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Foliar Nickel Application Can Increase the Incidence of Peach Tree Short Life and Consequent Peach Tree Mortality

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Abstract. **Peach tree short life (PTSL) is associated with the presence of ring nematode, *Mesocriconema xenoplax*, and poor orchard management practices. The ability of post-plant nickel (Ni) foliar application to suppress *M. xenoplax* population density and thereby prolong survival of peach trees on a PTSL site infested with *M. xenoplax* was investigated from 2004 to 2011. For this study, the site was divided into plots, which received the following treatments: 1) Ni (foliar-applied); 2) methyl bromide fumigation (MBr); and 3) an untreated control. Peach trees were planted into all plots in Mar. 2005 and the foliar Ni treatment was applied three times in 2005 and 2006. Nickel did not detectably suppress *M. xenoplax* populations as compared with MBr fumigation. The protective effect of MBr fumigation in suppressing *M. xenoplax* population density persisted for 27 months after orchard establishment. Trees receiving multiple foliar Ni applications at 0.45 g·L^{-1} over 2 years, while exposed to *M. xenoplax*, exhibited greater PTSL mortality than trees growing in untreated or MBr-fumigated plots. These results suggest that foliar applications of Ni to peach trees, growing on a PTSL site, should be used with caution in commercial orchards because these treatments can deleteriously disrupt tree metabolic/physiological processes sufficient to increase the incidence of PTSL tree mortality.**

The productive lifespan of peach [*Prunus persica* (L.) Batsch] trees in commercial orchards within the southeastern United States is generally only 6 to 10 years on some sites as a result of premature tree mortality (Brittain and Miller, 1978). Two common causes of early tree death are a disease complex known as PTSL and Armillaria root rot (Miller, 1994). Peach tree short life is reportedly caused by a predisposition of trees to bacterial canker (*Pseudomonas syringae* pv. *syringae* van Hall), cold injury, or a combination of both that is the consequence of root feeding by the ring nematode, *Mesocriconema xenoplax* (Raski, 1952) Loof and de Grisse, 1989 [= *Criconemoides xenoplax* (Raski, 1952; Loof and de Grisse, 1967)], (Brittain and Miller, 1978; Nyczepir et al., 1983). *Mesocriconema xenoplax* is a root ectoparasitic nematode that has the ability to adversely influence peach tree growth as a result of its feeding (Nyczepir et al., 1987). In field microplot studies, peach trees died from cold injury 4 years after parasitism by *M. xenoplax*, whereas trees in uninfested soil survived (Nyczepir et al., 1983). Also, the development of PTSL on land not planted with peaches for 75 years or longer depends on exposure of trees to the increasing population levels of *M. xenoplax* over time (Nyczepir et al., 2004). This evidence indicates that PTSL is a nematode-associated disease complex and is tightly linked to the presence of this ring nematode species.

