Mouse-ear of Pecan: A Nickel Deficiency

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Abstract. Mouse-ear (ME) is a potentially severe anomalous growth disorder affecting pecan [*Carya illinoensis* (Wangenh.) K. Koch] trees. It is especially severe in second generation sites throughout much of the Gulf Coast Coastal Plain of the southeastern U.S., but can also occur in potted nursery trees. Orchard and greenhouse studies on trees treated with either Cu or Ni indicated that foliar applied Ni corrects ME. ME symptoms were prevented, in both orchard and greenhouse trees, by a single mid-October foliar spray of Ni (nickel sulfate), whereas nontreated control trees exhibited severe ME. Similarly, post budbreak spring spray applications of Ni to foliage of shoots of orchard trees exhibiting severe ME prevented ME symptoms on subsequent growth, but did not correct morphological distortions of foliage developed before Ni treatment. Foliar application of Cu in mid-October to greenhouse seedling trees increased ME severity the following spring. Post budbreak application of Ni to these Cu treated MEed seedling trees prevented ME symptoms in post Ni application growth, but did not alter morphology of foliage exhibiting ME before Ni treatment. Thus, high leaf Cu concentrations appear to be capable of disrupting Ni dependent physiological processes. Foliar application of Ni to ME prone trees in mid-October or soon after budbreak, is an effective means of preventing or minimizing ME. These studies indicate that ME in pecan is due to a Ni deficiency at budbreak. It also supports the role of Ni as an essential plant nutrient element.

A Mouse-ear (ME) is a growth abnormality of pecan [*Carya illinoensis* (Wangenh.) K. Koch] first reported in 1918 by Marz (1918). It was initially exhibited by yard trees within certain Florida, southern Mississippi, and southeastern Georgia cities (Demaree, 1926). The disorder was initially attributed to spring cold injury before budbreak, but was later attributed to a disease pathogen (Demaree, 1926). It was evident in pecan orchards by the 1930's and is now a common anomaly. Gammon and Sharpe (1956) concluded that the problem was a manganese deficiency; however, soil or foliar application of Mn to affected trees does not correct ME in contemporary orchards. Mouse-ear symptoms range from mild morphological distortion of leaflets to gross deformity of shoot, foliar, and reproductive organs (Wood et al., 2003a).

There has been considerable establishment of second generation pecan orchards and replacement of missing orchard trees over the last 20 years in the southeastern U.S. It is common for these newly transplanted trees to exhibit ME symptoms the second or third year after transplanting. In many cases symptoms are so severe that transplants die. This replant-associated form of ME is a serious economic problem for many orchard operations throughout the Georgia pecan belt and certain orchards within the Gulf Coast Coastal Plain of the southeastern U.S.

Mouse-ear symptomology, as recently

described by Wood et al. (2003a), implicates a micro nutrient deficiency or imbalance as the causal factor. This conclusion was further supported by observations by Wood et al. (2003b) that soil applied Cu at time of transplanting corrected ME on a second generation site. Trees exhibiting mild to moderate ME growing on a first generation site were largely cured of ME by the third year after topical application of Cu to the soil; however, foliar applications of Cu generally had little or no influence on ME severity. Neither foliar or soil application of Cu to severely MEed trees were efficacious for reducing symptoms. It was also noted that on a first generation orchard site, soil application of high amounts of either P or S corrected ME symptoms three years after application. Wood et al. (2004a) also noted a strong statistical linkage between Cu and Zn in regards to ME severity, thus implicating a Zn induced temporary deficiency of Cu as the likely cause of ME. However, these findings did not exclude deficiencies of other divalent metallic cations (e.g., Ni, Co, Ti, or V) as being the cause of ME.

