Pathogenicity and Host Specificity of *Rhizoctonia solani* Isolated from Carrots

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ABSTRACT

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Twenty-eight isolates of *Rhizoctonia solani* representing anastomosis groups (AG) 1, 2, 4, and 5 were obtained from cankered carrots and hymenia formed on carrot petioles in Minnesota. Isolates differed in the type and amount of disease they caused and in host specificity on carrots, radishes, and potatoes. One AG1 isolate and six AG4 isolates caused damping-off of carrot seedlings, slight damage to mature carrot roots, and lesions on radish roots. The AG2 isolates were divided into two groups based on anastomosis tests and pathogenicity on carrot and radish. Fourteen AG2 type 2 isolates caused damping-off of carrot seedlings and cankers on mature carrot roots, and two representatives of this group caused lesions on radish roots. A second group of three AG2 type 1 isolates, obtained from hymenia on petioles of carrots, was nonpathogenic on seedling or mature carrots, but caused lesions on radish roots. The AG5 isolates were nonpathogenic on carrot seedlings or roots, or radish roots.

Additional key words: crop rotation, *Daucus carota* var. *sativa*, disease resistance, *Rhaphanus sativus, Solanum tuberosum*, vegetable.

In the north central United States, Rhizoctonia canker and crown rot caused by *Thanatephorus cucumeris* (Frank) Donk, the perfect state of *Rhizoctonia solani* Kühn, are serious problems on carrots in muck soil. These diseases have been reported from all major growing areas in Minnesota, but are more prevalent in fields with a history of continuous carrot cropping.

Crown rot of carrots was first reported by White (17) in 1926 to be caused by *Rhizoctonia solani*. Mildenhall and Williams (9) confirmed that isolates of *R. solani* belonging to anastomosis group 2 (AG2) induced crown rot and cankers of carrots in Wisconsin muck soils.

Schultz (14) and Richter and Schneider (13) proposed that the capacity of field isolates of *R. solani* to anastomose indicated a natural relationship within the species. Parmeter et al (12) found that most of the 138 isolates of *R. solani* tested fell into one of four anastomosis groups. Anastomosis occurred between isolates of the same group, but not between isolates of different groups. Therefore, they proposed that anastomosis groups are genetically isolated and incapable of nuclear exchange. A fifth anastomosis group was described by Ogoshi (11).

Isolates belonging to AG1, AG2 types 1 and 2, AG4, and AG5 of *R. solani* were found in association with muck-grown carrots. In this study, anastomosis group relationships, pathogenicity, and host specificity were investigated. Particular attention was given to the identification and characterization of isolates causing cankers on carrots.

MATERIALS AND METHODS

Isolation and identification of *R. solani* isolates. Isolates of *R. solani* were obtained from canker lesions, from sclerotia on carrot roots, and from hymenia on petioles of field-grown carrots and stems of black mustard, *Brassica nigra* (L.) Koch. The hymenia-bearing hosts had no disease symptoms. Cultures were maintained on potato-dextrose agar (PDA) by regular transfers at 30- to 60-day intervals.

Hyphal tips were excised from all field isolates and transferred from PDA to soil-extract agar (4) for basidiospore production. Single basidiospore cultures of unknown isolates and single basidiospore cultures of known AG1 and AG4 isolates were opposed on migration-complete agar (MCA) (16). A tuft of heterokaryotic hyphae formed at the junction of paired homokaryons belonging to the same anastomosis group (2,5). Anastomosis tests for field isolates that did not produce basidiospores were made by opposing them with field isolates of AG1, AG2-1, AG2-2 (provided by E. G. Ruppel), AG3, AG4, and AG5 (Ogoshi isolates 440, 441, and 443) on PDA- and water agar (WA)-coated microscope slides or in petri plates containing PDA, WA, or MCA.

Pathogenicity tests in the laboratory. Pathogenicity of *R. solani* isolated from carrot on germinating seedlings of carrot, radish, and parsnip was tested in the laboratory by a method similar to that of Garza-Chapa and Anderson (5). Surface-disinfested seeds were germinated on the surface of WA covered with the mycelium of the isolate being tested. Plates were incubated at 20–25 C in indirect light. Pathogenicity was evaluated by the occurrence of seed rot and hypocotyl infections. Hypocotyl infection was defined as distinct, brown, watery lesions limited to hypocotyl tissue resulting in rapid death of the seedling. Carrot cultivar Scarlet Nantes; radish cultivars Early Scarlet Globe, White Icicle, Red Prince, Far Red, and Fuego; and parsnip cultivar 640 Harris model were tested.