A 10-point Management Program is recommended to peach growers in the southeastern United States to reduce tree loss from PTSL (Brittain and Miller, 1978). Although this program fails to fully resolve the PTSL problem, it is to date the best management system for the disease complex. Two major points to this program are preplant soil fumigation to reduce *M. xenoplax* populations and the use of the Guardian® (= Guardian hereafter) rootstock, which is the recommended rootstock of choice for PTSL sites, in conjunction with preplant fumigation (Horton et al., 2011). This rootstock was identified as providing greater tree survival than the recommended Lovell on PTSL sites, although *M. xenoplax* is capable of reproducing on it (Nyczepir et al., 1983; Okie et al., 1994). It has also been suggested that one reason Guardian survives longer on PTSL sites than other rootstocks (i.e., Nemaguard) is because Guardian does not allow nonstructural carbohydrate reserves to be partitioned from shoot to root in response to ring nematode parasitism (Nyczepir et al., 1987; Olien et al., 1995). Furthermore, peach tree susceptibility to *Pseudomonas syringae* pv. *syringae* in the presence of *M. xenoplax* is enhanced by low nitrogen and high calcium plant tissue content (Cao et al., 2006). They postulate that one possible reason why nitrogen fertilization decreased host susceptibility to *Pseudomonas syringae* pv. *syringae* is by quantitatively reducing the plant metabolites that induce *syrB* (*syrB* being the gene responsible syringomycin synthesis) gene expression or by producing increased concentration of biochemicals that antagonize *syrB* inducing compounds (Cao et al., 2005). Additionally, the beneficial effect of copper sprays throughout the dormant season in combination with biannual applications of nitrogen–phosphorus–potassium plus micronutrients significantly reduced bacterial canker disease severity in French prune (*Prunus domestica* L.), whereas copper sprays alone were ineffective (Sayler and Kirkpatrick, 2003). In contrast, spray applications of copper alone effectively suppresses bacterial canker infection in apricot (*Prunus armeniaca* L.) (Wimalajeewa et al., 1991). Other micronutrients (e.g., Ni) have also been shown to be effective in managing plant diseases caused by fungi (Reilly et al., 2005), bacteria (Wang et al., 2000), or nematodes (Khan and Salam, 1990). It is possible that these essential micronutrients induce resistance through *in planta* phytoalexin production. Daylily rust (*Puccinia hemerocallidis* Thüm) was suppressed for up to 15 d with a single aqueous foliar application of Ni (as $NiSO_4$) (Reilly et al., 2005). Additionally, incidence of bacterial blight [*Xanthomonas oryzae* pv. *oryzae* (Ishiyama) Swings et al.] (Wang et al., 2000) and root galling caused by root-knot nematode [*Meloidogyne javanica* (Treub) Chitwood] (Khan and Salam, 1990) are less severe in rice and pigeon pea seedlings, respectively, after being exposed to Ni. Furthermore, Ni deficiency symptoms of pecan seedlings induced by root-knot nematode (*Meloidogyne partityla* Kleynhans) are correctable by timely foliar Ni application (Nyczepir et al., 2006; Wood et al., 2004). Plant Ni nutritional physiology can affect many physiological processes, especially those involving nitrogen (N)-associated metabolism (Bai et al., 2006, 2007, 2008); and N metabolism can affect cold-hardiness and the production of secondary metabolites potentially involved in disease resistance (Wood and Reilly, 2007). The influence of Ni on *M. xenoplax* reproduction and incidence of PTSL is unknown. The present study evaluates whether Ni foliar application suppresses *M. xenoplax* reproduction or influences tree survival on a PTSL site.

Materials and Methods

Field plot establishment. The experiment was initiated in May 2004 at the USDA, ARS Southeastern Fruit and Tree Nut Research Laboratory in Byron, GA. The study was established on a Faceville sandy loam soil (78% sand, 14% silt, 8% clay; pH 5.7; 1.79% organic matter) with a history of PTSL. Peach trees had been growing on this site since 1998

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and were removed May 2004. Immediately after tree removal, the test site was subsoiled (≈81 cm deep) and rotovated. The test site was then divided into six adjacent blocks, each measuring 27 × 6.1 m. Treatments within each block included: 1) Ni (foliar-applied); 2) MBr fumigation, which served as the positive control; and 3) an untreated control. Plots within each block were 10.7 × 3.1 m in size. Treatments were arranged in a randomized complete block design with six blocks and eight trees per plot.

On 9 Nov. 2004, the plots were re-rotovated. Methyl bromide (67% methyl bromide, 33% chloropicrin) was applied (Hendrix and Dail, Inc., Tifton, GA) in a strip (3.1 m wide) under a tarp (455 kg·ha^{-1}) on 10 Nov. 2004. Soil moisture (10 to 15 cm depth) was adequate for fumigation such that when the soil was compressed in the hand, it formed a ball that was easily broken with little disturbance. Tarps were removed on 19 Nov. 2004 (9 d posttreatment).