Wood et al. (2004a, 2004b) observed a strong relationship between ME severity and soil Zn content, thus implicating Zn as a contributing factor to ME. Because Zn, Cu and Ni ion uptake from soils by feeder roots appear to share the same ion channels for entry into the root vascular system (Kochian, 1991), it becomes apparent that ME might be caused by either a Ni or Cu deficiency that is being induced by excessive soil Zn on second generation sites and by low soil Ni or Cu on first generation sites. The present study evaluates whether severe ME can be corrected by foliar

applications of either Ni or Cu and concludes that ME is caused by Ni deficiency.

Materials and Methods

Field studies. Severely affected ME trees were treated in commercial orchards throughout the Gulf Coast Coastal Plain of Georgia. Soil types among affected orchards differed substantially, but were almost always either sandy loams or sands. Affected trees were found on sites previously supporting pecan orchards or were replacement trees in existing mature orchards. Although, ME affected trees sometime occurred on sites where pecan had not previously grown. In the latter case, these soils possessed very low cation exchange capacities. Most of the affected cultivars were 'Desirable' and were from 5 to 10 years old. The rootstocks are unknown, but are likely seedlings of 'Elliott', a commonly used seed source for pecan rootstock in the southeastern U.S.

Fall application. The ability of fall foliar application of either Cu or Ni to correct ME was evaluated in several orchards. The experiment consists of three micro nutrient treatments (Control, Cu and Ni) applied 15 Oct. as a foliar spray to major branches of severely MEed 'Desirable' trees. Individual trees served as replicates comprised of all three micro nutrient treatments. Treatments were spatially separated within the tree canopy so as to avoid cross-contamination of sprays. All treatments contained reagent grade urea (at 4.8 g·L⁻¹) and a nonionic surfactant (2.5 mL·L⁻¹) as additives. The Cu source was CuSO₄ · 5 H₂O at a concentration of 4 g·L⁻¹ (1 g·L⁻¹ Cu). The Ni source was NiSO₄ · 6 H₂O at a concentration of 3.5 g·L⁻¹ (0.8 g·L⁻¹ Ni). Both salts used in this experiment, and in the following experiments, were 99% A.C.S. reagent grade (Aldrich, Milwaukee, Wisconsin). Control treatments received only reagent grade urea and surfactant. Sprays were applied to foliage till run-off. There were 20 replications dispersed over two orchard sites. The experiment was a RCB design comprised of three treatments with single tree blocks. Treatments were evaluated the following spring (1 May) for severity of ME. Severity was based on the following scale; 1 = no symptoms, 2 = between 1% and 25% of number of leaflets per shoot exhibiting blunting; 3 = 26% to 50% of number of leaflets per shoot exhibiting blunting; 4 = >50% of number of leaflets per shoot exhibiting blunting; 5 = cupping of blunted leaflets; 6 = necrosis of leaflet tips; 7 = dark green zone near leaflet tip; 8 = stunted shoots; 9 = multiple new shoots (i.e., witches broom); 10 = dead shoot. Data were statistically analyzed for mean separation of treatments by use of JMP (SAS, Cary, N.C.; SAS Institute, 2002). Nickel content of foliage was determined by ICP spectroscopic analysis using standard techniques. Several leaves were collected per treatment in June, bulked, rinsed in 0.1 M HCl, triple rinsed in deionized water, dried at 55 °C, ground in a ceramic mortar and pestle before processing for analysis.

Spring application. This experiment addressed Cu or Ni applied to expanding shoots

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Table 1. Influence of fall and spring foliar applications of Cu or Ni on mouse-ear severity of pecan trees grown in orchards or potted seedlings in a greenhouse.

Treatment			Mouse-ear severity
Chemical ²	Application time ³	Tree type ⁴	(class) ⁵
Control	Fall–15 Oct.	Orchard transplant	7.4 a ^v
Cu	Fall–15 Oct.	Orchard transplant	7.6 a
Ni	Fall–15 Oct.	Orchard transplant	1.0 b
Control	Early spring–1 Apr.	Orchard transplant	7.7 b
Cu	Early spring–1 Apr.	Orchard transplant	8.1 a
Ni	Early spring–1 Apr.	Orchard transplant	1.0 c
Control	Mid-spring–Ma ⁶	Greenhouse seedling	8.1 a
Ni	Mid-spring–Ma	Greenhouse seedling	1.0 b

²Treated with sulfate salts of either copper or nickel with urea and nonionic surfactant as carrier. The control treatment received carriers but not the salts.