Pathogenicity tests in the greenhouse. *Carrot.* Fifteen isolates of *R. solani* from carrot canker lesions, three isolates from hymenia on carrot petioles, and two from other hosts were tested to determine their ability to cause seed and seedling disease of carrot. A cornmeal-sand-perlite mixture (1:10:10, v/v) infested with each isolate was added to pasteurized sandy-loam soil at a ratio of 1:20 (v/v). Two-hundred Scarlet Nantes carrot seeds were planted in infested soil and maintained at 24 C in the greenhouse. The number of surviving seedlings was counted 28 days after planting.

Ten 30-day-old Scarlet Nantes carrot seedlings maintained in the greenhouse at 24 C were inoculated with the 15 isolates by placing one infested corn kernel approximately 3 cm below the soil surface and 2 cm from each plant. The number of surviving plants was determined 48 days after inoculation. Ninety-day-old carrots were inoculated with corn kernels infested with isolate R-29 (AG1) from scurfed radishes, isolate R-31 (AG2-1) from large tan lesions on radishes, isolate R-33 (AG2-1) from root lesions on parsnip, and R-9 (AG2-2) from carrot canker lesions.

Radish. Six isolates of *R. solani* obtained from diseased carrot

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roots or hymenia on carrot petioles (AG1 isolate R-1; AG2-1 isolate R-17; AG2-2 isolates R-9 and R-11; AG5 isolate R-19; and AG4 isolate R-28) were tested for pathogenicity on five radish cultivars (Early Scarlet Globe, White Icicle, Red Prince, Far Red, and Fuego) in a replicated trial. Radishes were grown in pasteurized soil and maintained at 24 C. Plants were inoculated with infested corn kernels 2 or 4 wk after planting. Plants were harvested 14 and 10 days after inoculation, respectively. Disease symptoms and severity were noted.

Potato. Pathogenicity of five isolates of *R. solani* from carrot (AG1 isolate R-1; AG2-1 isolate R-17; AG2-2 isolates R-2 and R-10; and AG4 isolate R-28) to five potato cultivars (Cascade, Chieftan, Kennebec, Norland, and Russet Burbank) was tested in a greenhouse maintained at 18 C. Potato tissue with a single sprout was cut from a tuber visually free from scurf and planted in pasteurized sand. A 6-mm-diameter PDA inoculum disk was placed near each sprout.

Temperature effects. The influence of soil temperature on the ability of isolate R-9, an AG2-2 carrot canker isolate, to cause seedling blight and root canker, was investigated. Scarlet Nantes carrots were planted in infested soil adjusted to 10, 12, 15, 18, and 24 C. Counts of surviving seedlings were made 28 days after planting.

Scarlet Nantes carrots near maturity were transferred to controlled temperature tanks where soil temperatures were adjusted to 16, 22, 26, and 30 C. Plants were inoculated with infested corn kernels. Disease severity was measured 15 days after inoculation.

Pathogenicity tests in the field. *Becker, MN.* Pathogenicity to carrot of eight isolates of *R. solani* was tested on irrigated, sandy soil at the Sand Plain Experiment Station, Becker, MN, in 1977. The experimental design was a randomized complete block with four replicates and included a split-plot arrangement of eight isolates, a control, and six carrot cultivars. Cultivars were assigned to main plots of three 10.8-m rows 38 cm apart. A two-row border planted with carrot cultivar Hipak was used for each main plot. Cultivars tested were Scarlet Nantes, Royal Chantenay, Spartan Premium, Danvers, Imperator, and Grenadier. Subplots consisted of 120-cm sections of row within the main plots. The plants in a 30-cm section in the center of each subplot and border row were inoculated with the appropriate isolate. Isolates used were AG1, R-1; AG2-2, R-4, R-5, R-9, and R-10; AG2-1, R-17; AG4, R-28; and AG5, R-19 and R-21. An uninoculated check subplot was included as a control in each main plot. Sixty-five days after planting, one infested corn kernel was placed approximately 3 cm below the surface and 3 cm from each plant. An average of eight plants of each cultivar per replication was inoculated. Plants were harvested and evaluated 67 days after inoculation and disease severity was evaluated on a scale of 0–4 in which 0 = no symptoms and 4 = 76–100% of the carrot storage organ surface covered with lesions.

Scarlet Nantes plants were also inoculated with two AG2-1 isolates, one the causal agent of radish wirestem (R-30) and the other a crucifer pathogen (R-32). Eleven 30-cm sections of row were inoculated with each isolate. Inoculations were made 65 days after planting, and plants were harvested and evaluated (as described above) 67 days after inoculation.