Pre-fumigation *M. xenoplax* populations in soil were determined on 22 Jun. 2004 from four soil cores (2.5 cm in diameter × 30 cm deep) collected within each plot throughout the test site. The four soil cores were composited by plot within each block for a total of 18 samples. The nematodes were extracted from a 100-cm^3 soil subsample with a semiautomatic elutriator (Byrd et al., 1976) and centrifugal flotation (Jenkins, 1964) and counted.

All plots were planted to cv. Dixiland on Nemaguard rootstock at a tree spacing of 1.5 × 6.1 m in Mar. 2005. Each plot had eight trees, the outer two of which served as borders with the six center trees as the experimental unit. All border trees were planted first. Using shovels, the remaining trees were planted by treatment in the following order: MBr fumigation followed by the Ni and untreated control treatments. All shovels were dipped into an ≈20% NaOCl solution and rinsed with water between uses in each experimental unit.

All trees received annual applications of fertilizer (10N–10P–10K and 34N–0P–0K), insecticides, fungicides, and herbicides and pruned as recommended by the Georgia Cooperative Extension Service. All fertilizer, pesticide, and herbicide rates were according to the schedule outlined for non-bearing and bearing trees (Horton et al., 2011; Lockwood et al., 2005).

Nickel application. The source of Ni was $NiSO_4$•6 H_2O (99% A.C.S. reagent grade; Aldrich, Milwaukee, WI) at a concentration of 0.45 g·L^{-1}, an efficacious rate for correction of mouse-ear and Ni deficiency in pecan orchards (Wood et al., 2004, 2006). A nonionic surfactant (Freeway® at 2.5 mL·L^{-1}; Loveland Prod., Inc., Greeley, CO) was also added to the tank mix. Postplant Ni foliar applications were sprayed till runoff on 10 May, 15 Sept., and 17 Oct. 2005 and 21 June, 13 Sept., and 19 Oct. 2006 using a NorthStar spot sprayer (Northern Tool + Equipment, Burnsville, MN). Border trees allowed adjacent treatments to be spatially separated to prevent crosscontamination with the spray.

Field sampling. The preplant population density of *M. xenoplax* was determined on 2 Mar. 2005 (≈4 months after preplant fumigation treatment) from six soil cores collected from within each plot throughout the test site as described previously for the pre-fumigation sampling. *Mesocriconema xenoplax* postplant population density was determined on 9 June and 2 Dec. 2005; 29 Mar., 7 June, and 15 Nov. 2006; 9 Mar., 5 June, and 12 Dec. 2007; 27 Mar., 10 June, and 9 Dec. 2008; 12 Mar. and 8 June 2009; and 13 Jan., 23 Mar., 9 June, and 3 Dec. 2010 from one soil core collected within the drip line of each of six test trees of each experimental unit. The six soil cores were composited and nematodes were extracted from a 100-cm^3 subsample as described previously.

Trunk diameters were measured 20 cm above the soil surface on 24 Feb. 2006, 28 Feb. 2007, 12 Feb. 2008, 29 Jan. 2009, 1 Feb. 2010, and 21 Jan. 2011. Tree mortality as a result of bacterial canker infection was recorded on 24 May 2006, 2 May 2007, 22 May 2008, 14 May 2009, 3 June 2010, and 19 Apr. 2011 to monitor PTSL in the site.

Nematode data were transformed to $\log_{10}(x+1)$ and subjected to analysis of variance (ANOVA) using the general linear model procedure of SAS (SAS Institute, Cary, NC). Actual numerical nematode data were used for table presentation. ANOVA was also performed to determine treatment effect on trunk diameter. Nematode population and trunk diameter means were compared according to Fisher's protected least significant difference test following a significant *F* test. The proportion of peach tree survival within each experimental unit for the Ni, preplant MBr soil fumigation, and untreated control treatments was analyzed for each sampling date with ANOVA. Only significant differences ($P \leq 0.05$) are discussed unless stated otherwise.

