely used N fertilizer. Excellent reviews of the significance of Ni in plant nutrition have been published by Welch (1995), and Gerendas et al. (1999).

Crops Exhibiting Ni Deficiency Symptoms

Anecdotal observations at time of this writing has resulted in identification of Ni deficiency symptoms, as noted for pecan and river birch, for certain other crop species. It probably occurs in many species, but to date has only been noted in pecan, river birch, plum, peach, citrus, and a species of Pyracantha (an ornamental shrub). The search for field-level Ni deficiency has only now just begun. These deficiencies were associated with medium to low cation exchange capacity soils that had been heavily fertilized with Zn, Mn, Fe, Cu, Ca, or Mg. Although, in the case of citrus, foliar blunting was associated with an accidental double application of foliar applied urea at time of shoot expansion in an orchard soil possessing high Ca and Mg. Regardless of species, symptoms were most severe when soils were dry during early canopy expansion.

Species that are the most likely candidates for field-level Ni deficiency appear to be those whose native ecological niche is hydrophilic. Such species are exposed to moist soils rich in organic and mineral nutrients. Such soils possess a thick zone of humus and distinct organic horizon containing a relative plethora of available nutrient elements, plus water availability is generally good. These eutrophic soil environments would not likely exert much selection pressure for adaptation of high efficiency uptake or usage physiologies for micronutrients as for plants growing in oligotrophic soils. Thus, such species are more likely to possess relative high micronutrient requirements. This likelihood is supported in that both pecan and river birch also possess relatively high Zn and Ni requirements (Smith, 1991; Worley, 1994; Wood, 2004a, b).

It is also noteworthy that both pecan and river birch are hydrophilic species (e.g. adapted to a moist environment) and that both transport nitrogen via ureides. Many hydrophilic and tropical legume species transport substantial nitrogen as ureides rather than amides or amino acids. Ureides are structurally related to urea and some representative examples are allantoin, allantolic acid, citrulline, uric acid, hypoxanthine, xanthine, daffaine, hydroxyctrulline, and albizzine. Such compounds play an important role in the assimilation, metabolism, transport, and storage of nitrogen in many hydrophilic species. It has been suggested that urease plays a role in ureide catabolism (Polacco et al., 1985, in Schubert and Boland, 1990) and urease activity is dependent on Ni (Dixon et al., 1975). This background is evidence that species most likely to display Ni deficiencies in field or nursery situations are hydrophiles and/or ureide transporters. Examples of ureide transporting genera are: Acer, Almus, Annona, Betula, Carpinus, Carya, Cercis, Chamaecyparis, Cornus, Corylus, Diospyros, Juglans, Nothofagus, Ostrya, Platanus, Populus, Pterocarya, Salix and Vitis (Schubert and Boland, 1990). These genera represent only a partial list of woody perennial candidates, but include several major crops in which Ni deficiency might be most likely to be found. These include orchard and vineyard crops of pecan, the several walnut species, grape, persimmon, and filberts; plus, a multitude of landscape and ornamental crops. Many tropical legumes are also ureide transporters and are also likely candidates for Ni deficiency. Because the equilibrium between production of ureides and amides is influenced by factors such as shade and soil nitrate or ammonium sources (nitrate favors amides and ammonium favors ureides), the expression of Ni deficiency could also be partially dependent on orchard or nursery management strategies (Schubert and Boland, 1990).
Replant Disorders

Replant disorders in pecan orchards are becoming increasingly common. This disorder typically appears the second or third year after transplanting and often become progressively severe over the next few years. These replants are usually replacing missing trees in relatively old (> 50 years-old) orchards. In many cases, affected trees eventually outgrow the problem and begin to grow and appear normal.

Table 1 illustrates the soil characteristics exhibited at replant locations vs. locations exactly between replant locations when ‘Desirable’ replace pre-existing ‘Success’ trees. About 84% of replanted trees located at pre-existing tree sites (i.e. those where older trees previously stood) exhibited Ni deficiency, whereas only 1% of replants planted between pre-existing tree locations exhibited symptoms. It is therefore clear that pecan replant disorder is tied to unique soil factors associated with the site of the pre-existing tree. Soil characteristics between the two locations indicate that the key difference is that soil of the pre-existing site contains much more Zn and Mn. Both elements can inhibit Ni uptake by roots from soil. Thus, based on the fact that foliar sprays of Ni to these trees corrected deficiency symptoms (Fig. 3), and based on the fact that Ni uptake is inhibited by Zn and Mn, it is apparent that this particular Ni related replant disorder was likely induced by excessive accumulation of soil Zn and/or Mn as a result of being concentrated around the trunk of trees whose canopies received decades of annual foliar sprays of these elements.