Owatonna, MN. Since most commercially grown carrots in Minnesota are produced on organic soils, an experiment was performed on organic soil near Owatonna, MN, in 1977. Three sections (6.1 × 4.6 m) of a production field were fumigated with 1,200 kg a.i. metam-sodium per hectare and planted one cultivar per row, in nine rows 38 cm apart. Sixty-day-old plants were inoculated with corn kernels infested with AG2-2 isolate R-9. Three 30-cm sections of each row were inoculated in each fumigated area. Carrot cultivars tested were: Scarlet Nantes, Royal Chantenay, Spartan Premium, Hybrid 310 Hipak, Hybrid 308 Grenadier, G.T. 26, Danvers, Gold King, and (M872× M5931)×M107 (a Michigan State University experimental line).

Overwintering study. The potential of Rhizoctonia canker-causing isolates to survive from one season to the next was investigated. In April 1978 the Becker Sand Plain Experiment carrot field was fertilized and rototilled. Carrot cultivars Scarlet Nantes, Royal Chantenay, and Imperator were planted over the same rows used in 1977. Only every other row was planted. Radishes were planted in May 1978 to determine the potential of canker-causing isolates to overwinter and cause disease of radish. Five rows of radish cultivar Cherry Belle and four rows of cultivar Early Scarlet Globe were planted between rows of carrots planted earlier.

The radishes were harvested 30 days after planting, and the type of disease symptoms were noted.

RESULTS

Isolation and identification of isolates of *R. solani*. Twenty-eight cultures of *R. solani* isolated from carrots were identified as belonging to AG1 (microsclerotia type), AG2-1, AG2-2, AG4, and AG5 (Table 1). Cultural characteristics on PDA of the AG1 isolate were similar to the descriptions of Sherwood (15) for AG1 microsclerotial isolates. The AG4 isolates were also similar to his description for this group. The AG2-1 isolates were nearly white and formed sclerotia. The mycelium of the AG2-2 isolates was a moderately dark, chocolate brown and produced variable numbers of brown crusty sclerotia. The AG5 isolates produced light brown, sparse mycelium with irregularly shaped, light brown sclerotia.

Pathogenicity test in the laboratory. The AG2 isolates generally caused less carrot seed rot than did AG1 or AG4 isolates (Table 2). Hypocotyl infections were absent with the AG2-1 and AG2-2 isolates, rare (2%) with the AG1 isolate, and most frequent (19–31%) with the AG4 isolates. There was no seed rot of radish and parsnip, but the AG1 and AG4 isolates caused a high percentage of hypocotyl infections. The R-17 (AG2-1) isolate from the hymenium produced on carrot petioles caused small, rectangular black lesions on the hypocotyl and on primary roots of the germinating radish seedlings.

TABLE 1. Geographic origin, anastomosis group (AG), disease symptom or fungal structure, and host from which isolates of *Rhizoctonia solani* were obtained

Isolate number	AG	Type	Geographic origin	Symptom/structure
R-1	1	Microsclerotial	Owatonna, MN	Canker lesion on carrot roots
R-2 to R-4	2	2	Owatonna, MN	Sclerotia on carrot roots
R-5 to R-10	2	2	Owatonna, MN	Canker lesion on carrot roots
R-11 to R-15	2	2	St. Francis, MN	Canker lesion on carrot roots
R-16 to R-18	2	1	Anoka, MN	Hymenium on carrot petioles
R-19 to R-20	5		Ham Lake, MN	Canker lesion on carrot roots
R-21 to R-22	5		Owatonna, MN	Canker lesion on carrot roots
R-23 to R-28	4		Owatonna, MN	Canker lesion on carrot roots
R-29	1		Florida	Black sclerotia on radish roots
R-30	2	1	Ham Lake, MN	Wirestem on radish roots
R-31	2	1	Florida	Lesions on radish roots
R-32	2	1	Australia	Root lesion on crucifer
R-33	2	1	Anoka, MN	Root lesion on parsnip

TABLE 2. Mean percent seed rot and hypocotyl infection for isolates of *Rhizoctonia solani* from carrot tested on cultivars of carrot, radish, and parsnip[a]

AG	Carrot			Radish[b]			Parsnip		
	Isolates tested (no.)	Mean sr[c] (%)	Mean hy[d] (%)	Isolates tested (no.)	Mean sr (%)	Mean hy (%)	Isolates tested (no.)	Mean sr (%)	Mean hy (%)
1	1	45	2	1	0	88	1	0	46
2-1	3	22	0	1	0	0	1	0	0
2-2	14	3	0	6	0	0	6	0	0
4	6	63	27	1	0	81	1	0	33
5	4	37	0	1	0	0	1	0	0

[a] Cultivars tested were Scarlet Nantes carrot; Early Scarlet Globe, White Icicle, Red Prince, Far Red, and Fuego radish; and 640 Harris Model parsnip.
[b] Mean for all five radish cultivars.
[c] Mean percent seed rot (sr) of four replications per cultivar, 20 carrot seeds, 10 radish seeds, and 20 parsnip seeds per replications.
[d] Mean percent hypocotyl infection (hy) of seedlings surviving seed rot.