Results and Discussion

The mean population density of *M. xenoplax* in June 2004 before MBr application did not differ among the three treatments (untreated control = 33 *M. xenoplax*/100 cm^3 soil; Ni = 38 M. *xenoplax*/100 cm^3 soil; and MBr = 70 *M. xenoplax*/100 cm^3 soil), which indicated that the ring nematode was uniformly present throughout the test site. In Mar. 2005, the nematode population density after establishment of the MBr fumigation plots, but before replanting peach trees, was greatest ($P \leq 0.05$) in Ni (33 *M. xenoplax*/100 cm^3 soil) and untreated control (35 *M. xenoplax*/100 cm^3 soil) and lowest in the MBr-fumigated (0 *M. xenoplax*/100 cm^3 soil) plots. These results indicate that MBr fumigation was effective in suppressing ring nematode populations to undetectable levels before orchard establishment. Three months (Jun. 2005) after orchard establishment, nematode populations in untreated control and Ni plots were similar and remained greater than in MBr-fumigated plots until Mar. 2007 (Table 1). At this date, 24 months after orchard establishment, *M. xenoplax* was first detected in the MBr-fumigated plots, indicating that the nematode had reinfested and begun to reproduce in the nematicide-treated plots. These results confirmed the prolonged (28 months) beneficial effect of the fumigant nematicide in suppressing the nematode soil densities and the importance of preplant fumigation as a key component of the 10-point Management

Table 1. Populations of *Mesocriconema xenoplax* on 'Nemaguard' peach (*Prunus persica* cv. Dixiland) as influenced by foliar applications of Ni and preplant fumigation with methyl bromide in field plots on a PTSL site in Byron, GA.[z]

	No. *M. xenoplax*/100 cm^3 soil										
	2005		2006			2007			2008		
Treatment	9 June	2 Dec.	29 Mar.	7 June	15 Nov.	9 Mar.	5 June	12 Dec.	27 Mar.	10 June	9 Dec.
Untreated control	75 a[y]	70 a[y]	113 a[y]	207 a[y]	123 a[y]	186 a[y]	225 a	978 ab[x]	501 b[y]	301 b[y]	36 b[y]
Ni[w]	83 a	53 a	158 a	155 a	110 a	123 a	218 a	99 b	34 c	120 b	15 b
MBr[v]	0 b	0 b	0 b	0 b	0 b	18 b	233 a	1533 a	1993 a	1583 a	688 a

[z]Data are means of six replications per treatment, except on 7 June and 15 Nov. 2006 and 9 Mar.; 5 June and 12 Dec. 2007; amd 27 Mar., 10 June, and 9 Dec. 2008, which had five replicates for the untreated control treatment and on 5 June and 12 Dec. 2007 and 27 Mar., 10 Jun., and 9 Dec. 2008, which had four replicates for the Ni treatment.

[y]Means within a column followed by the same letter are NS ($P \leq 0.05$), Fisher's protected LSD. Nematode data were transformed to [$\log_{10}(x+1)$] for analysis and were back-transformed for presentation in this table.

[x]Means within a column followed by the same letter are NS ($P = 0.10$), Fisher's protected LSD. Nematode data were transformed to [$\log_{10}(x+1)$] for analysis and were back-transformed for presentation in this table.

[w]Ni = Nickel ($NiSO_4$•6 H_2O) was foliar applied until runoff at a concentration of 0.45 g·L^{-1} along with a nonionic surfactant (2.5 mL·L^{-1}) on 10 May, 15 Sept., and 17 Oct. 2005 and 21 June, 13 Sept., and 19 Oct. 2006.

MBr = Methyl bromide (67% methyl bromide, 33% chloropicrin) was applied at a rate of 455 kg·ha^{-1}; application date was 10 Nov. 2004. Tarps were removed on 19 Nov. 2004.

PTSL = peach tree short life; LSD = least significant difference.

