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Interaction of *Pratylenchus penetrans* and *Rhizoctonia fragariae* in Strawberry Black Root Rot

J. A. LaMondia[1]

Abstract: A split-root technique was used to examine the interaction between *Pratylenchus penetrans* and the cortical root-rotting pathogen *Rhizoctonia fragariae* in strawberry black root rot. Plants inoculated with both pathogens on the same half of a split-root crown had greater levels of root rot than plants inoculated separately or with either pathogen alone. Isolation of *R. fragariae* from field-grown roots differed with root type and time of sampling. Fungal infection of structural roots was low until fruiting, whereas perennial root colonization was high. Isolation of *R. fragariae* from feeder roots was variable, but was greater from feeder roots on perennial than from structural roots. Isolation of the fungus was greater from structural roots with nematode lesions than from non-symptomatic roots. *Rhizoctonia fragariae* was a common resident on the sloughed cortex of healthy perennial roots. From this source, the fungus may infect additional roots. The direct effects of lesion nematode feeding and movement are cortical cell damage and death. Indirect effects include discoloration of the endodermis and early polyderm formation. Perhaps weakened or dying cells caused directly or indirectly by *P. penetrans* are more susceptible to *R. fragariae,* leading to increased disease.

Key words: Black root rot, *Fragaria x ananassa,* interaction, lesion nematode, *Pratylenchus penetrans, Rhizoctonia fragariae,* strawberry.

Strawberry black root rot is a root cortical disease caused by the binucleate fungus *Rhizoctonia fragariae* Husain and McKeen (Martin, 1988; Wilhelm et al., 1972). The lesion nematode, *Pratylenchus penetrans* (Cobb) Filip & Schur.Stek., has been associated with increased severity of black root rot under field conditions (Goheen and Bailey, 1955; Goheen and Smith, 1956; LaMondia, 1994) and in controlled experiments (Chen and Rich, 1962; LaMondia and Martin, 1989). The mechanism by which *P. penetrans* affects the development of black root rot is unknown.

A number of nematode-fungal pathogen interactions have been described (Abawi and Chen, 1998). The mechanisms of nematode-fungus interactions in disease may differ with the particular nematode, the plant, the interacting pathogen(s), or other factors. The role of the interacting nematode in complex diseases may be to cause wounds that serve as infection courts (Brodie, 1984; Conroy et al., 1972; Mai et al., 1981), to alter the physiology or biochemistry of the plant leading to increased disease (Faulkner et al., 1970; Mai and Abawi, 1987; Webster, 1985), or to modify the expression of host plant resistance (Maheshwari et al., 1995; Mai and Abawi, 1987; Webster, 1985).

The objectives of this research were to (i) determine whether *P. penetrans* had local or systemic influence on *R. fragariae* infection and strawberry root rot and (ii) examine the extent of *R. fragariae* infection of morphologically different strawberry root types alone and in relation to lesion nematode infection.

Materials and Methods

The local and systemic effects of *P. penetrans* on strawberry black root rot by *R. fragariae* were investigated using a split-root system. Two 6.5-cm square pots (9 cm deep) each were notched 2.5 × 2.5 cm on one side and stapled together. Roots from 1-year-old 'Honeoye' strawberry crowns were evenly divided between the two pots, and the crown was positioned in the notch between pots. Five-cm lengths of duct tape were folded in half to avoid adhesive contact for the center 3 cm and attached to the inside of each pot to allow the roots access to the pots while isolating the crown from the soil. No adhesive was in contact with the crown. Roots were distributed in 250 cm^3 of pasteurized Merrimac sandy loam field soil (73.4% sand, 22.3% silt, 4.3% clay; pH 6.0) in each pot.