There are several economically important diseases, or declines, of unknown or uncertain cause affecting many different tree crops (Margarey, 1999). Many of these have been associated with one or more biotic factors as contributing agents, yet there also appears to be associated abiotic causal factors (Derrick and Timmer, 2000). Replant diseases or declines are among these and have been noted on fruit trees for over 200 years. According to Derrick and Timmer (2000), “...newly planted trees grow very poorly, are stunted, and have small leaves and short internodes. Root systems may be reduced, fibrous roots may be decayed and if trees live, they are slow to come into production.” These exact symptoms were exhibited by pecan trees displaying the Ni associate replant disorder; and in the case of pecan, symptoms were always corrected by timely foliar Ni application (Wood et al., 2004a, b).

Nematodes are often associated with replant diseases, with the presence of certain species enhancing severity in proportion to their population or damage to roots. The role of certain nematodes in Ni deficiency is briefly noted later in this communication. The symptomatology for Ni deficiency, the role of nematodes in enhancing symptom severity, the fact that Ni has received little or no attention regarding these diseases, and evidence that edaphic factors contribute to the disease complex, collectively indicate a need to ascertain the role of Ni in such diseases and disorders.

Circumstances Associated with Induction of Ni Deficiency

1. Excessive Fertilization. Table 2 presents certain soil characteristics of six pecan orchards exhibiting severe Ni deficiency (where deficiency was corrected by foliar sprays of Ni). These six orchards are typical of most of those exhibiting Ni deficiency in the southeastern U.S. In each case, there was relatively high soil content of either Cu or Zn. In many cases, this high Cu or Zn content occurred simultaneously with high Ca and/or Mg content.

Because essentially all soils contain enough Ni to meet plant needs, and because Ni is a component of most P fertilizers (Martens and Westerman, 1991), deficiency likely arises from soil chemistry environments that reduce Ni uptake by roots. A key factor noted in all orchard situations exhibiting Ni deficiency was that there was always another metallic cation present in excessive amounts in the soil. In the case of pecan, this was usually Zn, but has also been observed to be Cu, Ca, Mn and Mg, or the combination thereof. Ni deficiency (i.e. ME-LL) was induced in potted greenhouse grown pecan trees by addition of high amounts of Fe, Mn, Zn, or Cu to soils. In the case of Ni deficiency symptoms of plum, symptoms were associated with high soil Zn, Mn or Fe. These
observations indicate the likelihood of either competitive and/or non-competitive inhibition of root Ni uptake by several metallic cations being widely used as fertilizers. Such elements are prone to accumulate in soils over many years or decades. These observations support those of Kochian (1991) who reported that Zn, Cu and Ni ion uptake from soils by feeder roots appears to be such that these ions share the same ion channels for entry into the root vascular system and thus competitively inhibit each other's uptake. It appears that the first period transition metals are absorbed as divalent ions via divalent ion channels (Graham and Stangoulis, 2003). Calcium and Mg appear to function as non-competitive inhibitors of Ni uptake. Ni deficiency was observed in both acidic and alkaline soils.

2. Nematodes. The feeder root system of most tree species is parasitized by one or more species of nematodes. In the case of pecan, it was found that root knot (Meliodogyne partiityla) can inflict enough damage to roots to induce severe Ni deficiency symptoms when grown in a soil from an orchard where certain trees exhibit Ni deficiency (Table 3). Anecdotal evidence indicates that this propensity is greatly enhanced when nematode infested trees are also growing in soils high in one or more divalent cations of the standard micronutrients. Severe Ni deficiency in young pecan trees was often (but not always) associated with root damage by nematodes. It appears that galling and feeding by nematodes act to limit Ni uptake and/or translocation. When this limitation occurs in concert with excessive amounts of certain metals in the soil, or the soil is cool and/or dry during early leaf development, then the severity of Ni deficiency increases.

The potential for the adverse influence of nematodes on plant Ni nutrition, plus the wide range of physiological processes being disrupted by insufficient Ni, raises the notion that Ni may be a missing link in certain recalcitrant disorders affecting several different crop species; especially those classified as replant disorders involving root damaging nematodes or fungi. Plant nutrition can potentially influence disease resistance (Graham, 1983, and Graham et al., 1985), thus Ni potentially may play a role in resistance to certain organisms associated with disease complexes and declines in certain crops.

3. Soil Temperature and Moisture. Many years of observation indicate that Ni deficiency symptoms usually are most frequently observed, and are often most severe, on soils that are dry, cool, or both, during early canopy development. Under such conditions, the severity of Ni deficiency clearly increases from base to top of the canopy. Both frequency and severity of Ni deficiency is most prevalent during dry springs.

