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

Andrew P. Nyczepir* and Bruce W. Wood
U.S. Department of Agriculture, Agricultural Research Service, Southeastern Fruit and Tree Nut Research Laboratory, 21 Dunbar Road, Byron, GA 31008

Abstract. Peach tree short life (PTSL) is associated with the presence of ring nematode, Mesocricotome 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, Mesocricotome 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). Mesocricotome 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 uninested 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 bio-chemicals 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 (Sayer 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 fungicide-resistant bacteria (Wang et al., 2000), nematodes (Khan and Salam, 1990). It is possible that these essential micronutrients induce resistance through in planta phytoalexin production. Daylily rust (Puccinia hemerocallis Thüm) was suppressed for up to 15 d with a single aqueous foliar application of NiSO₄ (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 Kleyhans) are correctable by timely foliar Ni application (Nyczepir et al., 2006; Wood et al., 2004). Plant Ni nutritional physiology can affect many physiological processes (Sabar et al., 2005) including 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
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 at a rate of 455 kg ha⁻¹ of soil subsample with a semiautomatic fumigation applicator (Jenkins, 1964) and counted.

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 season. Each of the four soil cores were composited by plot within each block for a total of 18 samples. The nematodes were extracted from a 100-cm³ soil subsample with a semiautomatic elutriator (Byrd et al., 1976) and centrifugal flotation (Jenkins, 1964) and counted.

All plots were planted to cv. Dixiland orange ('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.)*

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 NiSO₄;H₂O (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 through 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 trees of each experimental unit. The six soil cores were composited and nematodes were extracted from a 100-cm³ 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 log₁₀ (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 using ANOVA. Only significant differences (P ≤ 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 cm³ soil; Ni = 38 M. xenoplax/100 cm³ soil; and MBr = 70 M. xenoplax/100 cm³ 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 ≤ 0.05) in Ni (33 M. xenoplax/100 cm³ soil) and untreated control (35 M. xenoplax/100 cm³ soil) and lowest in the MBr-fumigated (0 M. xenoplax/100 cm³ 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.*

| Treatment | 2005 | 2006 | 2007 | 2008 |
|-----------|------|------|------|------|
|           | 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 a¹ | 70 a¹ | 113 a¹ | 207 a¹ | 123 a¹ | 186 a¹ | 225 a | 978 ab¹ | 501 b¹ | 301 b¹ | 36 b¹ |
| Ni² | 83 a | 53 a | 158 a | 155 a | 110 a | 123 a | 218 a | 99 b | 34 c | 120 b | 15 b |
| MBr³ | 0 b | 0 b | 0 b | 0 b | 18 b | 233 a | 1533 a | 1993 a | 1583 a | 688 a |

¹Data are means of six replications per treatment, except on 7 June and 15 Nov. 2006 and 9 Mar.; 5 June and 12 Dec. 2007 and 27 Mar., 10 June, and 9 Dec. 2008, which had five replications for the untreated control treatment and on 5 June and 12 Dec. 2007 and 27 Mar., 10 June, and 9 Dec. 2008, which had four replications for the Ni treatment.

²Means within a column followed by the same letter are NS (P > 0.05). Fisher’s protected LSD. Nematode data were transformed to [log₁₀(x+1)] for analysis and were back-transformed for presentation in this table.

³Means within a column followed by the same letter are NS (P > 0.10). Fisher’s protected LSD. Nematode data were transformed to [log₁₀(x+1)] for analysis and were back-transformed for presentation in this table.

⁴Ni = Nickel (NiSO₄;6 H₂O) 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.

⁵MBr = Methyl bromide (67% methyl bromide, 33% chloropicrin) was applied at a rate of 455 kg ha⁻¹; application date was 10 Nov. 2004. Trees were removed on 19 Nov. 2004.

PTSL = peach tree short life; LSD = least significant difference.
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 ≤ 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 ≤ 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 density in soil as reported for another nematode species (Nyczepir et al., 2004). In our study, the ring nematode population density in Ni and untreated control plots was greater than in MBr-fumigated plots (Table 1); therefore, exposing the 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 ≤ 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 PTSL 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 tree, resulting in a subsequent suppression 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 population in 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\(_2\)·6H\(_2\)O) was inhibitory to M. javanica egg hatch and caused greater than 94% mortality of infective juveniles (I.J.) 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\(_4\)·6H\(_2\)O foliar spray vs. the root-knot nematode study (i.e., NiCl\(_2\)·6H\(_2\)O 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 fungals (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 growing peach trees on Ni-fertilized soil may be the cause of PTSL disease when using micronutrients 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

| Treatment | May 2006 | May 2007 | May 2008 | May 2009 | June 2010 | Apr. 2011 |
|-----------|---------|---------|---------|---------|-----------|----------|
| Ni\(^{-2}\) | 52\(^{a}\) | 75\(^{a}\) | 77\(^{a}\) | 81\(^{a}\) | 83\(^{a}\) | 87\(^{a}\) |
| Untreated control | 36\(^{a}\) | 53\(^{b}\) | 56\(^{b}\) | 63\(^{b}\) | 63\(^{b}\) | 63\(^{b}\) |
| MBr\(^{-2}\) | 0\(^{b}\) | 0\(^{c}\) | 3\(^{c}\) | 11\(^{c}\) | 11\(^{c}\) | 14\(^{c}\) |

\(^{-2}\)Ni = Nickel (NiSO\(_4\)·6H\(_2\)O) was foliar applied until runoff at a concentration of 0.45 g L\(^{-1}\) alongside a nontoxic surfactant (2.5 mL L\(^{-1}\)) on 10 May, 15 Sept., and 17 Oct. 2005 and 21 June, 13 Sept., and 19 Oct. 2006.

\(^{a}\)Means within a column followed by the same letter are NS (P > 0.05). Fisher’s protected least significant difference.

\(^{b}\)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.

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

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