Twenty split-root plants were inoculated with pathogens to create five replicates of each of four treatments. Treatments consisted of: (i) *P. penetrans* and *R. fragariae* inoculated in separate halves of the same root system; (ii) *P. penetrans* and *R. fragariae* inoculated to the same half of the root system, and the second half left uninoculated; (iii) *P. penetrans* alone inoculated to only one half of the root system; and (iv) *R. fragariae* alone inoculated to only one half of the root system. The experiment was performed three times. Crowns were planted on 16 April 1999, 15 December 1999, and 5 May 2000. Inoculum of *P. penetrans* was extracted from carrot disk culture. A suspension of 4,000 individuals was added to four 2-cm-deep holes per treated pot. Uninoculated pots received water in four 2-cm-deep holes per pot. Inoculum of *R. fragariae* was prepared on autoclaved rye seeds (Martin, 1988). Anastomosis groups (AG) A, G, and I were introduced on one colonized rye seed per AG per plot. Uninoculated pots received three sterile (autoclaved) seeds per pot. In the first experiment, nematodes were inoculated on 2 May 1999 and *R. fragariae* inoculated on 12 May 1999. In subsequent experiments, both pathogens were inoculated on the same day on 19 January 2000 and 22 May

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[1] Plant Pathologist/Nematologist, Department of Plant Pathology and Ecology, The Connecticut Agricultural Experiment Station, Valley Laboratory, P. O. Box 248, Windsor, CT 06095.

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E-mail: James.LaMondia@po.state.ct.us

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2000. After 10 weeks in the greenhouse, roots of each half of the root system were separated, washed free of soil, and rated for disease. Two independent ratings were averaged. Shoot and root weights were recorded. *Rhizoctonia fragariae* was isolated from ten 0.5-cm sections of surface-sterilized roots. Roots were exposed to 0.5% NaOCl for 30 seconds and placed on acidified water agar for 48 hours. Nematodes were extracted from 2 g of root tissue placed in a flask containing 50 ml water and shaken for 7 days using a wrist-action shaker. Data were analyzed by analysis of variance and means separated by Fisher's LSD test.

The extent of *R. fragariae* infection of morphologically different strawberry roots was determined from an established planting at The Connecticut Agricultural Experiment Station Valley Laboratory research farm in Windsor, Connecticut (Merrimac fine loamy sand). Existing 3-year-old Honeoye strawberry crowns in field plots infested with lesion nematodes and the black root rot pathogen, *R. fragariae,* were sampled on 23 March, 8 April, 6 May, 28 May, and 11 June 1999 and on 30 May, 7 June, and 12 June 2000. Several crowns (4 to 6) were sampled on each date. Soil was removed, and entire root systems were washed free of soil and separated into four classes: black suberized perennial roots, new healthy structural roots, fine lateral (feeder) roots from perennial roots, and fine lateral roots from structural roots. Structural roots were further subdivided into roots with or without typical lesions caused by *P. penetrans. Rhizoctonia fragariae* was isolated from ten 0.5-cm sections of surface-sterilized roots in each class (exposed to 0.5% NaOCl for 30 seconds and then rinsed in sterile water) and placed on acidified water agar for 48 hours. Nematodes in roots of each type were extracted by shaker extraction over a 10-day period. Fungi were examined microscopically and hyphal tips transferred to PDA to confirm identity as *R. fragariae.* The proportion of morphologically different strawberry root segments infected with *R. fragariae* from before and after fruiting were subjected to Chi-square analysis. An additional statistical test was based on non-overlap of 95% confidence limits on the proportion of infected roots (Steel and Torrie, 1980).

Cross sections of morphologically different strawberry roots were made, mounted in water, and examined by microscope (×100–×1,000) to observe healthy strawberry roots and the damage associated with *P. penetrans* and *R. fragariae.*

Results

Data from the three split-root strawberry experiments were similar and were combined for analyses (Table 1). Plants inoculated with *P. penetrans* and *R. fragariae* on the same half of a split-root strawberry crown had greater levels (P = 0.0001) of black root rot than plants inoculated with *P. penetrans* and *R. fragariae* on different halves of the root system or plants inoculated with *P. penetrans* or *R. fragariae* alone. Lesion nematodes were recovered from all inoculated pots in similar numbers. The level of *R. fragariae* isolation from strawberry roots was similar from all pots inoculated with *R. fragariae* (37% to 49% recovery) and least from pots inoculated with *P. penetrans* alone (15% recovery). Root and shoot weights were not different between treatments.

Table 1. Effects of *Pratylenchus penetrans* (Pp) and *Rhizoctonia fragariae* (Rf) inoculated in different combinations in a split-root strawberry system on black root rot.