Correction of Ni Deficiency

Previous work by Wood et al. (2004a, b) showed that Ni deficiency in pecan is easily corrected by timely foliar application of Ni salts or ligands. Observations by Wood et al. (2004a, b) of inconsistent partial reduction of ME-LL symptoms by application of Cu or P or S was subsequently found by Wood et al., (2004a, b) to be a Ni deficiency; with Cu and P sources containing Ni as a trace contaminant, and S apparently increasing soil Ni availability due to a reduction in soil pH.

One or two applications of Ni to foliage during the early canopy expansion phase, or soon after bud break, at a Ni concentration of 10-100 mg L⁻¹ (plus urea and a surfactant) corrects deficiency and ensures normal growth of tissues and organs (Wood et al., 2004a, b). Fig. 3 contrasts normal canopy development as a consequence of timely Ni treatment to abnormal growth on non-treated canopy of a tree exhibiting severe Ni deficiency. In the case of severe acute deficiency, an autumn application prior to leaf drop also ensures Ni availability to developing canopy the following spring. Foliage analysis during mid summer usually indicates Ni content considerably greater than does spring leaf analysis. It should be noted that Ni application only corrects morphological symptoms associated with tissue and organ growth subsequent to application and has no curative action on morphological growth distortions existing prior to application.
CONCLUSIONS

The discovery of field-level Ni deficiencies in pecan, river birch, and likely several other woody perennial crop species supports conclusions by Brown et al. (1987a) that Ni is indeed an essential plant nutrient. It is also evidence that undiagnosed Ni deficiency may be occurring in many woody perennial crops. Ni is therefore a mineral nutrient meriting consideration in husbandry of orchard, vine, and nursery operations.

Acute deficiency is recognizable by several morphological symptoms, with the key discriminating symptoms being dwarfed foliage with blunted necrotic apexes soon after bud break. To date, acute Ni deficiency has usually been associated with excessive soil Zn, Cu, Mn, Fe, Ca or Mg. Thus, decades of fertilizer applications to orchard or vineyard crops is likely to increase the probability of Ni deficiency becoming a problem, even though the soils may contain considerable amounts of Ni. Ni deficiency in orchards is most typically a fertilizer induced problem that has the potential of becoming increasingly common. This likelihood is increased when there is concurrent root damage by nematodes, especially in cool-dry soils during early canopy development.

The potential for induction of Ni deficiency by nematodes raises the possibility that replant problems exhibited by many crops may also include a Ni factor that has heretofore been overlooked. The discovery of Ni deficiency in orchard crops raises questions regarding the role of Ni in certain recalcitrant complex disorders of nutrition, disease, or tree decline.

Literature Cited


Tables

Table 1. Soil characteristics of a mouse-ear affected 2nd-generation ‘Desirable’ orchard planted at the same site where an 80-year-old ‘Success’ orchard previously grew. Half of the newly planted ‘Desirable’ trees were transplanted on the exact spatial location of pre-existing ‘Success’ trees (previous tree) and half were midway between pre-existing ‘Success’ trees (between trees).

<table>
<thead>
<tr>
<th>Transplant Location</th>
<th>Macronutrients (kg ha(^{-1}))</th>
<th>Micronutrients (kg ha(^{-1}))</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>p(H) CEC P K Mg Ca</td>
<td>Zn Mn Fe Cu Ni Co</td>
</tr>
<tr>
<td>Previous tree</td>
<td>6.1 8.4 205 323 224 1300</td>
<td>90 59 27 0.5 2.2 0.2</td>
</tr>
<tr>
<td>Between trees</td>
<td>6.1 8.1 230 310 220 1258</td>
<td>52 46 31 0.6 1.7 0.2</td>
</tr>
<tr>
<td>Significance</td>
<td>n.s.2 n.s. n.s. n.s. n.s. n.s.</td>
<td>* * n.s. n.s. * n.s.</td>
</tr>
</tbody>
</table>

1Transplants planted at exact spatial location where a 80-year-old ‘Success’ tree previously stood vs. planted mid-way between where ‘Success’ trees previously grew.
2* = significantly different by ANOVA at \(p\leq0.05\) level; n.s. = nonsignificant.

Table 2. Soil characteristics of orchards exhibiting severe Ni deficiency.