³Treatments were applied to foliage in either mid-October (fall) or early April about 10 d postbudbreak (spring).

⁴Treated trees were either orchard transplants or seedlings growing in a greenhouse.

⁵Severity class of 1 = no mouse-ear symptoms, whereas class 10 = most severe symptoms (i.e., gross blunting, curling, necrosis of leaflets; plus stunted shoots, multiple shoots, and death of shoots).

⁶Treatment means within each experiment are statistically different according to Tukey-Kramer HSD test ($\alpha = 0.05$) if followed by different letters.

⁷Treatments applied early May after shoots were about one-half expanded, but youngest leaves and leaflets were in early stage of expansion.

soon after budbreak. The experimental design, treatments, and chemical sources were identical to the above fall application, except different trees were used. The expanding foliage and shoots were treated during the late parachute stage of leaf development in early April. All treatments contained reagent grade urea (4.8 g·L⁻¹) and a nonionic surfactant (2.5 mL·L⁻¹) as additives and applied till runoff. Treatments were assessed and analyzed as described above.

Greenhouse studies. The influence of Cu and Ni on ME of potted seedlings was evaluated by treating 1-year-old 'Desirable' pecan seedlings potted in soil from an orchard exhibiting severe ME. Seedling trees were germinated in May in vermiculite and transplanted in June to 15 × 10 cm rectangular plastic pots containing

soil (95% sand, 0% silt, 4% clay; pH 7.0, o.m. 1.3%) from a severely MEed orchard near Cordele, Ga. Seedlings were assessed and analyzed as described above.

Fall application. Trees were treated in October, just before leaf drop, with a single foliar spray of either Cu or Ni. Copper and Ni were applied at one of five concentrations; i.e., 0, 1x, 2x, 4x, or 8x; where for Cu, X = 1.98 g CuSO₄ · 5 H₂O/L; and for Ni, X = 3.53 g NiSO₄ · 6 H₂O/L. Spray application till run-off soaked the foliage while a barrier was used to prevent soil contamination. Trees were maintained until natural leaf drop in November by watering beneath the foliar canopy. Treated seedlings were left in unheated greenhouse during the dormant season. Trees broke bud about 15 Mar. and were subsequently rated for ME severity

using the above described scale and data analyzed for treatment differences. The study was structured as two distinct experiments (one Cu and the other Ni) in or with a RCB design with three blocks of five elemental concentrations (0x, 1x, 2x, 4x, and 8x) and eight trees per experimental unit. All treatments contained urea (4.8 g·L⁻¹) and a nonionic surfactant (2.5 mL·L⁻¹) as additives.

Spring application. A third Ni study was conducted using seedling trees of the above-mentioned greenhouse Cu study. Because Cu treatments, regardless of the amount of Fall Cu applied to foliage, exhibited severe ME the following spring, affected trees were segregated and randomized and structured in a third experiment in which they were treated with a foliar spray of Ni (NiSO₄ · 6 H₂O). Experimental design was a RCB comprised of three blocks (blocked by severity of ME) of two Ni treatments (0 vs. 3.53 g·L⁻¹ as the sulfate salt) with 4 trees per experimental unit. Ni treatments were applied early May and the new shoot growth subsequently evaluated for ME severity. All treatments included urea (4.8 g·L⁻¹) and a nonionic surfactant (2.5 mL·L⁻¹) as additives. Data were then statistically analyzed for treatment differences.