Pot 1	Pot 2[a]	Root rot (%)	Pp/2 g root	Root wt g	Shoot wt g	Rf infection[b]
Pp	Rf	11.3 a[c]	1,162 a	10.8	26.9	3.9 a
—	Pp + Rf	21.1 b	1,852 a	8.4	24.9	4.9 a
—	Pp	8.8 a	1,277 a	9.7	29.0	1.5 b
—	Rf	11.1 a	0 b	9.1	26.6	3.7 a
	P =	0.0001	0.001	ns	ns	0.001

[a] Pathogens inoculated alone or in combination in the same or different halves (pots) of a split-root strawberry crown.
[b] Number of ten 0.5-cm root segments from which *R. fragariae* was recovered on water agar.
[c] Means within columns followed by the same letter are not significantly different (LSD).
ns = not significant.

Isolation of *R. fragariae* from field-grown morphologically distinct strawberry roots differed with root type and time of sampling in naturally infested soil (Tables 2 and 3). In both 1999 and 2000, the proportions of morphologically distinct strawberry root segments infected with *R. fragariae* were different (Chi-square, P = 0.001). Isolation of *R. fragariae* from structural roots was consistently low until fruiting. Fungal colonization of perennial roots was uniformly high. Isolation of *R. fragariae* from feeder roots was variable but generally greater from feeder roots attached to perennial roots than to structural roots. Isolation of *R. fragariae* from

Table 2. Proportion of morphologically different strawberry root segments infected with *Rhizoctonia fragariae* at each sample date, 1999.

	Root types				
Sample date	Structural[a]	Perennial	Feeder (S)	Feeder (P)	Nematode lesions
23 March	0.00[b]	0.65*[c]	0.00	0.10*	0.33*
8 April	0.00	0.21*	0.01	0.23*	0.17*
6 May	0.00	0.70*	0.26*	0.16*	0.00
28 May	0.31	0.75*	0.05	0.13	0.73*
11 June	0.58	0.80*	0.38	0.28	nd

[a] Root types: Structural roots with a well-developed cortex; black suberized perennial roots; and fine feeder roots attached to white structural roots or black suberized perennial roots; and structural roots with typical sunken elliptical lesions infected with *Pratylenchus penetrans.*
[b] Proportion of forty 0.5-cm root segments from which *R. fragariae* was recovered on water agar.
[c] Means followed by asterisks are different from structural roots sampled on the same date based on non-overlapping 95% confidence intervals. The proportion of morphologically distinct strawberry root segments infected with *R. fragariae* was greater after fruiting (May 28 and June 11) (Chi-square, *P* = 0.001).
nd = no data.

TABLE 3. Proportion of morphologically different strawberry root segments infected with *Rhizoctonia fragariae* at each sample date, 2000.

	Root types				
Sample date	Structural[a]	Perennial	Feeder (S)	Feeder (P)	Nematode lesions
30 May	0.05[b]	0.50*[c]	0.00	0.00	0.15
7 June	0.05	0.75*	0.10	0.43*	0.50*
12 June	0.45	0.60	0.18	0.50	0.70*

[a] Root types: structural roots with a well-developed cortex; black suberized perennial roots; and fine feeder roots attached to white structural roots or black suberized perennial roots; and structural roots with typical sunken elliptical lesions infected with *Pratylenchus penetrans*.

[b] Proportion of twenty 0.5-cm root segments from which *R. fragariae* was recovered on water agar.

[c] Means followed by asterisks are different from structural roots sampled on the same date based on non-overlap of 95% confidence intervals. Differences in proportion of morphologically different strawberry root segments infected with *R. fragariae* were determined by Chi-square ($P = 0.001$).

structural roots with typical lesion nematode symptoms was greater than from non-symptomatic structural roots at three of four sampling dates in 1999 and two of three sampling dates in 2000. Fruit production on Honeoye crowns typically occurs in late May, and ripe fruit are harvested in June in Windsor, Connecticut. Isolation of *R. fragariae* from structural roots before (23 March to 6 May) and after (28 May and 11 June) fruiting in 1999 was determined to be different by Chi-square (P = 0.001).