Program of PTSL (Brittain and Miller, 1978). However, 3 months later (June 2007), the nematode population density increased greatly in the fumigated plots and did not differ from those in the Ni and untreated control plots. It is not unusual for the effect of preplant fumigation to lessen over time (2 years) (Nyczepir and Bertrand, 2000; Sharpe et al., 1989; Zehr and Golden 1986). The increase in the nematode population density persisted in MBr-fumigated plots and in time (Dec. 2007 to Dec. 2008) became more accentuated ($P \leq 0.05$) than in the other two treatments (Table 1). This trend lasted until June 2009 (51 months after orchard establishment, data not presented) and could have been a consequence of the larger root system rich in feeder roots available to the nematode in MBr-fumigated soil than that of the trees growing in untreated soil or receiving Ni foliar applications. A suppression of nematode antagonists by the nematicide also might have favored the increase of *M. xenoplax* in the MBr-fumigated plots. In contrast, trees growing in Ni and untreated control soil were weaker, thus likely providing a reduced food source, which is known to limit nematode reproduction (Nyczepir et al., 1987). From Jan. 2010 (58 months after orchard establishment) to Dec. 2010 (69 months after orchard establishment), the *M. xenoplax* population density in MBr-treated plots did not differ from the untreated control plots and was only greater than the Ni-treated plots on the last two sampling dates (data not presented). These results indicate stabilization in ring nematode population density as related to treatment effect over time.

Tree growth in MBr-fumigated plots resulted in larger ($P \leq 0.01$) trunk diameter of these trees (34 mm) compared with those grown in the untreated (26 mm) or Ni (26 mm) -treated plots. However, this improvement in tree growth was evident only 11 months after orchard establishment and ceased in time. In subsequent sampling dates (Jan. 2009, Feb. 2010, and Jan. 2011), tree growth was generally lower in MBr-fumigated plots than the Ni or untreated control plots, although differences were not significant ($P \geq 0.11$). One explanation for this turnaround in treatment effect on tree growth might be related to the number of test trees remaining alive within a respective treatment. More test trees remaining alive in a particular treatment over time would result in increased competition for space among the trees, therefore resulting in a slower growing tree (Table 2). In contrast, fewer living test trees in the plots would have avoided competition, therefore attaining a larger size. Another explanation for tree growth suppression in MBr-fumigated plots on these sampling dates may be the result of the rapid increase in ring nematode population density as discussed previously.

Peach trees developed typical PTSL symptoms and died during the experiment. In May 2006 (14 months after orchard establishment), more ($P \leq 0.01$) trees in the Ni and untreated control plots developed PTSL symptoms and died than in MBr-fumigated plots (Table 2). No difference in PTSL tree mortality was detected between the Ni and untreated control treatment plots on this sampling date. Beginning in May 2007 (26 months after orchard establishment) until Apr. 2011 (73 months after orchard establishment), PTSL tree mortality was greatest ($P \leq 0.01$) in Ni-treated plots, intermediate in untreated control plots, and lowest in MBr-fumigated plots, except in May 2009, when no difference in tree mortality was detected between the Ni and untreated control plots. The beneficial effect of preplant MBr fumigation persisted during this 6-year experiment as evident by enhancing early tree growth and survival. Furthermore, tree mortality (35% or greater) in plots treated with Ni and the untreated control occurred 14 months after orchard establishment (May 2006). This greater ($P \leq 0.01$) early tree mortality (36% to 52%) compared with the MBr (0%) -fumigated plots could be attributed to a higher initial *M. xenoplax* population at the time of orchard establishment in Mar. 2005, as discussed previously.

This orchard site has a known history of PTSL and has been replanted to peach four times over the past 40 years. Additionally, PTSL development is dependent on cumulative population exposure of trees to *M. xenoplax* (Nyczepir et al., 2004). In our study, the ring nematode population density in Ni and untreated control plots from 3 (June 2005) to 24 (Mar. 2007) months after orchard establishment was higher than in MBr-fumigated plots (Table 1); therefore, exposing trees in Ni and untreated control plots to greater nematode-feeding induced stress during a sensitive biological time period, which resulted in elevated PTSL tree death. Furthermore, something that was interesting and unexpected was that PTSL tree mortality was greater ($P \leq 0.01$) in Ni-treated plots on all sampling dates, except in May 2006 and 2009, as compared with the untreated control and MBr-fumigated plots (Table 2). Visual observations did not indicate that Ni treatments caused visual phytotoxicity symptoms. Peach is a transition metal-sensitive species with foliar sprays of metals such as zinc causing phytotoxicity to foliage and even potential defoliation (Johnson, 2008). There is the possibility that the exposure to Ni at the concentration used in the current study may have caused subtle adverse effects on peach tree metabolism/physiology and/or disease resistance processes.