AG-2 isolates caused a generalized root rot of all three hosts by the end of the test period, in contrast to the distinct hypocotyl lesions caused by AG1 and AG4 isolates.

Pathogenicity tests in the greenhouse. *Carrot.* Damping-off incidence caused by the 20 isolates of *R. solani* differed among anastomosis groups (Table 3). The AG1 isolate caused pre- and postemergence damping-off, while the AG2-2 isolates primarily caused preemergence damping-off. The AG2-1 and AG5 isolates were not pathogenic on carrots. The AG1, AG2-1, and AG4 isolates caused no observable symptoms on 30-day-old plants (Table 3). The darkly pigmented AG2-2 isolates were the only pathogenic isolates.

On 90-day-old carrots, isolate R-33 from parsnip did not cause visible symptoms; isolate R-31 from large tan lesions on radish caused large, corky lesions on the carrot not typical of canker lesions; and AG1 isolate R-29, which causes scurf of radish, also produced callus lesions, primarily in the hypocotyl region of the carrot storage organ. None of these three isolates were as damaging to carrot as the AG2-2 isolate, R-9, which caused typical cankers on the roots.

Radish. The six isolates from carrot differed as to type of disease caused and severity of the infection on radish (Table 4). The AG1 isolate (R-1) caused distinct black lesions on radish (Fig. 1A), as did the AG4 isolate (R-28). Isolates R-9 and R-11 (AG2-2) caused large tan lesions (Fig. 1B) and the hymenial isolate, R-17 (AG2-1), caused uneven development of the expanded taproot and brown-to-black irregularly shaped lesions (Fig. 1C). Isolate R-19 (AG5) caused no symptoms. When 2- and 4-wk-old plants were inoculated, the types of lesions produced were the same; however, where less mature plants were inoculated, infection was more severe with the AG2-1 and AG2-2 isolates and less with the AG1 and AG4 isolates.

Differences in cultivar response were noted only in 4-wk-old plants inoculated with AG4 isolate R-28 and with 2-wk-old plants inoculated with AG2-2 isolate R-11 and AG2-1 isolate R-17. No cultivar was resistant to any pathogenic isolate.

Potato. AG1, 2, and 4 isolates of *R. solani* from carrot did not cause symptoms on potato sprouts.

Temperature effects. AG2-2 isolate R-9 did not cause damping-off at soil temperatures of 10, 12, 15, or 18 C in *R. solani*-infested pots, but did so at 24 C (Table 5). Cankers on carrot roots 2 wk after inoculation were less severe in plants grown at a soil temperature of 16 C than at 22, 26, or 30 C (Table 6).

Pathogenicity tests in the field. *Becker, MN.* AG2-2 isolates R-4, R-5, R-9, and R-10 were determined to be causal agents of Rhizoctonia canker and crown rot of carrot (Fig. 2A). These darkly pigmented AG2-2 isolates of *R. solani* were consistently recovered from diseased plants. When a one-way ANOVA was run for each cultivar, a Duncan's multiple range test showed that isolate R-10 was less virulent than the other canker-causing isolates (R-4, R-5, and R-9) on all six cultivars for $\alpha < 0.05$.

No cultivar tested was resistant to the canker-causing organisms; however, there were significant differences in cultivar response (Table 7).

Occasionally, plants inoculated with isolate R-1 (AG1) and isolate R-28 (AG4) had callus-filled lesions (Fig. 2B). Callus-filled lesions were also found on 19 of 55 Scarlet Nantes carrots inoculated with AG2-1 isolate R-30, the causal agent of radish wirestem (Fig. 2C), and on 34 of 91 plants inoculated with AG2-1 isolate R-32, the crucifer pathogen. Neither *R. solani* nor any other fungus was consistently recovered from these lesions.

Owatonna, MN. Sixty days after inoculation of carrots grown on organic soil at Owatonna, MN, all plants in the 30-cm-inoculated section were dead. The approximate distance of spread down the row from the point of inoculation was 43 cm for AG2-2 isolate R-9. No difference in cultivar response was noted.