Observations were made of healthy strawberry roots and the damage associated with *P. penetrans* and *R. fragariae* in cross sections of morphologically different strawberry roots. Healthy strawberry structural roots had a well-defined cortex (Fig. 1A) that became disrupted and detached as a result of secondary growth (Fig. 1B). Perennial roots consisted in large measure of woody xylem, polyderm, and phellogen (Fig. 1C). Lesion nematode damage to strawberry structural roots consisted of individual injured cells through which lesion nematodes had moved and adjacent discolored cells (Fig. 2A). Discoloration of the endodermis was observed in proximity to nematode infection without visible damage to adjacent cells (Fig. 2B). Areas of *P. penetrans* aggregation in the cortex were distinguished by a reddish-brown discoloration and eventually by a distinct sunken lesion resulting from cell necrosis. Fungal hyphae that appeared to be *R. fragariae* were observed in typical lesions resulting from *P. penetrans* infection (Fig. 2C). Infection of primary feeder roots by *R. fragariae* resulted in complete colonization as well as collapse and loss of the root tissue (Fig. 3A). Infection of structural roots generally girdled the root and resulted in a black rotted cortex from the epidermis in toward the stele, leaving a diagnostic healthy white or lighter-colored stele in structural roots (Fig. 3B). Eventually, the entire root, including the stele, became infected and collapsed. Often, roots exhibited a range of symptoms of cortical rot with a viable stele progressing toward a complete collapse and rat-tail progression of disease over a 3 to 4-cm length.

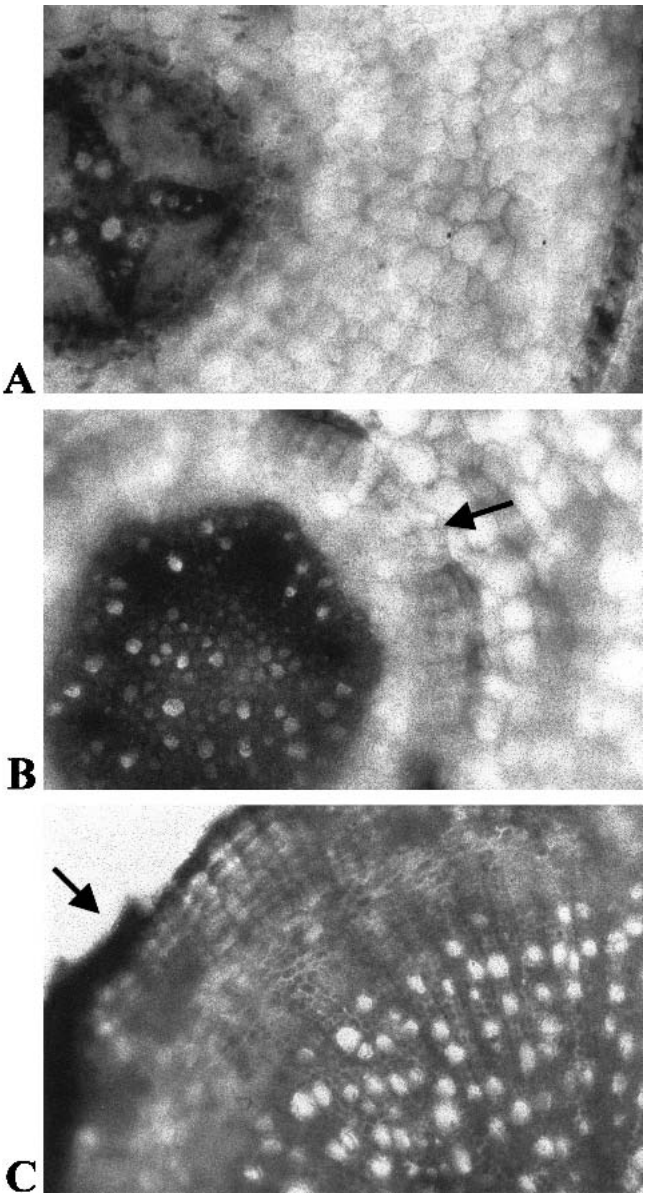

FIG. 1. Healthy strawberry roots. A) First-year strawberry structural roots had a well-defined cortex. B) The root cortex became disrupted and detached by polyderm and phellogen formation (arrow) as a result of secondary growth. C) Perennial roots consisted in large measure of woody xylem, polyderm, and phellogen (alternating layers of suberized and unsuberized cells). The remains of the cortical cells are retained on the exterior of the perennial roots as an amorphous black layer (arrow).