Results and Discussion

Field Studies

Fall application. Limbs of trees receiving foliar sprays of Ni in October did not exhibit symptoms of ME the following spring whereas Cu treated and nontreated controls on the same tree exhibited severe ME (Table 1, Fig. 1). Shoots arising from Ni treated branches exhibited a ME rating of 1.0 (i.e., no symptoms of ME) whereas control trees exhibited a ME severity rating of 7.4 (i.e., severe ME) and the Cu treatment exhibited a severity rating of 7.6.

The October Ni treatment did not visually appear to be phytotoxic to either fall or spring foliage. These data indicate that the ME replant disorder of young pecan trees is tightly linked to a physiological deficiency of Ni. This conclusion is supported by previous research reporting that Ni, Zn, and Cu are absorbed by the same carrier channel in roots and therefore competitively inhibit each others uptake from the soil solution (Kochian, 1991). Circumstantial evidence indicates that high soil Zn, arising from many decades of foliar sprays to correct Zn deficiency, is likely contributing to a competitive inhibition of spring Ni uptake by roots of young transplanted trees. A similar reduction in Ni uptake may also occur in mature trees, but with the degree of inhibition usually being insufficient to trigger ME symptoms; although, there may be unrecognized disruption of one or more growth and developmental processes via disruption of Ni associated physiology.

These data indicate that Ni is absorbed by pecan foliage and is translocated from foliage to storage pools in dormant season tissues of shoots and/or buds. Although these amounts were not quantified, the amount translocated was enough to correct the deficiency of Ni

Fig. 1. Comparison of April 2003, shoot growth from severely mouse-eared trees treated with a foliar spray of Ni the previous October and a nonsprayed control. The Ni treatment is on the left and the nontreated control is on the right.

Fig. 2. Influence of spring foliar application of Ni on severity of mouse-ear symptoms of orchard trees. The branch on the left side of the tree was treated with Ni soon after budbreak whereas the right portion of the trees was not treated.

at budbreak and to enable normal growth processes. Foliar Ni content the following spring was $7 \text{ mg} \cdot \text{kg}^{-1}$ in treatments receiving foliar Ni the previous October (Table 2). This compares to Ni at $0.5 \text{ mg} \cdot \text{kg}^{-1}$ in foliage from non treated tissues of trees exhibiting severe ME. Adjacent trees not exhibiting ME had a foliar Ni concentration of $4 \text{ mg} \cdot \text{kg}^{-1}$.

Spring application. Randomly selected limbs of all treatments initially exhibited severe ME immediately after budbreak, but before treatment with either Cu or Ni. However, after Ni was applied, subsequent shoot, leaf, and leaflet growth (after about 10 to 15 d) no longer exhibited ME; whereas subsequent growth after Cu application continued to exhibit severe ME symptoms (Table 1). Nontreated control shoots also exhibited severe ME. Sub-

sequent shoot growth arising from Ni treated branches exhibited a ME rating of 1.0 (i.e., no ME symptoms) whereas control branches exhibited severe ME at a rating of 7.7 (Table 1, Fig. 2). Conversely, shoots after the spring Cu treatment exhibited a severity rating of 8.1, which was considerably more severe than the control treatment. Thus, the severity of ME can be increased by application of Cu. The April Ni application induced slight, yet noticeable, phytotoxicity to young foliage inasmuch that leaflet tips and margins sometime possessed small necrotic spots.

Ni concentration of foliage from branches of trees treated with Ni to correct ME was $26 \text{ mg} \cdot \text{kg}^{-1}$ compared to $0.4 \text{ mg} \cdot \text{kg}^{-1}$ in foliage of branches on the same trees that exhibited severe ME (Table 2). Ni concentration of

foliage in adjacent trees not exhibiting ME was $5 \text{ mg} \cdot \text{kg}^{-1}$.