Ni foliar applications did not induce nematode resistance within the peach trees, resulting in a subsequent suppressing of *M. xenoplax* soil population density. The mechanisms that mediate the observed increase in PTSL mortality by the addition of Ni were not addressed in this study. However, one possible explanation may be the result of the increase in *P. syringae* pv. *syringae* populations on these Ni-treated peach trees as suggested by studies conducted by Spain (2003). An accelerated increase in *P. syringae* pv. *syringae* population under suitable environmental conditions and nematode-stressed trees could likely result in greater incidence in PTSL tree mortality.

In summary, the present study indicates that postplant foliar application of Ni to young peach trees did not suppress ring nematode populations in soil as reported for another plant-parasitic nematode (Khan and Salam, 1990). In the previous study, Ni ($NiCl_2 \cdot 6H_2O$) was inhibitory to *M. javanica* egg hatch and caused greater than 94% mortality of infective-stage juveniles (J2) at a concentration of 971 mg·L^{-1} under laboratory conditions. In pot studies, the number of galls was reduced on pigeon pea roots after Ni was applied as a soil drench. The different molecular salts and application method for Ni used in the current study (i.e., $NiSO_4 \cdot 6\ H_2O$ foliar spray) vs. the root-knot nematode study (i.e., $NiCl_2 \cdot 6H_2O$ soil drench) may explain the lack of ring nematode control. Furthermore, Ni did not appear to induce bacterial canker (*P. syringae* pv. *syringae*) resistance by phytoalexin production as reported for other foliar fungal (Reilly et al., 2005) and bacteria (Wang et al., 2000) diseases, because PTSL tree mortality was increased in Ni-treated plots. These results indicate that foliar-applied Ni, at the concentration used in the present study, altered tree metabolism/physiology (e.g., possibly favoring *P. syringae* pv. *syringae* growth) such that trees become more susceptible to PTSL mortality. The present study also indicates that growers should be careful when using micronutrient sprays containing Ni on newly planted peach trees growing on PTSL sites. Trees in the present study received relatively heavy exposure to Ni over the growing season; thus, reduced Ni exposure might produce different results. The

Table 2. Effect of foliar applications of Ni and preplant fumigation with methyl bromide on development of peach tree short life (PTSL) of 'Dixiland' trees on Nemaguard rootstock in field plots on a PTSL site in Byron, GA (n = 6).

	Cumulative PTSL mortality (%)					
Treatment	May 2006	May 2007	May 2008	May 2009	June 2010	Apr. 2011
Ni[z]	52 a[y]	75 a	77 a	81 a	83 a	87 a
Untreated control	36 a	53 b	56 b	63 a	63 b	63 b
MBr[x]	0 b	0 c	3 c	11 b	11 c	14 c

[z]Ni = Nickel ($NiSO_4 \cdot 6\ H_2O$) was foliar applied until runoff at a concentration of 0.45 g·L^{-1} along with a nonionic surfactant (2.5 mL·L^{-1}) on 10 May, 15 Sept., and 17 Oct. 2005 and 21 June, 13 Sept., and 19 Oct. 2006.

[y]Means within a column followed by the same letter are NS ($P \leq 0.01$), Fisher's protected least significant difference.

[x]MBr = Methyl bromide (67% methyl bromide, 33% chloropicrin) was applied at a rate of 455 kg·ha^{-1}; application date was 10 Nov. 2004. Tarps were removed on 19 Nov. 2004.

nature of the Ni-associated mechanisms remains unknown and merits further investigation within the context of the PTSL disease complex.