DISCUSSION

Black root rot is a debilitating disease of strawberry that causes a decline in perennial strawberry production over a number of years (Maas, 1984). The binucleate *R. fragariae* has been variously described as a pathogen of strawberry roots (Husain and McKeen, 1963; LaMondia and Martin, 1989; Martin, 1988) and a root inhabitant with some mycorrhizal properties (Ribeiro

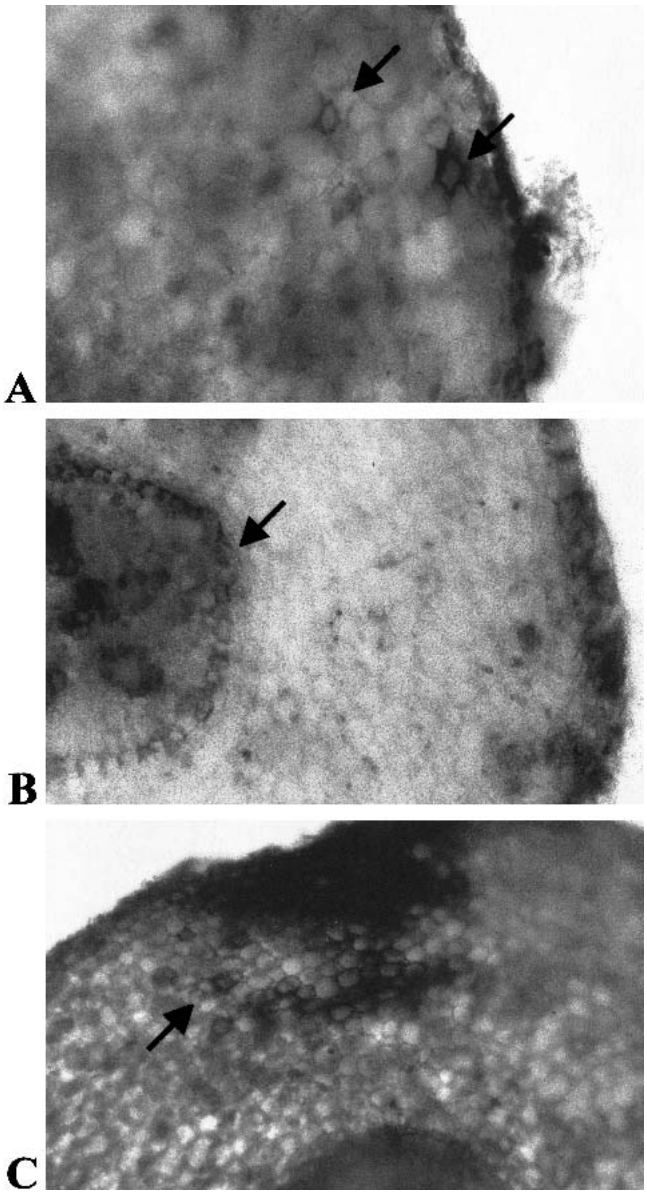

FIG. 2. Lesion nematode damage to strawberry structural roots. A) Individual cells through which lesion nematodes had moved became necrotic, and cells adjacent to these dead cells were discolored (arrows). B) Discoloration of the endodermis was observed in proximity to nematode infection without adjacent cell damage (arrow). C) Areas of *Pratylenchus penetrans* aggregation in the cortex were distinguished by a reddish-brown discoloration and eventually by a distinct sunken lesion resulting from cell necrosis (arrow). Fungal hyphae that appeared to be *Rhizoctonia fragariae* were observed in typical lesions resulting from *P. penetrans* infection.