These data confirm the findings of the fall treatments that the ME replant disorder of young pecan trees is tightly linked to Ni and is Ni deficiency. The spotty necrosis of very young developing foliage indicates that the optimal concentration of Ni for spring applications is less than that used in the present study. The ability of foliar sprays of Cu to increase the severity of ME is taken as evidence that high Cu content may potentially disrupt Ni associated physiological processes.

Greenhouse Studies

Fall application. October foliar application of Ni and Cu to seedling trees in the greenhouse was such that Ni reduced and Cu increased ME severity (Fig. 3). The severity of ME decreased greatly with Ni treatments $\geq 1\times$. No ME was evident in the $2\times$, $4\times$, and $8\times$ treatments, and very little in the $1\times$ treatment. These data support those from field trees that fall applications of Ni to foliage will prevent the occurrence of ME the following spring. Conversely, fall application of Cu to seedling trees failed to correct ME and actually increased the severity of ME as the concentration of applied Cu increased (Fig. 3). Thus, ME does not appear to be associated with a Cu deficiency, as was previously suspected (Wood et al., 2004a, 2004b). This increased severity of ME in Cu treated seedlings, as a result of increasing amounts of foliar Cu applied the previous October, provides evidence that excessive Cu is interfering with Ni related physiological processes.

Spring application. Application of Ni two weeks after budbreak to ME seedlings previously treated with high amounts of Cu, corrected ME symptoms in developing shoots, but had no curative effect on the morphology of older leaflets present before Ni application (Table 1). These data, a) support the above described field studies that post budbreak foliar application of Ni can correct ME of expanding foliage, b) indicates that Ni corrects the increased severity of ME caused by foliar sprays of Cu, and c) that Ni and Cu are likely competing in certain physiological processes. Previous observations regarding the occurrence of severe ME at an orchard site also high in Zn, plus increased ME severity when Zn is placed in the planting hole of new transplants (Wood et al., 2004b), is evidence that Zn may possibly compete with Ni in certain key physiological processes or is somehow reducing the physiological availability of Ni.

Eskew et al. (1983 and 1984) identified Ni a likely essential micronutrient in higher plants. Subsequently, Brown et al. (1987a, 1987b) demonstrated nickel's essentiality as an essential plant nutrient element in several grain crops and suggested that it be recognized as an essential nutrient element for higher plants. It has therefore been noted as being of potential important in the cultivation of parsley [*Petroselinum crispum* (Mill.) Nyman ex A.W. Hill] (Atta-Aly, 1999), grains (Brown et al, 1990), legumes (Eskew, et al., 1983, 1984), and cucurbids (Watanabe and Shimada, 1990); po-

Table 2. Ni content of foliage from orchard 'Desirable' trees exhibiting severe mouse-ear (ME) symptoms and trees in which ME was corrected with a foliar application of Ni.

Treatment ^a	Mouse-ear severity (class) ^b	Foliar Ni concn ^c (mg·kg ⁻¹)
Normal tree control	1.0 b ^w	4.0 b ^w
Severe ME control	7.4 a	0.5 c
Severe ME + fall Ni	1.0 b	7.0 a
Normal tree control	1.0 b	5.0 b
Severe ME control	7.7 a	0.4 c
Severe ME + spring Ni	1.0 b	26.0 a

^aTreated with sulfate salts of nickel using with urea and nonionic surfactant. Treatments were applied to foliage in either mid-October (fall) or early April about 10 d postbuddbreak (spring).

^bSeverity was based on the following scale: 1 = no symptoms, 2 = between 1 and 25% of number of leaflets per shoot exhibiting blunting; 3 = 26% to 50% of number of leaflets per shoot exhibiting blunting; 4 = >50% of number of leaflets per shoot exhibiting blunting; 5 = cupping of blunted leaflets; 6 = necrosis of leaflet tips; 7 = dark green zone near leaflet tip; 8 = stunted shoots; 9 = multiple new shoots (i.e., witches broom); 10 = dead shoot. Note that severity of the 'Severe ME + spring Ni' treatment is based on a rating of post-treatment foliage.