Literature Cited

Bai, C., C.C. Reilly, and B.W. Wood. 2006. Nickel deficiency disrupts metabolism of ureides, amino acids, and organic acids of young pecan foliage. Plant Physiol. 140:433–443.

Bai, C., C.C. Reilly, and B.W. Wood. 2007. Nickel deficiency affects nitrogenous forms and urease activity in spring xylem sap of pecan. J. Amer. Soc. Hort. Sci. 132:302–309.

Bai, C., B.W. Wood, and C.C. Reilly. 2008. Insights into the nutritional physiology of nickel. Acta Hort. 772:365–368.

Brittain, J.A. and R.W. Miller. 1978. Managing peach tree short life in the Southeast. Clemson Univ. Ext. Serv. Bul. 585.

Byrd, D.W., Jr., K.R. Barker, H. Ferris, C.J. Nusbaum, W.E. Griffin, R.H. Small, and C.A. Stone. 1976. Two semi-automatic elutriators for extracting nematodes and certain fungi from soil. J. Nematol. 8:206–212.

Cao, T., R.A. Duncan, M.V. McKenry, K.A. Shackel, T.M. DeJong, and B.C. Kirkpatrick. 2005. Interaction between nitrogen-fertilized peach trees and expression of *syrB*, a gene involved in syringomycin production in *Pseudomonas syringae* pv. *syringae*. Phytopathology 95:581–586.

Cao, T., M.V. McKenry, R.A. Duncan, T.M. DeJong, B.C. Kirkpatrick, and K.A. Shackel. 2006. Influence of ring nematode infestation and calcium, nitrogen, and indoleacetic acid applications on peach susceptibility to *Pseudomonas syringae* pv. *syringae*. Phytopathology 96:608–615.

Horton, D., P. Brannen, B. Bellinger, D. Lockwood, and D. Ritchie. 2011. 2011 Southeastern peach, nectarine, and plum pest management and culture guide. Univ. of Georgia Coop. Ext. Serv. Bul. #1171.

Jenkins, W.R. 1964. A rapid centrifugal-flotation technique for separating nematodes from soil. Plant Dis. Rpt. 48:692.

Johnson, R.S. 2008. Nutrient and water requirements of peach trees, p. 303–331. In: Layne, D.R. and D. Bassi (eds.). The Peach: Botany, production and uses. CABI, Wallingford, UK.

Khan, M.W. and M.A. Salam. 1990. Interactions of *Meloidogyne javanica, Fusarium udum* and *Rhizobium* on pigeon pea in the presence of nickel and cobalt as pollutants. Ann. Appl. Biol. 11:549–556.

Lockwood, D.W., M.E. Ferree, and S.C. Meyers. 2005. Nutrition, p. 75–84. In: Horton, D. and D. Johnson (eds.). Southeastern peach growers' handbook. University of Georgia College of Agriculture, Athens, GA.

Loof, P.A.A. and de Grisse, A. 1967. Re-establishment of the genus *Criconemoides* Taylor, 1936 (Nematoda: Criconematidae). Mededelingen van de Rijksfac. der Landbouww. Gent 32: 466–475.

Loof, P.A.A. and de Grisse, A. 1989. Taxonomic and nomenclatorial observations on the genus *Criconemella* De Grisse & Loof, 1965 *sensu* Luc & Raski, 1981 (Criconematidae). Mededelingen Fac. Landbouww. Rijksuniv. Gent 54:53–74.

Miller, R.W. 1994. Estimated peach tree losses 1980 to 1992 in South Carolina: Causes and economic impact, p. 121–127. In: Nyczepir, A.P., P.F. Bertrand, and T.G. Beckman (eds.). Proc. 6th Stone Fruit Decline Workshop, 26–28 Oct. 1992, Ft. Valley, GA.