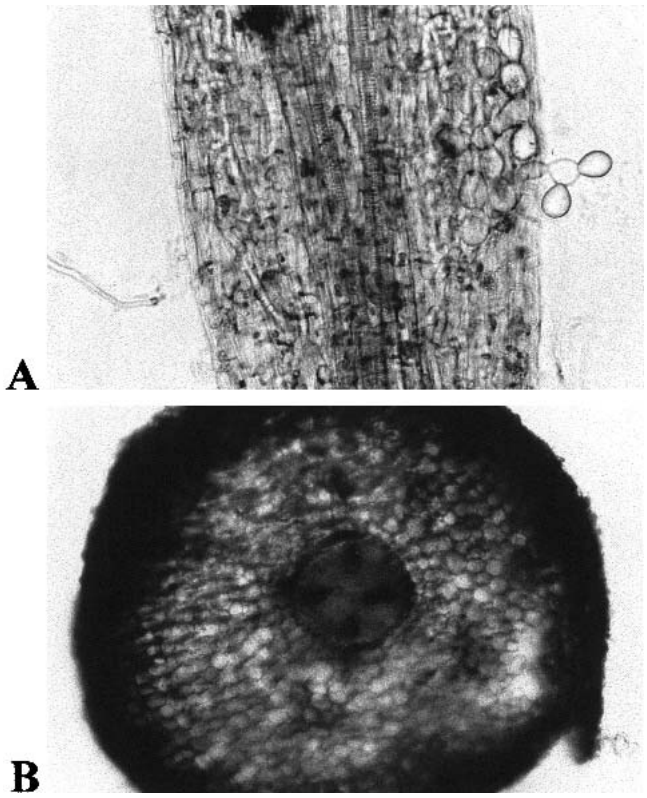

FIG. 3. A) *Rhizoctonia fragariae* infection of primary feeder roots resulted in complete colonization, and a collapse and loss of the root tissue. B) Infection of structural roots generally girdled the root and resulted in cell death from the epidermis in toward the stele.

and Black, 1971). Anastomosis groups differ in pathogenicity and temperature requirements, and isolates within anastomosis groups ranged from virulent to nonpathogenic (Martin, 1988).

First-year strawberry structural roots have a well-defined cortex that becomes disrupted and detached as a result of secondary growth. Polyderm and phellogen formation isolates and ruptures the cortex, which is sloughed off with the endodermis and epidermis as secondary growth occurs. Resulting perennial roots consist in large measure of woody xylem, polyderm, and phellogen (alternating layers of suberized and unsuberized cells) (Esau, 1977). The remains of the cortical cells are retained on the exterior of the perennial roots as an amorphous black layer.

Rhizoctonia fragariae infection and isolation from structural roots was increased in root segments with typical *P. penetrans* cortical lesions. Isolation of *R. fragariae* from structural roots increased during strawberry fruit production and the initiation of secondary growth associated with the transition to perennial roots. This is consistent with our previous observation that strawberry root mass declined in field soils at fruiting due to fungal infection and root rot (LaMondia, 2002). May and Pritts (1994) also noted that the strawberry root system was the only plant part to be reduced in biomass during fruiting. Townshend (1963) and Wilhem and Vertrees (1964) observed that an excessive development of polyderm due to *P. penetrans* infection was associated with the presence of fungal hyphae in the cortex of strawberry roots.

Pratylenchus penetrans primarily infects feeder and structural roots rather than perennial roots (LaMondia, 2002) and tends to be aggregated in the root cortex. Zunke (1990) described two types of feeding. Brief feeding episodes weakened cells without cell death, and extended feeding resulted in cell collapse and death.

We observed the death of individual cells through which lesion nematodes moved and the lighter discoloration of cells adjacent to these dead cells, perhaps as a result of feeding or diffusion of substances. Discoloration of the endodermis was observed in proximity to nematode infection without adjacent cell damage. Townshend (1963) and Zunke (1990) also reported that the presence of lesion nematodes was associated with discoloration of the endodermis despite the observation that lesion nematodes did not feed on the endodermis cells. Areas of *P. penetrans* aggregation in the cortex were distinguished by a reddish-brown discoloration and eventually by a distinct sunken lesion resulting from cell necrosis. Fungal hyphae that appeared to be *R. fragariae* were observed in typical lesions resulting from *P. penetrans* infection. Chen and Rich (1962) observed that fungi infected roots more readily at necrotic areas caused by *P. penetrans* than at healthy areas of the root.

We observed that *R. fragariae* was consistently associated with both healthy and diseased perennial roots, which is consistent with previous observations that *R. fragariae* infects the sloughed cortex (Wilhelm and Nelson, 1970; Wilhelm et al., 1972). This colonized tissue on the exterior of woody perennial roots may act as a source of infection for feeder and structural roots. In fact, we determined that feeder roots attached to perennial roots were more likely to be infected with *R. fragariae* than feeder roots attached to structural roots.