^cFoliar Ni content is based on sampling of foliage in mid July, after Ni treatments the previous October and April. Content is based on dry weight.

^wMeans are statistically different according to Tukey-Kramer HSD test ($\alpha = 0.05$) if followed by different letters.

tato (*Solanum tuberosum*) (Roach and Barklay, 1946), grape (*Vitis vinifera* L.) (Dobrolyubskii and Slavko, 1957), and soybean [*Glycine max* (L.) Merrill] (Bertrand and de Wolf, 1973), but has received little or no attention as a needed supplement in tree crops or in fields, orchards, or vineyards. Because of the relative abundance of Ni in soils, and the apparent low Ni requirement of plants, it was thought that Ni would not be a practical problem in the field (Brown et al., 1987a). The above described studies on pecan indicate that the role of Ni in pecan tree nutrition has heretofore been overlooked. The chlorotic, or yellowish, coloration of leaflets and meristems of pecan trees with moderate to severe ME (Wood et al., 2004a) is consistent with Ni deficiency symptoms of several plant species (Welch, 1995). ME symptoms of growth depression and apparent disrupted protein synthesis and enzyme activity in pecan are consistent with Ni deficiency characteristics noted in other crops (Brown, 1990).

Wood et al. (2004b) noted that topical application of Cu, P, or S corrected ME symptoms by three years after application to a first generation orchard site. It was also noted that inclusion of either Cu or P in the backfill soil of transplanted trees reduced ME symptoms in proportion to the amount added, whereas Zn increased ME severity in proportion to the amount added. On the surface, these data provided evidence that Cu was the likely cause of ME. However, foliar applications of Cu over a wide concentration range over many different application dates did not correct the severe form of ME, but could occasionally reduce severity of a mild form of ME. Subsequent analyses of the P and Cu sources used in these studies

Fig. 3. Influence of October foliar application of Cu and Ni at different treatment concentrations on severity of mouse-ear the following spring. Treatments are such that for Ni $X = 3.53 \text{ g NiSO}_4 \cdot 6 \text{ H}_2\text{O/L}$ (A), and for Cu $X = 1.98 \text{ g CuSO}_4 \cdot 5 \text{ H}_2\text{O/L}$ (B). For Ni, $y = 1/(x + 0.205)$, $R^2 = 0.45$, $P = 0.0001$; for Cu: $y = 2.95 + 2.06x - 0.183x^2$, $R^2 = 0.54$, $P = 0.0001$

revealed the presence of trace amounts of Ni (3 to 8 mg·kg⁻¹). Thus, it appears that ME was not being corrected by Cu or P, but rather by the Ni contaminate. Phosphorus fertilizers typically contain Ni as a trace contaminate (Martens and Westermann, 1991). Correction using S was likely because of a substantial soil acidification and subsequent increase in Ni availability on an orchard site low in both soil Ni and Zn. The influence of Zn on ME severity is evidence that high soil Zn indeed competitively inhibits the uptake of Ni, thus resulting in Ni deficiency. The observation by Gammon and Sharp (1956) that Mn corrected Ni deficiency, but failed to do so in recent decades, is taken as evidence that the source of Mn used during that era likely contained trace amounts of Ni whereas improved manufacturing processes in recent years have substantially reduced the amount of Ni contamination in the Mn fertilizers.

The mid July to mid August Ni concentration of pecan foliage in Georgia is usually <4 mg·kg⁻¹ and is often <1 mg·kg⁻¹ in many orchards. This period is when foliage is sampled to assess tree nutritional status and fertilizer needs for the following growing season. Results from the present studies indicate that Ni nutritional needs of pecan merit reassessment. It is possible that pecan trees require foliar Ni concentrations higher than those typically reflected in leaf analysis reports. The role of Ni in pecan nutrition is not fully understood and was not included in the two most recent reviews of pecan macro and micro nutrient

nutrition (Worley, 1994; Smith, 1991).