<table>
<thead>
<tr>
<th>Orchard</th>
<th>pH</th>
<th>CEC 2</th>
<th>P</th>
<th>K</th>
<th>Mg</th>
<th>Ca</th>
<th>Mn</th>
<th>Fe</th>
<th>Zn</th>
<th>Cu</th>
<th>B</th>
<th>Ni</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fla. #1</td>
<td>6.8</td>
<td>3.9</td>
<td>77S 1</td>
<td>49VL</td>
<td>114S</td>
<td>736S</td>
<td>7L</td>
<td>26H</td>
<td>16L</td>
<td>4VH</td>
<td>0.3</td>
<td>0.6</td>
</tr>
<tr>
<td>Fla. #2</td>
<td>6.6</td>
<td>5.3</td>
<td>117H 1</td>
<td>74L</td>
<td>160H</td>
<td>1005H</td>
<td>10L</td>
<td>27VH</td>
<td>36S</td>
<td>6VH</td>
<td>0.4S</td>
<td>0.5</td>
</tr>
<tr>
<td>Fla. #3</td>
<td>6.2</td>
<td>3.0</td>
<td>34L</td>
<td>19VL</td>
<td>68L</td>
<td>366L</td>
<td>4VL</td>
<td>21H</td>
<td>5L</td>
<td>4VH</td>
<td>0.2</td>
<td>0.4</td>
</tr>
<tr>
<td>Ga. #1</td>
<td>7.1</td>
<td>12.0</td>
<td>137VH</td>
<td>1458</td>
<td>561VH</td>
<td>2976VH12L</td>
<td>12S</td>
<td>58VH</td>
<td>2H</td>
<td>0.7S</td>
<td>1.4</td>
<td></td>
</tr>
<tr>
<td>Ga. #2</td>
<td>6.8</td>
<td>7.5</td>
<td>77S 1</td>
<td>53L</td>
<td>122H</td>
<td>1062H</td>
<td>10L</td>
<td>20H</td>
<td>261VH2H</td>
<td>0.9S</td>
<td>0.9</td>
<td></td>
</tr>
<tr>
<td>Ga. #3</td>
<td>6.6</td>
<td>13.3</td>
<td>403VH</td>
<td>863VH</td>
<td>196H</td>
<td>2952VH61VH24H</td>
<td>228VH4VH</td>
<td>1.4S</td>
<td>1.1</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

1Example orchards in Florida and Georgia exhibiting severe Ni deficiency.
2Cation exchange capacity (meq 100 g\(^{-1}\) soil).
3Elemental content according to Georgia Cooperative Extension Service recommendations for pecan (VL=very low; L=low; S=sufficient; H=high; VH=very high).
Table 3. Influence of *Meliodogyne partityla* nematode on Ni deficiency symptoms exhibited by ‘Desirable’ pecan grown in field microplots and rated 9 and 23 months after inoculation.

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Mouse-ear severity rating</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>May 17&lt;sup&gt;th&lt;/sup&gt;, 2004</td>
</tr>
<tr>
<td>Pasteurized soil + <em>M. partityla</em></td>
<td>8.3 a&lt;sup&gt;2&lt;/sup&gt;</td>
</tr>
<tr>
<td>Non-pasteurized soil</td>
<td>5.9 b</td>
</tr>
<tr>
<td>Pasteurized soil</td>
<td>4.6 b</td>
</tr>
</tbody>
</table>

1 = no ME; 2 = 1-25% leaf distortion; 3 = 26-50% leaf distortion; 4 = >50% leaf distortion; 5 = >50% leaf distortion + leaflet cupping; 6 = #5 plus necrosis of leaflet tips; 7 = #6 plus dark green zone; 8 = #7 plus stunted shoots; 9 = #8 plus multiple stunted shoots; 10 = #9 plus death.

Figure 1. Influence of Ni deficiency on pecan. A. reduced leaf and leaflet size as degree of Ni deficiency increases; B, altered shape of blade to produce mouse-ear-like or little-leaf foliage; C, blunting, crinkling, dark green zone, and necrosis of the apical portion of the leaf blade; D, absence of leaf lamina development in severe cases, plus deformed and pointed buds.
Fig. 2. Influence of Ni deficiency on pecan. A, loss of apical dominance and resetting; B, delayed bud break (degree of Ni deficiency decreasing from left to right); C, shorten internodes, dwarfed and weakened shoots possessing weak pointed buds (Ni deficient on left and Ni sufficient on right); D, dwarfed trees (tree is 1m tall at age 10 years).
Fig. 3. Influence of post bud break foliar spray of 100 mg L\(^{-1}\) Ni (as nickel sulfate) on correction of Ni deficiency of pecan. Branches on the left were treated with Ni whereas those on the right of the canopy were non-treated. Canopy development is about five weeks after bud break.