Overwintering study. When carrots were planted in the

TABLE 3. Pathogenicity of isolates of *Rhizoctonia solani* on carrot seeds and seedlings and on established carrots

AG	Isolates tested[a] (no.)	Seedlings surviving 28 days after planting[b] (mean no.)	Plants surviving 48 days after inoculation[c] (mean no.)
Control		56	10
1	1	12[d]	10
2-1	3	53	10
2-2	9	3	1
5	4	46	10
4	1	23	10
2-1	1[e]	43	10
2-1	1[f]	41	10

[a] All isolates from carrot except as noted in footnotes e and f.
[b] Number of seedlings out of 200 seeds planted in infested soil.
[c] Number of surviving plants of 10 inoculated.
[d] Pre- and postemergence damping-off observed.
[e] Isolate from radish.
[f] Isolate from crucifer.

TABLE 4. Disease severity on radish taproots inoculated with isolates AG1, AG2-1, AG2-2, AG4, and AG5 of *Rhizoctonia solani* from carrot in the greenhouse

Isolate	Disease severity[x] in plants:	
	2 wk old	4 wk old
R-9 (AG2-2)	4.2 a[y]	0.3 d
R-11 (AG2-2)	4.0 a[z]	1.0 c
R-17 (AG2-1)	3.8 a[z]	...
R-1 (AG1)	1.7 b	2.2 a
R-28 (AG4)	0.5 c	1.4 b
R-19 (AG5)	...	0.0 d
Control	0.0 d	0.0 d

[x] Mean disease severity of four replications, except as noted in *z*, rated on a 0–5 scale in which 0 = no symptoms, 1 = 1–20%, 2 = 21–40%, 3 = 41–60%, 4 = 61–80%, and 5 = 81–100% of the expanded taproot surface covered with lesions.
[y] Means in a column followed by the same letter do not differ significantly at $P = 0.05$ by Duncan's multiple range test.
[z] Mean based on three replications per cultivar.