In the case of ME, it is possible that absolute or physiologically available Ni (and possibly Cu) at budbreak, and a few weeks thereafter, is at a subcritical content due to any one of several factors. These factors could be adsorption to N-ligands, uptake inhibition by other divalent metals in the soil (e.g., Zn) or due to dry soils. The absolute concentration of Ni in foliage may not be a reliable indication of Ni nutritional status, as the severity of ME in the above described greenhouse study was such that increasing the amount of Cu applied to foliage increased the ME severity. This implies that leaf Cu concentration can alter the physiologically available concentration of Ni or its ability to be used by certain enzymes. Additionally, Wood et al. (2004b) previously observed that foliar treatment of MEed trees with Fe enhanced the severity of ME (this was also observed with soil applications of Fe or Cu to potted pecan trees, unpublished observations). It therefore appears that the content of other trace metals in cells of pecan tissues can disrupt Ni nutrition.

The Ni nutrition of pecan, and likely that of other woody perennials, is likely affected by a wide variety of orchard management strategies known to influence Ni availability and uptake. Most notable among these are soil moisture and soil solution and leaf content of divalent cations, soil pH, P or Ca or Mg fertilizers, and nitrogen fertilization practices (Wood et al., 2004b). Thus there is much that an orchardist can do to enhance the Ni nutrition of trees without application of Ni. These are discussed in detail by Wood et al. (2004b) within the context of Cu, but the same actions and interactions for Cu are likely to also be generally true for Ni.

Conclusions

The correction and prevention of ME symptoms in both field and greenhouse trees by foliar sprays of Ni indicates that the primary fundamental cause of ME is either closely related to, or is caused by, Ni deficiency. The occurrence and prevention of ME with Ni, in first generation orchards, some of which had not supported crops for at least 100 years, is evidence that pecan trees growing on certain orchard sites are likely Ni deficient during a portion of the spring, yet are not so deficient as to exhibit ME symptoms. Thus, pecan Ni nutrition management needs to be reassessed.

In cases of severely mouse-eared trees growing in second generation orchards, it appears that ME is caused by a severe Ni deficiency induced by high soil Zn. This high soil Zn is a side-effect of ≥50 years of Zn applications to pecan orchards for the correction of Zn deficiency. Thus, high soil Zn, and perhaps high plant Zn, appear to be directly or indirectly disrupting Ni related physiological processes of pecan. These results indicate that Zn management strategies for pecan merit reevaluation and imply that long-term applications of Cu or Zn containing fertilizers to woody plant crops may possibly induce Ni deficiency.

The influence of Ni on mouse-eared pecan

trees provides field evidence to support the contentions of Eskew et al. (1983, 1984) and Brown et al. (1987a, 1987b) that Ni is an essential plant nutrient element. This is further supported in that Ni deficiency in pecan can be so severe as to cause young trees to die before fruiting, thus satisfying a key criteria for essentiality (Marschner, 2002). Because Ni uptake is directly inhibited by excessive Zn and Cu and indirectly inhibited by excessive nitrate-N, Ca and Mg; and because Ni is typically overlooked in plant nutrition management, it is postulated that Ni deficiencies are far more common in potted woody plants and orchard trees than commonly recognized. Such deficiencies are most likely to occur as a result of excessive fertilization with other metals or with N. Based on our experience with pecan, extreme cases of Ni deficiency are likely to be recognized by some combination of the following symptoms: blunted foliage, dwarfing, delayed budbreak, necrotic zones at the tip of leaves or leaflets, brittle shoots and branches, loss of apical dominance, rosetting, crinkled leaf or leaflet margins, slightly chlorotic foliage that later turns dark green, reduced growth, short internodes, and reduced flowering. The results of this study raises a multitude of questions regarding the economic, nutritional, and environmental impact of potentially unrecognized Ni deficiencies in a wide variety of horticultural crops and supports the role of Ni as an essential plant nutrient element.

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