Nyczepir, A.P. and P.F. Bertrand. 2000. Preplanting bahiagrass or wheat compared for controlling *Mesocriconema xenoplax* and short life in a young peach orchard. Plant Dis. 84:789–793.

Nyczepir, A.P., W.R. Okie, and T.G. Beckman. 2004. Creating a short life site for *Prunus* rootstock evaluation on land with no innate *Mesocriconema xenoplax* population. HortScience 39:124–126.

Nyczepir, A.P., C.C. Reilly, and W.R. Okie. 1987. Effect of initial population density of *Criconemella xenoplax* on reducing sugars, free amino acids, and survival of peach seedlings over time. J. Nematol. 19:296–303.

Nyczepir, A.P., B.W. Wood, and C.C. Reilly. 2006. Association of *Meloidogyne partityla* with nickel deficiency and mouse-ear of pecan. HortScience 41:402–404.

Nyczepir, A.P., E.I. Zehr, S.A. Lewis, and D.C. Harshman. 1983. Short life of peach trees induced by *Criconemella xenoplax*. Plant Dis. 67:507–508.

Okie, W.R., G.L. Reighard, T.G. Beckman, A.P. Nyczepir, C.C. Reilly, E.I. Zehr, and W.C. Newall, Jr. 1994. Field-screening *Prunus* for longevity in the southeastern United States. HortScience 29:673–677.

Olien, W.C., C.J. Graham, M.E. Hardin, and W.C. Bridges, Jr. 1995. Peach rootstock differences in ring nematode tolerance related to effects on tree dry weight, carbohydrate and prunasin contents. Physiol. Plant. 94:117–123.

Raski, D.J. 1952. On the morphology of *Criconemoides* Taylor, 1936, with descriptions of six new species (Nematoda: Criconematidae). Proc. Helminthol. Soc. of Wash. 19:85–99.

Reilly, C., M. Crawford, and J. Buck. 2005. Nickel suppresses daylily rust, *Puccinia hemerocallidis* on susceptible daylilies, *Hemerocallis* in greenhouse and field trials. Phytopathology 95:S88.

Sayler, R.J. and B.C. Kirkpatrick. 2003. The effect of copper sprays and fertilization on bacterial canker in French prune. Can. J. Plant Pathol. 25:406–410.

Sharpe, R.R., C.C. Reilly, A.P. Nyczepir, and W.R. Okie. 1989. Establishment of peach in a replant site as affected by soil fumigation, rootstock, and pruning date. Plant Dis. 73:412–415.

Spain, A. 2003. Implications of microbial heavy metal tolerance in the environment. Rev. in Undergraduate Res. 2:1–6.

Wang, H., F. Zeng, and J. Kang. 2000. Resistance to bacterial blight induced by nickel nitrate in rice seedlings. J. Hunan Agr. Univ. 26:119–121.

Wimalajeewa, D.L.S., R. Cahill, G. Hepworth, H.G. Schneider, and J.W. Washbourne. 1991. Chemical control of bacterial canker (*Pseudomonas syringae* pv. *syringae*) of apricot and cherry in Victoria. Aust. J. Exp. Agr. 31:705–708.

Wood, B.W., R. Chaney, and M. Crawford. 2006. Correcting micronutrient deficiency using metal hyper accumulators: *Alyssum* biomass as a natural product for nickel deficiency correction. HortScience 41:1231–1234.

Wood, B.W. and C.C. Reilly. 2007. Nickel and plant disease, p. 215–231. In: Datnoff, L.E., W.H. Elmer, and D.M. Huber (eds.). Mineral nutrition and plant disease. APS Press, St. Paul, MN.

Wood, B.W., C.C. Reilly, and A.P. Nyczepir. 2004. Mouse-ear of pecan: A nickel deficiency. Hort Science 39:1238–1242.

Zehr, E.I. and J.K. Golden. 1986. Strip and broadcast treatments of dichloropropene compared for controlling *Criconemella xenoplax* and short life in a peach orchard. Plant Dis. 70:1064–1066.