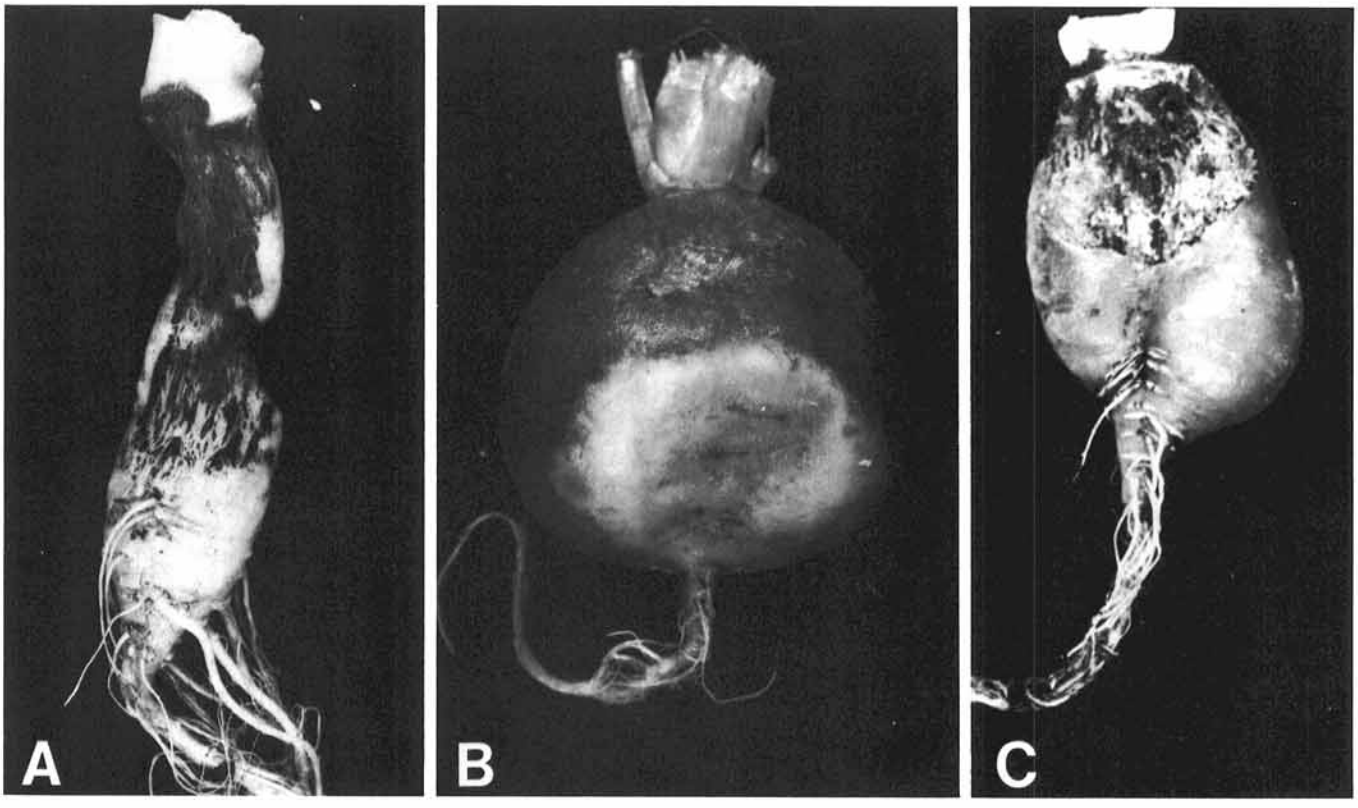

Fig. 1. Radish cultivars inoculated with isolates of *Rhizoctonia solani* associated with carrots. **A,** Four-week-old White Icicle plant inoculated with AG1 isolate R-1; **B,** 4-wk-old cultivar Far Red plant inoculated with AG2-2 isolate R-9; and **C,** 2-wk-old Far Red plant inoculated with AG2-1 isolate R-17.

TABLE 5. Influence of soil temperature on the number of carrot plants per pot 28 days after seeding carrots in soil infested with isolate R-9 (AG2-2) of *Rhizoctonia solani*

Temp (C)	Number of plants[a]		Statistical significance[b]
	Inoculated	Control	
10	58	52	n.s.
12	46	38	n.s.
15	55	45	n.s.
18	31	45	n.s.
24	34	70	sig.

[a] Mean of four replications. Number of plants per 100 seeds planted.
[b] Differences in number of plants between control and inoculated treatments were analyzed by Student's *t*-test; ($P = 0.05$). n.s. = not significant; sig. = significant.

TABLE 6. Mean disease severity on carrot taproots inoculated with isolate R-9 (AG2-2) of *Rhizoctonia solani* at 16, 22, 26, and 30 C

Temp (C)	Disease severity[x]	
	Inoculated	Control
16	1.32 a[y]	0 a
22	4.49 b	0 a
26	3.87 b	0 a
30	3.22 b	0 a

[x] Mean disease severity of three replications. A disease severity rating of 0–5 was used where 0 = no symptoms; 5 = 81–100% of the carrot taproot covered with lesions.
[y] Mean values within a column followed by a common letter do not vary significantly according to Duncan's multiple range test ($P = 0.05$).

inoculated field at Becker, MN, dead plants and plants with crown rot symptoms were observed 56 days after planting. The incidence of diseased and dead carrots increased appreciably between 56 and 80 days after planting. Dark brown, dry lesions formed at the base of the leaves and in a band 1- to 2-cm wide in the hypocotyl region just below the soil surface. Movement of the pathogen in the soil by rototilling appeared to be minimal, since infections occurred very near the areas of the Rhizoctonia canker infections the year before. Dark-pigmented AG2-2 cultures of *R. solani* were consistently isolated from diseased plant tissue.

Radishes of both cultivars became diseased in areas that were inoculated with AG2-2 isolates R-4, R-5, R-9, and R-10 the preceding season and from which cankered carrots had been harvested. Symptoms ranged from small lesions to plant death, and included large tan lesions similar to those produced in the greenhouse when radishes were inoculated with the same isolates. Disease severity ranged from one lesion on a single plant to the death of several plants in each treatment. With a single exception, no disease development occurred in areas infested with the other isolates of *R. solani* the preceding season. All isolates recovered from diseased radishes were *R. solani* AG2-2.

DISCUSSION

Isolates of *R. solani* used in these studies differed in pathogenicity and host range. The AG1 and AG4 isolates from carrot are potentially destructive to carrots in causing seedling blight. The AG1 isolate caused pre- and postemergence damping-off while the AG4 isolates caused primarily preemergence damping-off. Sherwood (15) observed similar effects with the AG1 and AG4 isolates with which he worked.

The AG2-2 isolates caused seedling blight and Rhizoctonia canker of carrot and lesions on mature radish roots, while AG2-1 isolates caused lesions on mature radish roots but almost no disease symptoms on carrot. AG2-1 isolates were all from hymenia found on carrot and black mustard in areas that had been planted to radish the previous year. Carrots were not a host for the AG2-1 isolates, but did provide a site for sexual recombination of these isolates. AG5 isolates failed to cause disease on carrot or radish.