The role of *P. penetrans* in disease complexes may differ with the interacting root pathogen, the crop plant, or environmental conditions. The lesion nematode systematically increased Verticillium wilt in mint, caused by the vascular wilt pathogen *Verticillium dahliae,* in a split-root system (Faulkner et al., 1970). *Pratylenchus penetrans,* but not *P. crenatus,* interacted with *V. dahliae* to increase the severity of Verticillium wilt in potato, suggesting that the mechanism of interaction may be due to more than wounding. *Pratylenchus scribneri* increased wilt under high-temperature stress but not in a cool year (Riedel et al., 1985). No systemic effect of lesion nematodes on Verticillium wilt was evident in tomato in a split-root system. Instead, disease increase was attributed to an increase in local infection courts (Conroy et al., 1972).

The use of individual plants with split-root systems allowed the investigation of local vs. systemic pathogen interactions. *Pratylenchus penetrans* increased the severity of strawberry black root rot caused by *R. fragariae* in a local, rather than systemic manner in these experiments. Infection of one half of a split-root system with both pathogens exhibited greater levels of black root rot than when similar numbers of the same pathogens infected different halves of the same root system. As cortical root rot pathogens, *Rhizoctonia* species have not been associated with systemic disease. *Meloidogyne hapla* also has been shown to increase root rot by *R. solani.* The nematode increased the number of infection courts and the density of growth in infected tissues, presumably due to wounding and increased nutrition available to the fungal pathogen (Khan and Muller, 1982). Similarly, the local influence of *P. penetrans* on black root rot suggests that increased numbers of infection sites due to wounding or the predisposition of limited areas of the cortex to infection may be responsible for the increased disease in strawberry roots seen in these experiments.

It appears that *R. fragariae* commonly resides on the sloughed cortex of healthy perennial roots. In fact, our isolation of the pathogen from uninoculated crowns in split-root experiments has demonstrated that *R. fragariae* was present at low levels on commercially produced healthy strawberry crowns. From this source, the fungus may then infect structural or feeder roots, especially when the plant is under stress or roots are damaged. Lesion nematodes aggregate in the root cortex. Nematode feeding and movement directly result in cell damage and death. The indirect effects of lesion nematode infection are discoloration of the endodermis and early polyderm formation, followed by localized areas of secondary growth and cortical cell weakening or death. Weakened or dying cells resulting from direct or indirect effects of *P. penetrans* are more susceptible to *R. fragariae,* thereby increasing infection and cortical root rot.

Literature Cited

Abawi, G. S., and J. Chen. 1998. Concomitant pathogen and pest interactions. Pp. 135–158 *in* K. R. Barker, G. A. Pederson, and G. L. Windham, eds. Plant and nematode interactions, agronomy monograph no. 36, Madison, WI: American Society of Agronomy.

Brodie, B. B. 1984. Potato nematodes. Pp. 167–212 *in* W. R. Nickle, ed. Plant and insect nematodes. New York: Marcel Dekker, Inc.

Chen, T. A., and A. E. Rich. 1962. The role of *Pratylenchus penetrans* in the development of strawberry black root rot. Plant Disease Reporter 46:839–843.

Conroy, J. J., R. J. Green, Jr., and J. M. Ferris. 1972. Interaction of *Verticillium albo-atrum* and the root lesion nematode, *Pratylenchus penetrans,* in tomato roots at controlled inoculum densities. Phytopathology 62:362–366.

Esau, K. 1977. Anatomy of seed plants, 2nd ed. New York: John Wiley & Sons, Inc.

Faulkner, L. R., W. J. Bolander, and C. B. Skotland. 1970. Interaction of *Verticillium dahliae* and *Pratylenchus minyus* in Verticillium wilt of peppermint: Influence of the nematode as determined by a double root technique. Phytopathology 60:100–103.

Goheen, A. C., and J. S. Bailey. 1955. Meadow nematodes in strawberry plantings in Massachusetts. Plant Disease Reporter 39:879–880.