Damping-off was caused by the AG2-2 isolates in the greenhouse, but was not observed in the spring in production fields where Rhizoctonia canker incidence was quite high later in the

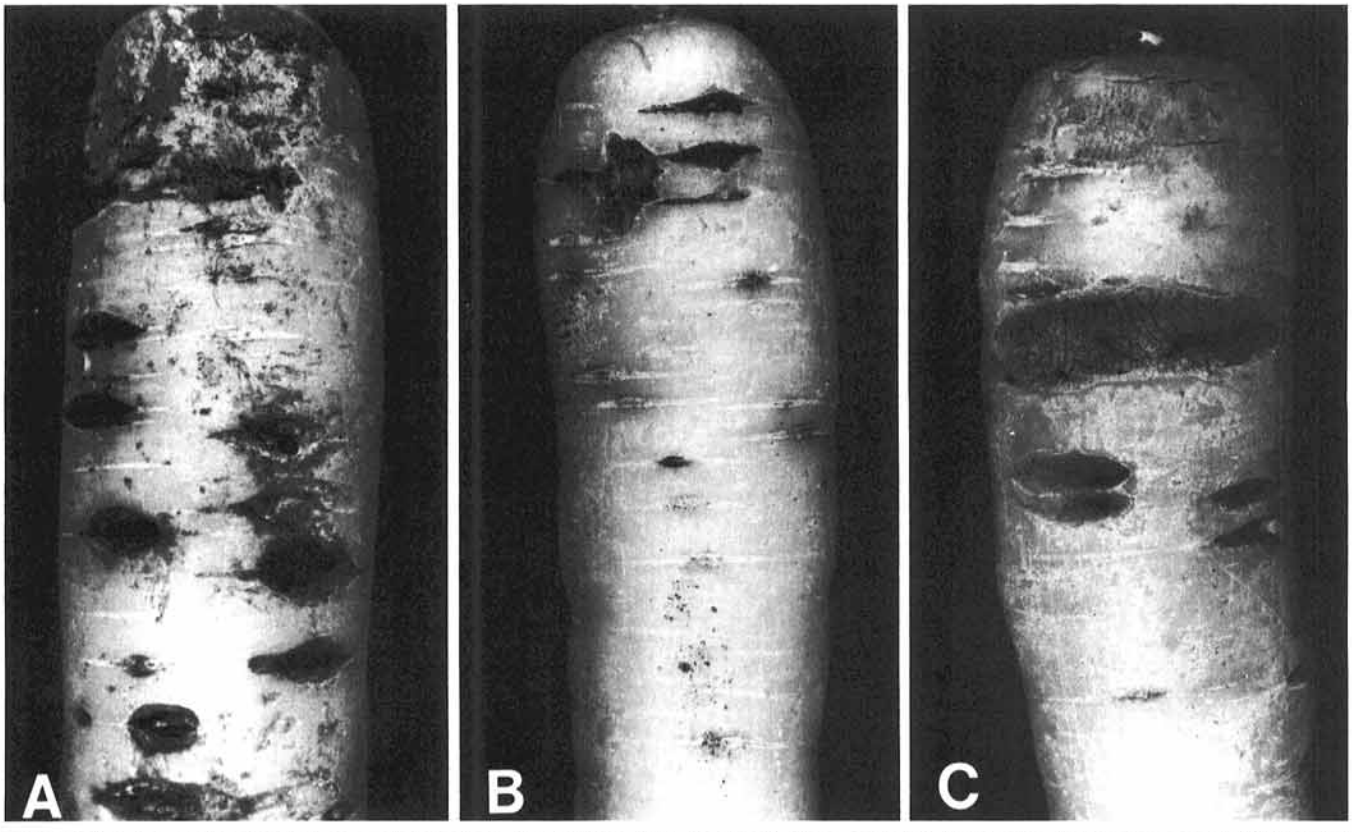

Fig. 2. Sixty-five-day-old Scarlet Nantes carrot plants inoculated with **A,** AG2-2 isolate R-4 from carrot; **B,** AG1 isolate R-1 from carrot; **C,** AG2-1 isolate R-30 from radish.

TABLE 7. Mean canker disease severity for six cultivars of carrots inoculated with nine isolates of *Rhizoctonia solani* at Becker, MN

	Disease severity[x]									
	AG1	AG2-1	AG2-2				AG4	AG5		
Cultivars	R-1	R-17	R-4	R-5	R-9	R-10	R-28	R-19	R-21	Control
Grenadier	0.2 a[y]	0.0 a	3.5 a	3.5 a	3.9 ab	1.9 a	0.1 a	0.0 a	0.1 a	0.0 a
Imperator	0.3 a	0.0 a	3.3 ab	3.4 ab	4.0 a	1.8 a	0.1 a	0.0 a	0.1 a	0.3 a[z]
Spartan Premium	0.3 a	0.0 a	3.0 b	3.0 bc	3.5 b	1.3 b	0.1 a	0.0 a	0.0 a	0.0 a
Royal Chantenay	0.5 a	0.0 a	3.2 ab	3.1 abc	3.5 ab	1.4 ab	0.2 a	0.2 a	0.1 a	0.0 a
Scarlet Nantes	0.4 a	0.1 a[z]	3.1 ab	3.0 bc	3.4 b	0.8 c	0.1 a	0.0 a	0.1 a	0.0 a
Danvers	0.3 a	0.0 a	2.3 c	2.7 c	3.7 ab	0.5 c	0.1 a	0.0 a	0.0 a	0.0 a

[x] Mean disease severity of four replicates, rated 0–4, in which 0 = no symptoms, 1 = 1–25%, 2 = 26–50%, 3 = 51–75%, and 4 = 76–100% of the carrot storage organ covered wtih lesions.