Goheen, A. C., and J. B. Smith. 1956. Effects of inoculation of strawberry roots with meadow nematodes, *Pratylenchus penetrans.* Plant Disease Reporter 40:146–149.

Husain, S. S., and W. E. McKeen. 1963. Interactions between strawberry roots and *Rhizoctonia fragariae.* Phytopathology 53:541–545.

Khan, M. W., and J. Muller. 1982. Interaction between *Rhizoctonia solani* and *Meloidogyne hapla* on radish in gnotobiotic culture. The Libyan Journal of Agriculture 11:133–140.

LaMondia, J. A. 1994. The association of lesion nematodes with black root rot in strawberry fields. NE-SARE Small Fruits Newsletter 4:10–11.

LaMondia, J. A. 2002. Seasonal populations of lesion and root-knot nematodes in strawberry roots. Journal of Nematology 34:409–413.

LaMondia, J. A., and S. B. Martin. 1989. The influence of *Pratylenchus penetrans* and temperature on black root rot of strawberry by binucleate Rhizoctonia spp. Plant Disease 73:107–110.

Maas, J. L. 1984. Compendium of strawberry diseases. St. Paul, MN: The American Phytopathological Society.

Maheshwari, T. U., S. B. Sharma, D. D. R. Reddy, and M. P. Haware. 1995. Co-infection of wilt-resistant chickpeas by *Fusarium oxysporum* f. sp. *ciceri* and *Meloidogyne javanica*. Journal of Nematology 27:649–653.

Mai, W. F., and G. S. Abawi. 1987. Interactions among root-knot nematodes and Fusarium wilt fungi on host plants. Annual Review of Phytopathology 25:317–338.

Mai, W. F., B. B. Brodie, M. B. Harrison, and P. Jatala. 1981. Nematode diseases of potatoes. Pp. 93–101 *in* W. J. Hooker, ed. Compendium of potato diseases. St. Paul, MN: The American Phytopathological Society.

Martin, S. B. 1988. Identification, isolation frequency, and pathogenicity of anastomosis groups of binucleate *Rhizoctonia* spp. from strawberry roots. Phytopathology 78:379–384.

May, G. M., and M. P. Pritts. 1994. Seasonal patterns of growth and tissue nutrient content in strawberry. Journal of Plant Nutrition 17: 1149–1162.

Ribeiro, O. K., and L. L. Black. 1971. *Rhizoctonia fragariae:* A mycorrhizal and pathogenic fungus of strawberry plants. Plant Disease Reporter 55:599–603.

Riedel, R. M., R. C. Rowe, and M. J. Martin. 1985. Differential interactions of *Pratylenchus crenatus, P. penetrans,* and *P. scribneri* with *Verticillium dahliae* in potato early dying disease. Phytopathology 75: 419–422.

Steel, R. G. D., and J. H. Torrie. 1980. Principles and procedures of statistics: A biometrical approach, 2nd ed. New York: McGraw-Hill Co.

Townshend, J. L. 1963. The pathogenicity of *Pratylenchus penetrans* to strawberry. Canadian Journal of Plant Science 43:75–78.

Webster, J. M. 1985. Interaction of *Meloidogyne* with fungi on crop plants. Pp. 183–192 *in* J. N. Sasser and C. C. Carter, eds. An advanced treatise on *Meloidogyne,* vol 1. Raleigh, NC: North Carolina State University Graphics.

Wilhelm, S., and P. E. Nelson. 1970. A concept of rootlet health of strawberries in pathogen-free soil achieved by fumigation. Pp. 208–215 *in* T. A. Toussoun, R. V. Bega, and P. E. Nelson, eds. Root diseases and soil-borne pathogens. Berkeley: University of California Press.

Wilhelm, S., P. E. Nelson, H. E. Thomas, and H. Johnson. 1972. Pathology of strawberry root rot caused by *Ceratobasidium* species. Phytopathology 62:700–705.

Wilhelm, S., and I. Vertrees. 1964. Pathological changes induced in strawberry rootlets attacked by *Rhizoctonia*. Phytopathology 54:1438.

Zunke, U. 1990. Observations on the invasion and endoparasitic behavior of the root lesion nematode *Pratylenchus penetrans*. Journal of Nematology 22:309–320.