[y] Means in column followed by the same letter do not differ significantly at $P = 0.05$ according to Duncan's multiple range test.

[z] Contaminated by a canker-causing isolate of *R. solani*.

season. Mildenhall and Williams (10) made similar observations and suggested that the lower soil temperatures the first 4 wk of growth could inhibit the activity of *R. solani*. The controlled soil temperature studies reported in Table 5 support their suggestion. Soil temperatures of 10–15 C are encountered at planting time in Minnesota. Damping-off was not observed at 18 C and below.

Results of pathogenicity tests in petri dishes support the hypothesis that the AG1 and AG4 isolates attack seedlings by a different mechanism than do the AG2 isolates. The AG1 and AG4 isolates caused distinct hypocotyl lesions of carrots, radishes, or parsnips, whereas AG2 isolates caused generalized browning of the primary root and hypocotyl regions of affected seedlings.

The AG2-2 isolates from carrot produce different symptoms on carrot at different stages of plant maturity. Pre- and postemergence damping-off was observed in the greenhouse for approximately 30 days after planting. Crown rot symptoms were predominant in plants inoculated 30–60 days after seeding and in naturally infected plants of similar age in the field. Rhizoctonia canker was the predominant symptom in infected plants 60 or more days old. The incidence and severity of canker increased throughout the growing season, even during the final 30 days of a 150-day season. Mildenhall and Williams (9) made similar observations in Wisconsin. Their histological work indicated that in carrots inoculated prior to cambial activity, the cortex, endodermis, and the primary phloem and xylem become invaded. The fungus rapidly colonized the partially sloughed cortex and invaded the periderm of carrots inoculated 30 days or more after seeding. They also suggested that canker development may be related to the formation of the horizontal grooves. With expansion of the storage organ of the carrot, the cortex and endodermis are shed and the periderm, which arises from the pericycle, becomes a protective layer (3). *R. solani* was consistently recovered from small areas of purplish discoloration located at the horizontal grooves of carrots. These purplish areas are believed to be an early symptom of Rhizoctonia canker.

Canker severity is influenced by soil temperature. The results of the studies reported here are in agreement with the findings of Mildenhall and Williams (10), who showed that significantly less

infection occurred at 16 C and that 20–28 C favored canker development. This observation may partially explain the appearance of cankers in the fields at mid-season and subsequent progress of the disease throughout the season.

Carrot cultivars differed in susceptibility in the field at Becker, MN, but no cultivar tested was resistant. In subsequent years this same plot was used for cultivar testing, and moderate-to-partial carrot canker resistance was found (1). Mildenhall (8) screened 132 carrot lines in the greenhouse for potential resistance to a canker-causing isolate. He found greater resistance among open-pollinated cultivars than in inbred cultivars.

More information is needed on the genetics of the pathogenicity of the canker-causing isolates and on the potential for change in the field. Basidiospore production was not induced in the laboratory for the AG2-2 isolates, and hymenia of the isolates have not been observed in the field. Anastomosis of field isolates does not appear to be a mechanism of genetic exchange since fused cells die soon after fusion. If nuclear exchange is limited under field environments, resistance, if found, may prove to be stable. Differences were observed among the canker causing isolates. Isolate R-10 was significantly less virulent than any other AG2-2 isolate tested.

A carrot-potato-radish rotation is recommended for Minnesota and Wisconsin vegetable growers. Potato is recommended as an intermediate crop since it was not susceptible to AG1, AG2, or AG4 isolates tested. Previous research indicated that *R. solani* AG3 is responsible for black scurf and stem and stolon lesions on potato (6) and that AG3 isolates from potato are not pathogenic on 90-day-old carrots (7). Except for inability to infect potato, AG1 and AG4 isolates lacked host specificity. The AG2-1 and AG2-2 isolates, however, exhibited greater host specificity. Since radish was susceptible to the AG2-2 isolates from carrot and carrot was not susceptible to the two AG2-1 isolates from radish, it is recommended that radish precede carrot in the rotation.

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